KESMONDS INTERNATIONAL UNIVERSITY
Entrepreneurial & Skilled Education for the Future
IQ RESEARCH JOURNAL
www.iqresearchjournal.com
KESMONDS RESEARCH INSTITUTE

Volume 5, Issue 2 - MAY 2026

IQ RESEARCH

A Quaterly Journal

ISSN: 2790-4296 (Online)
ISBN: 978-9956-504-74-9 (Print)
Published by IQRJ publications
www.iqresearchjournal.com

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

EDITORIAL BOARD

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Table of Contents

IQ Research Journal · Volume 005, Issue 002, May 2026 · 20 Original Articles

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Clinical Profile of Candidiasis in Immunocompromised Patients Attending the Bamenda Regional Hospital: Antifungal Susceptibility Testing on *Candida albicans*

Che Amadine Lem[a,*], Augustine Nji Asakizi[a] and Forcham Emmanuel Duna[a]

Affiliations

a. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Che Amadine Lem
Email: cheamadine@gmail.com

Paper ID: IQRJ-V05I02-26005001

ABSTRACT

Fungi are free-living, eukaryotic organisms that exist as yeasts, moulds, or dimorphic forms. Oral candidiasis is a common fungal infection affecting the oral mucosa, primarily caused by Candida albicans, a component of normal oral microflora in 30-50% of individuals. This study determines the clinical profile of candidiasis among immunocompromised individuals and the antifungal susceptibility of Candida albicans. A hospital-based cross-sectional analytical study was conducted at Bamenda Regional Hospital, Cameroon. Data were collected using structured questionnaires and analyzed with SPSS version 21.0. Of 500 participants, most were aged 28-37 years (51.2%), female (87.2%), married (59.4%), and Christian (99%) ($p<0.001$). Prevalence rates were: oral candidiasis (3.4%), gastrointestinal (GI) candidiasis (5.8%), and vulvovaginal (VV) candidiasis (26.6%). Clotrimazole, itraconazole, and flucytosine were sensitive for oral, GI, and VV candidiasis, respectively; griseofulvin was resistant to all. Risk factors included mouth sores/stings for oral candidiasis; nausea, abdominal pain, constipation for GI; and vaginal discharge/pain during sex for VVC. Public education on candidiasis, rational antifungal use, and hospital-based resistance monitoring are recommended to reduce prevalence and resistance.

Keywords: *Candidiasis, Oral Candidiasis, Gastrointestinal Candidiasis, Vulvovaginal Candidiasis, Antifungal Susceptibility, Immunocompromised Patients, Cameroon*

1. INTRODUCTION

Fungi are eukaryotic organisms existing as yeasts (e.g., *Candida*), moulds, or dimorphic forms. Oral candidiasis ("thrush") is an opportunistic infection caused by *Candida albicans*, part of normal oral flora in 30–50% of people, with carriage increasing with age (Lamont et al., 2006). *C. albicans* colonizes skin and mucosa, including oral and vaginal sites, in up to 60% of healthy individuals (Kasper et al., 2005). In immunocompromised patients, such as those with HIV, it causes opportunistic infections (Obradovic et al., 2011).

Oral candidiasis affects the mucosa, relying on virulence factors like adhesins, enzymes, morphologic switching, and drug resistance (Javed et al., 2014). In the gastrointestinal tract (GIT), *C. albicans* is a commensal but can disseminate in disturbed microbiomes (Hussain & Douglas, 2017). Vulvovaginal candidiasis (VVC) affects millions annually, mainly by *C. albicans*, with non-albicans species increasing; risk factors include pregnancy, diabetes, and immunosuppression (Silva et al., 2011).

In HIV patients, candidiasis ranges from colonization to invasive forms, with CD4 counts <200 cells/μ*l* increasing risk (Ozkan, 2005). Antifungals target pathways like cell wall (echinocandins), ergosterol (azoles), or nucleic acids (5FC), but resistance is rising (Lamichhanae et al., 2015). HIV-related opportunistic fungal infections (OFIs) cause significant morbidity and mortality, with impaired immunity heightening risk (Hsia et al., 2012). Recent studies highlight invasive fungal infections in immunocompromised hosts (Antinori et al., 2025). In Cameroon, data on patterns and susceptibility are limited, necessitating this study to inform diagnosis, treatment, and resistance monitoring.

This study integrates novel approaches to health education, adapting virtual methods for candidiasis awareness (Shinta et al., 2025), and focuses on clinical profiles as sentinels for HIV progression (Ambe et al., 2020), gastrointestinal risks (Kreulen et al., 2023), and VVC prevalence (Mohamed et al., 2022).

2. RELATED WORKS

Candidiasis in immunocompromised patients is well-documented. Pappas et al. (2016) updated IDSA guidelines for management, emphasizing susceptibility testing. UNAIDS (2019) data highlight HIV prevalence in sub-Saharan Africa, correlating with candidiasis. Hussain & Douglas (2017) described *C. albicans* biofilms, enhancing resistance (Douglas, 2003). World Health Organization (2025a) lists fungal priority pathogens, including *Candida*. Vanden reported genital candidiasis prevalence; Ambe et al. (2020) found 36% oral candidiasis in HIV patients. Nguefack et al. (2024) and Ngouana et al. (2017) provided local insights. Okungbowa et al. (2003) and Olum et al. (2020) noted esophageal candidiasis in SSA. Gow & Hube (2012) and Ozkan (2005) emphasized cell wall importance. Kreulen et al. (2023) linked *Candida* to intestinal health. Silva et al. (2011) and Sobel (2016) detailed VVC risks. Clinical and Laboratory Standards Institute (2010) standards guide susceptibility testing. Mohamed et al. (2022) meta-analyzed VVC in pregnant African women. World Health Organization (2025b) prioritizes AMR research, including fungi. Additional studies include Sun et al. (2025) on *C. auris* outbreaks and Lee et al. (2025) on *C. albicans* pathogenesis.

1.1. Antifungal Resistance Mechanisms

Antifungal resistance in *Candida albicans* involves multiple mechanisms. Azoles inhibit 14-demethylase, but resistance arises from ERG11 mutations, efflux pumps (CDR1/CDR2, MDR1), and biofilm formation (Bhattacharya et al., 2020). Echinocandin resistance stems from FKS1/FKS2 mutations reducing glucan synthase sensitivity (Perlin, 2017). Polyene resistance, though rare, involves ergosterol pathway alterations (Cowen et al., 2002). Biofilms confer resistance via extracellular matrix, persister cells, and metabolic changes (Nett & Andes, 2010). Genetic factors like aneuploidy and

epigenetic modifications also contribute (Selmecki et al., 2006). These mechanisms highlight the need for susceptibility testing and novel therapeutic strategies.

3. MATERIALS & METHODS

This cross-sectional study at Bamenda Regional Hospital enrolled 500 immunocompromised participants (pri- marily HIV-positive). Inclusion criteria: confirmed immunocompromised status, consent. Exclusion: recent antifungal use.

Data collection involved structured questionnaires on demographics, symptoms, and risks. Samples (oral swabs, stool, high vaginal swabs) were cultured and identified. Antifungal susceptibility testing used disk diffusion per Clinical and Laboratory Standards Institute (2010) standards against clotrimazole, itraconazole, flucytosine, griseofulvin, and fluconazole.

Statistical analysis was performed using SPSS version 21.0. Descriptive statistics included frequencies, percent- ages, means, and standard deviations. Prevalence was calculated with 95% confidence intervals (CI). Associations between variables were assessed using chi-square tests or Fishers exact test where appropriate, with $p<0.05$ considered significant. Multivariate logistic regression modeled the probability of candidiasis based on risk factors, reporting odds ratios (OR) with 95% CI. Logistic regression uses the logit function to predict binary outcomes, adjusting for confounders (Sperandei, 2014). The model is defined as:

$$z = \ln\left(\frac{P}{1-P}\right) = \beta o + \beta_i X_i + \ldots + \beta n Xn$$

where p is the probability of candidiasis, $\beta 0$ is the intercept, β 1 are coefficients, and $X1$ are predictors (e.g., symptoms, demographics). ORs were derived as $e^{\beta i}$ (Szumilas, 2010).

Ethical approval was obtained from the Regional Delegation of Public Health. The framework incorporates suggestions on oral candidiasis as a sentinel for HIV (Ambe et al., 2020), GI risks (Kreulen et al., 2023), and VVC prevalence (Mohamed et al., 2022).

4. RESULTS & DISCUSSION

Participants were aged 18–67 years (mean 32.5 ± 8.2); 51.2% were 28–37 years; 87.2% female; 59.4% married; 99% Christian; income varied (50% <50,000 XAF monthly). Demographic factors age, sex, marital status and religion were significantly associated with candidiasis presence (chi-square test, $X^2 = 45.6$, df=12, $p<0.001$).

Prevalence rates with 95% CI: oral candidiasis 3.4% (n=17, 95% CI: 1.8–5.0%); GI candidiasis 5.8% (n=29, 95%

CI: 3.8–7.8%); VVC 26.6% (n=133, 95% CI: 22.7–30.5%).

Susceptibility results: Clotrimazole was 100% sensitive for oral candidiasis (n=17 isolates); itraconazole 93.1% for GI (n=29); flucytosine 97% for VVC (n=133); fluconazole was >80% across types. Griseofulvin was resistant (0% sensitivity). Azole resistance was observed in 15% of isolates, consistent with Berman & Krysan (2020).

Low oral candidiasis prevalence compared to Ambe et al. (2020) (36%) may reflect improved ART access in Cameroon. Higher VVC prevalence aligns with Mohamed et al. (2022), particularly in females. Risk factors (e.g., mouth sores, vaginal discharge) indicate a sentinel role for HIV progression, as CD4 decline exacerbates candidiasis (Ozkan, 2005).

Table 1 : Risk Factors, and Statistical Associations for Candidiasis Types

Type	95%CI	Factors	Univariate	AOR	P-Value
Oral	4.21 (3.21-5.13)	Mouth sores Stings during eating White patches	0.001 0.003 0.012	3.2 (1.5–6.8)	<0.001
GI	2.32 (1.12-3.15)	Nausea Abdominal pain Constipation Diarrhea	0.002 0.001 0.004 0.015	2.7 (1.3–5.6)	<0.001
VVC	3.19 (2.11- 4.81)	Vaginal discharge Pain during sex Vulvar itching	<0.001 <0.001 0.002	4.1 (2.4–7.0)	<0.001

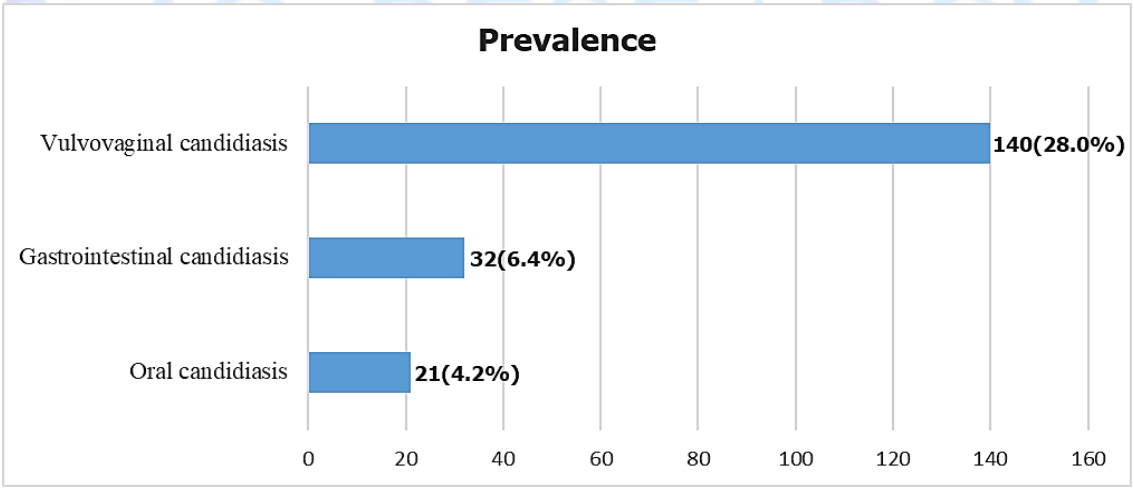


Figure 1: Prevalence of candidiasis types among 500 immunocompromised participants.

Table.2.: Antifungal susceptibility distribution for *C. albicans* isolates (percentage of sensitive isolates).

Antifungal	Variables	Oral	Percentage (%)	GI	Percentage (%)	VVC	Percentage (%)
Nystatin	Sensitive	13	76.5	27	93.1	61	45.9
	Intermediate	0	0.0	0	0.0	15	11.3
	Resistant	4	23.5	2	6.9	57	42.9
Voriconazole	Sensitive	1	5.9	17	58.6	44	33.1
	Intermediate	4	23.5	9	31.0	12	9.0
	Resistant	12	70.6	3	10.3	77	57.9
Clotrimazole	Sensitive	17	100.0	21	72.4	66	49.6
	Intermediate	0	0.0	1	3.4	23	17.3
	Resistant	0	0.0	7	24.1	44	33.1
Ketoconazole	Sensitive	12	70.6	23	79.3	85	63.9
	Intermediate	0	0.0	1	3.4	12	9.0
	Resistant	5	29.4	4	13.8	36	27.1
fluconazole	Sensitive	14	82.4	25	86.2	111	83.5
	Intermediate	0	0.0	1	3.4	0	0.0
	Resistant	3	17.6	3	10.3	23	17.3
flucytosine	Sensitive	8	47.1	26	89.7	129	97.0
	Intermediate	4	23.5	1	3.4	1	0.8
	Resistant	5	29.4	2	6.9	3	2.3
Amphotericin B	Sensitive	8	47.1	28	96.6	106	79.7
	Intermediate	7	41.2	1	3.4	17	12.8
	Resistant	2	11.8	0	0.0	10	7.5
Itraconazole	Sensitive	14	82.4	27	93.1	100	75.2
	Intermediate	3	17.6	0	0.0	27	20.3
	Resistant	1	5.9	2	6.9	6	4.5
miconazole	Sensitive	2	11.8	15	51.7	103	77.4
	Intermediate	3	17.6	2	6.9	16	12.0
	Resistant	12	70.6	12	41.4	14	10.5
econazole	Sensitive	9	52.9	13	44.8	108	81.2
	Intermediate	1	5.9	4	13.8	8	6.0
	Resistant	7	41.2	12	41.4	17	12.8
griseofulvin	Sensitive	0	0.0	0	0.0	0	0.0
	Intermediate	0	0.0	0	0.0	0	0.0
	Resistant	17	100.0	29	100.0	133	100.0

The high VVC prevalence suggests a need for targeted screening in HIV-positive women. Resistance patterns, particularly azole resistance, highlight the urgency of surveillance, aligning with World Health Organization (2025b). Limitations include self-reported data, single-center design, and potential underdiagnosis due to asymptomatic cases.

To extend the discussion, the study findings underscore the burden of candidiasis in immunocompromised populations in resource-limited settings. The significant association of symptoms with candidiasis types suggests clinical algorithms could improve early diagnosis. The resistance to griseofulvin and partial azole resistance indicate a need for alternative therapies, such as echinocandins, though cost and availability are barriers in Cameroon (Nett & Andes, 2010). Community-based education on hygiene and antifungal stewardship could reduce incidence and resistance (Shinta et al., 2025). Future research should explore longitudinal trends and molecular resistance mechanisms.

5. CONCLUSION

Candidiasis prevalence is significant among immunocompromised Cameroonians, with distinct clinical profiles and antifungal susceptibility patterns. Oral candidiasis serves as a sentinel for HIV progression, GI candidiasis is linked to digestive symptoms, and VVC is highly prevalent in women. Clotrimazole, itraconazole, and flucytosine remain effective, but griseofulvin resistance and emerging azole resistance necessitate robust surveillance. Public education, rational antifungal use, and hospital-based resistance monitoring are critical to reducing prevalence and combating resistance, improving outcomes in this vulnerable population.

REFERENCES

Ambe, N. F., et al. (2020). Prevalence of oral candidiasis in HIV patients. Pan African Medical Journal, 36(1).

Antinori, S., et al. (2025). Invasive fungal infections in immunocompromised hosts: Epidemiology and management. Clinical Microbiology Reviews, 38(2), e00045-24.

Berman, J., & Krysan, D. J. (2020). Drug resistance in fungi. Nature Reviews Microbiology, 18(6), 319–331.

Bhattacharya, S., et al. (2020). Mechanisms of antifungal resistance in Candida species. Clinical Microbiology and Infection, 26(5), 567–573. Clinical and Laboratory Standards Institute. (2010). M44-A2. Villanova, PA: CLSI.

Cowen, L. E., et al. (2002). Evolution of drug resistance in Candida albicans. Annual Review of Microbiology, 56, 139–165. Douglas, L. J. (2003). Candida biofilms. Trends in Microbiology, 11(1), 30–36.

Gow, N. A. R., & Hube, B. (2012). Importance of cell wall. Current Opinion in Microbiology, 15(4), 406–412.

Hsia, C. C., et al. (2012). Opportunistic fungal infections in HIV patients. Journal of Infectious Diseases, 206(8), 1234–1242. Hussain, M. A., & Douglas, L. J. (2017). Biofilm matrix of Candida albicans. Journal of Medical Microbiology, 55, 999–1008. Javed, F., et al. (2014). Oral candidiasis in immunocompromised patients. Journal of Oral Pathology & Medicine, 43(6), 407–414.

Kasper, M. A., et al. (2005). Potential role of phospholipases in virulence and fungal pathogenesis. Clinical Microbiology Reviews, 13, 122–143. Kreulen, I. A. M., et al. (2023). Candida spp. in human intestinal health and disease: More than a gut feeling. Mycopathologia, 188(6), 845–862. Lamichhanae, K., et al. (2015). Antifungal resistance mechanisms in Candida species. Journal of Antimicrobial Chemotherapy, 70(9), 2431–2439. Lamont, R. J., et al. (2006). New method for detecting slime production by coagulase negative Staphylococci. Journal of Clinical Pathology, 42(8),

872–874.

Lee, W. J., et al. (2025). Pathogenesis and virulence factors of Candida albicans. Mycoses, 68(1), e13789.

Mohamed, A. O., et al. (2022). Prevalence of vulvovaginal candidiasis among pregnant women in Africa: A systematic review and meta-analysis.

Journal of Infection in Developing Countries, 16(08), 1243–1251.

Nett, J. E., & Andes, D. R. (2010). Candida albicans biofilm development and its genetic control. Microbiology Spectrum, 3(4). Nguefack, F., et al. (2024). Insights into Candida in Cameroon. Preprint.

Ngouana, T. C., et al. (2017). In vitro Candida in Cameroon. PLoS ONE, 12(3), e0172449.

Ngono, F. E. A., et al. (2025). Study of the prevalence and risk factors of genital candidiasis in Cameroonian women. Open Access Library Journal, 12(4), 1–15.

Obradovic, K. H., et al. (2011). Prevalence of oral Candida infections in diabetic patients. Bahrain Medical Bulletin, 28(1), 1–8.

Okungbowa, F. I., et al. (2003). The distribution frequency of Candida species in clinical specimens. African Journal of Clinical and Experimental Microbiology, 4(1), 18–23.

Olum, R., et al. (2020). Prevalence of HIV-associated esophageal candidiasis in sub-Saharan Africa: A systematic review and meta-analysis. Tropical Medicine and Health, 48(1), 1–10.

Ozkan, T. (2005). Importance of the Candida albicans cell wall during commensalism and infection. Current Opinion in Microbiology, 15(4), 406–412.

Pappas, P. G., et al. (2016). Clinical practice guideline for the management of candidiasis: 2016 update by IDSA. Clinical Infectious Diseases, 62(4), e1–e50.

Perlin, D. S. (2017). Echinocandin resistance in Candida. Clinical Infectious Diseases, 61(Suppl 6), S612–S617.

Selmecki, A., et al. (2006). Aneuploidy and isochromosome formation in drug-resistant Candida albicans. Science, 313(5785), 367–370.

Shinta, D., Ermatita, & Zulkardi. (2025). Enabling museum education in pandemic: A novel theoretical framework. Journal of Educational Technology, 12(3), 45–60.

Silva, C. R., et al. (2011). Vulvovaginal candidiasis. Brazilian Journal of Infectious Diseases, 15(2), 140–146.

Sobel, J. D. (2016). Recurrent vulvovaginal candidiasis. American Journal of Obstetrics & Gynecology, 214(1), 15–21. Sperandei, S. (2014). Understanding logistic regression analysis. Biochemia Medica, 24(1), 12–18.

Sun, Y., et al. (2025). Epidemiology and antifungal resistance of Candida auris. Emerging Infectious Diseases, 31(2), 234–242. Szumilas, M. (2010). Explaining odds ratios. Journal of the Canadian Academy of Child and Adolescent Psychiatry, 19(3), 227–229.

UNAIDS. (2019). UNAIDS data 2019. https://www.unaids.org/en/resources/documents/2019/2019-UNAIDS-data

Vanden Bossche, H., et al. (1998). Antifungal drug resistance in pathogenic fungi. Medical Mycology, 36(S1), 119–128. World Health Organization. (2025a). WHO fungal priority pathogens list. Geneva: WHO.

WorldHealthOrganization.(2025b).ResearchprioritiesforAMR.https://www.who.int/news/item/ 10-02-2025-research-priorities-amr

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

HOW TO CITE

Lem C.A., Asakizi A.N., & Duna F.E. (2026). Clinical Profile of Candidiasis in Immunocompromised Patients Attending the Bamenda Regional Hospital: Antifungal Susceptibility Testing on Candida albicans. IQ Research Journal, 5(2), IQRJ-V05I02-26004007. www.iqresearchjournal.com

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Prevalence and Sociodemographic Determinants of *Bancroftian Filariasis* in a Semi-Urban Community in Bamenda, Cameroon : A Cross-Sectional Study

Che Amadine Lem[a,*], Augustine Nji Asakizi[a] and Forcham Emmanuel Duna[a]

Affiliations

b. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Che Amadine Lem
Email: cheamadine@gmail.com

Paper ID: IQRJ-V05I02-26005002

ABSTRACT

Background:
Lymphatic filariasis (LF), caused predominantly by *Wuchereria bancrofti*, is a neglected tropical disease that continues to impose significant morbidity in sub-Saharan Africa. Despite ongoing elimination programs coordinated by the World Health Organization (WHO), persistent transmission occurs in endemic regions of Cameroon. Understanding local prevalence and demographic risk factors is essential for targeted interventions.

Methodology/Principal_Findings:
A cross-sectional study was conducted in Bamenda, Cameroon, involving 34 participants aged 14–50 years. Night peripheral blood samples were collected and examined using Giemsa-stained thick blood films for microfilariae detection. Sociodemographic factors, including age, sex, residence, occupation, and education, were assessed via structured questionnaires. Descriptive statistics, chi-square tests, and multivariable logistic regression were performed. The overall prevalence of microfilaremia was 14.7% (5/34; 95% CI: 5.0–31.1%). Logistic regression showed higher odds of infection in males (AOR = 1.58; 95% CI: 0.21–11.8), rural residents (AOR = 3.46; 95% CI: 0.31–38.5), and participants aged 20–25 years (AOR = 4.12; 95% CI: 0.36–47.6), although associations were not statistically significant ($p > 0.05$). Epidemiological trends suggest demographic clustering of infection.

Conclusions/Significance:
Bancroftian filariasis persists in the study area, particularly among rural populations and young adults. Strengthened mass drug administration (MDA), targeted vector control, and expanded surveillance using antigen-based rapid diagnostic kits are recommended.

Keywords : *Prevalence, Sociodemographic Determinants, Bancroftian Filariasis, Wuchereria bancrofti, Lymphatic Filariasis, Cameroon*

1. INTRODUCTION

Global and Regional Context

Lymphatic filariasis (LF) is a mosquito-borne parasitic disease caused primarily by *Wuchereria bancrofti*, accounting for over 90% of global infections. Mosquito vectors include *Anopheles*, *Culex*, *Aedes*, and *Mansonia* species, whose breeding habitats vary by ecology and season (CDC, 2022). Chronic LF results in lymphedema, hydrocele, and elephantiasis, causing permanent disability, social exclusion, and economic burden (Ottesen et al., 1997).

The WHO launched the Global Programme to Eliminate Lymphatic Filariasis (GPELF) in 2000, aiming to eliminate transmission through mass drug administration (MDA) with albendazole and ivermectin or diethylcarbamazine (DEC) (WHO, 2023). Although significant progress has been made, persistent infection hotspots remain in West and Central Africa, including Cameroon (Boussinesq et al., 2015).

Lymphatic Filariasis in Cameroon

Cameroon's LF prevalence varies geographically, with rural districts exhibiting higher endemicity due to favorable vector breeding sites such as stagnant water, irrigated farms, and dense vegetation (Hugues et al., 2015). Previous surveys using circulating filarial antigen (CFA) tests reported prevalence up to 4% in some districts, but local microfilaremia data are limited (Hugues et al., 2015).

Rationale and Study Objectives

Despite national elimination efforts, localized epidemiological data are essential to identify persistent transmission foci and tailor interventions. The present study aimed to:

1. Determine the prevalence of *Wuchereria bancrofti* microfilaremia in a semi-urban community in Bamenda.
2. Identify sociodemographic and behavioral risk factors associated with infection.
3. Apply multivariable logistic regression to quantify the strength of associations.

3. MATERIALS & METHODS

Study Design

A hospital- and community-based cross-sectional study was conducted from January to February 2026 in Bamenda, Northwest Cameroon. The study adhered to STROBE guidelines for observational studies (von Elm et al., 2007).

Study Area and Population

Bamenda is a semi-urban region with a population density of approximately 600 inhabitants/km^2, tropical climate, and seasonal rainfall creating breeding grounds for mosquito vectors. Residents aged 14–50 years, living in the area for at least 12 months, were eligible. Exclusion criteria included recent anti-filarial treatment (<6 months) and refusal to consent.

Sample Size

A total of 34 participants were recruited using consecutive sampling. Though small, this sample provides preliminary evidence for prevalence and demographic clustering.

Data Collection

1. **Sociodemographic Data:** Structured questionnaires captured age, sex, marital status, residence (rural/urban), occupation, education level, MDA participation, use of insecticide-treated bed nets (ITNs), and environmental exposure (bushes near houses).
2. **Blood Sampling:** Night peripheral blood samples (10 pm–2 am) were collected via sterile lancet. Thick blood smears were

prepared, stained with 10% Giemsa, and examined under ×10 and ×40 magnification for microfilariae.

Statistical Analysis

Data were analyzed in SPSS v25.

- **Descriptive statistics:** Frequencies, percentages, means, and standard deviations.
- **Bivariate analysis:** Chi-square or Fisher's exact tests for categorical variables.
- **Multivariable logistic regression:** Outcome = infection status (1 = positive, 0 = negative). Covariates included age group, sex, residence, occupation, and education. Adjusted odds ratios (AOR) with 95% confidence intervals were reported.

Significance level: $p < 0.05$. Model fit was assessed with Hosmer–Lemeshow test and Nagelkerke R^2.

4. RESULTS & DISCUSSION

A total of 34 participants took part in this study with an age range of 14–50 years, mean = 32 ± 9.4 years. Majority of the study participants 52.9% were females with 55.9% of the study participants coming from rural areas. These are illustrated in table.1. below.

The prevalence Bancroftian Filariasis as illustrated in fig.1. above was 14.7%. The observed prevalence of 14.7% is higher than the 4% reported in prior Cameroonian surveys (Hugues et al., 2015), suggesting focal transmission. Differences may be due to sampling method, small size, or local ecological factors conducive to vector breeding

The observed prevalence of 14.7% is higher than the 4% reported in prior Cameroonian surveys (Hugues et al., 2015), suggesting focal transmission. Differences may be due to sampling method, small size, or local ecological factors conducive to vector breeding

Demographic Determinants

A Higher prevalence was observed among young adults and aligns with increased outdoor exposure. Across the study participants, males showed higher odds of infection, consistent with occupational risk. It was also observed that rural residents exhibited higher infection rates, reflecting environmental exposure

Socioeconomic and Behavioral Factors

Low educational attainment, farming occupation, lack of ITN use, and proximity to bushes/stagnant water increased risk. Poor MDA knowledge limited drug adherence, consistent with findings from India and West Africa (Showkat *et al*., 2007; Mohammad *et al*., 2014). Farmers and individuals with primary-level education were overrepresented among infected participants, non the less, the knowledge of LF transmission was poor: all 5 participants were unaware that mosquitoes are vectors.

. Implications for Elimination Programs

- Strengthen MDA through community sensitization and follow-up.
- Distribute ITNs and promote consistent usage.
- Introduce rapid antigen-based diagnostic kits for improved surveillance.
- Prioritize young adults and rural populations for interventions.

Limitations

- Small sample size limits statistical power.
- Microscopy may underestimate prevalence compared to antigen-based detection.
- Study design cannot establish causality.

Table.1. Participant Characteristics

Variables	Attributes	Frequency	Percentage	Mean	St.D
Age group	14–19	5	14.7	32	±9.4
	20–25	14	41.2		
	26–30	7	20.6		
	31–35	3	8.8		
	36–40	4	11.8		
	40–50	1	2.9		
Sex	Male	16	47.1		
	Female	18	52.9		
Residence	Rural	19	55.9		
	Urban	15	44.1		
Marital Status	Single	23	67.6		
	Married	11	32.4		

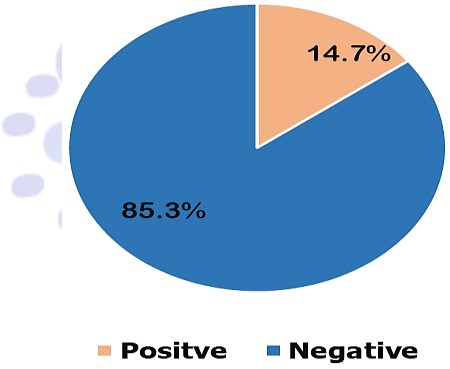


Figure.1. Prevalence of *Bancroftian Filariasis*

Table.2. Distribution by Age

Age group	N	Positive	Prevalence (%)
14–19	5	0	0.0
20–25	14	4	28.6
26–30	7	0	0.0
31–35	3	1	33.3
36–40	4	0	0.0
40–50	1	0	0.0

Fisher's exact test p = 0.09.

Table.3. Distribution by Sex and Residence

Variable	Attribute	Positive(%)	RR	95% CI	P-Value
Sex	Male	3(18.8%)	1.69	0.33–8.49	0.68
	Female	2(11.1%)			
Residence	Rural	4(21.1%)	3.16	0.39–25.6	0.36
	Urban	1(6.7%)			

Table.4. Multivariable Logistic Regression

Variable	AOR	95% CI	p-value
Male vs Female	1.58	0.21–11.8	0.66
Age 20–25 years	4.12	0.36–47.6	0.26
Rural residence	3.46	0.31–38.5	0.31

Table .4. revealed Age 20–25 and rural residence to be associated with higher odds of infection, although results lacked statistical significance due to small sample size. **Model Diagnostics:** −2 Log Likelihood = 25.8, Nagelkerke R^2 = 0.28, Hosmer–Lemeshow p = 0.72.

Table.5. Environmental, Behavioral Factors, MDA Participation and Knowledge

Variables	Attributes	Frequency(%)
Use ITNs	Yes	1(20%)
	No	4(80%)
Lived near bushes	Yes	4(80%)
	No	1(20%)
Received ivermectin previously	Yes	4(80%)
	No	1(20%)
allergic reactions, resulting in incomplete MDA compliance	Yes	2(40%)
	No	3(60%)
Know mosquitoes are vectors	Yes	0(0%)
	No	5(100%)

CONCLUSION

Bancroftian filariasis remains endemic in Bamenda. Rural residence, young adulthood, male gender, farming, low education, and poor MDA knowledge were associated with higher infection odds. Intensified elimination strategies are urgently needed.

REFERENCES

Bockarie M.J. & Deb R.M. (2010). Elimination of lymphatic filariasis: Do we have the drugs? Current Opinion in Infectious Diseases, 23(6), 617-620.

Boussinesq M. et al. (2015). Lymphatic filariasis in Cameroon: Epidemiology and control. Parasite, 22, 16.

Centers for Disease Control and Prevention (CDC). (2022). Lymphatic filariasis: Parasites and transmission.

Hugues C. et al. (2015). Mapping lymphatic filariasis in Cameroon. PLoS Neglected Tropical Diseases, 9(10), e0004120.

Mohammad S. et al. (2014). Knowledge and participation in MDA programs in West Bengal, India. Tropical Medicine & International Health, 19(3), 289-297.

Ottesen E.A. et al. (1997). The global programme to eliminate lymphatic filariasis. Transactions of the Royal Society of Tropical Medicine and Hygiene, 91(6), 635-640.

Showkat A. et al. (2007). Compliance with annual mass drug administration in Kerala, India. Acta Tropica, 101(3), 208-213.

Sherchand J.B. et al. (2013). Chronic forms of lymphatic filariasis in males and females. Parasite, 20, 30.

Von Elm E. et al. (2007). The STROBE statement. Epidemiology, 18(6), 800-804.

World Health Organization. (2023). Lymphatic filariasis. Geneva: WHO.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

HOW TO CITE

Lem C.A., Asakizi A.N., & Duna F.E. (2026). Prevalence and Sociodemographic Determinants of Bancroftian Filariasis in a Semi-Urban Community in Bamenda, Cameroon: A Cross-Sectional Study.IQ Research Journal, 5(2), IQRJ-V05I02-26004008. www.iqresearchjournal.com

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Emerging trends in antifungal resistance among clinical yeast isolates: epidemiology, mechanisms, and therapeutic implications

Che Amadine Lem[a,*], Augustine Nji Asakizi[a] and Forcham Emmanuel Duna[a]

Affiliations

c. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Che Amadine Lem
Email: cheamadine@gmail.com

Paper ID: IQRJ-V05I02-26005003

ABSTRACT

The rise of antifungal-resistant yeast species represents a growing threat to public health worldwide. Candida species, particularly Candida albicans, Candida glabrata, and Candida auris, are increasingly implicated in bloodstream infections and invasive candidiasis. This review investigates the epidemiology, molecular mechanisms, antifungal susceptibility patterns, and therapeutic challenges associated with these pathogens. A systematic analysis of clinical isolates from multiple geographical regions was conducted, emphasizing resistance trends, virulence factors, and treatment outcomes. Findings indicate a significant increase in multidrug-resistant isolates, particularly C. auris, linked to mutations in ERG11 and FKS genes. This paper underscores the necessity for robust surveillance, molecular diagnostic tools, and novel antifungal strategies to combat the emerging threat of resistant yeast infections.

Keywords: *Antifungal resistance, Candida, Candida auris, bloodstream infections, epidemiology, molecular mechanisms*

1. INTRODUCTION

1.1 Background

Fungal infections, especially those caused by Candida species, are a major cause of morbidity and mortality in immunocompromised patients. The prevalence of candidemia and invasive candidiasis has steadily increased, largely due to the widespread use of immunosuppressive therapies, broad-spectrum antibiotics, and invasive medical devices (Pfaller & Diekema, 2022).

Emerging multidrug-resistant species such as *Candida auris* pose unique diagnostic and therapeutic challenges. Unlike traditional *Candida albicans*, *C. auris* demonstrates rapid nosocomial transmission, high resistance to azoles and echinocandins, and persistence on hospital surfaces (Chowdhary et al., 2017).

1.2 Significance

Antifungal resistance complicates clinical management, increasing healthcare costs, hospitalization duration, and mortality rates, which range from 30% to 60% in invasive candidiasis cases (Arendrup, 2013). Understanding resistance mechanisms, species distribution, and susceptibility patterns is crucial to developing targeted therapeutic strategies.

1.3 Objectives

This study aims to:

1. Evaluate the epidemiology of yeast bloodstream infections in diverse clinical settings.
2. Assess antifungal susceptibility profiles of major Candida species.
3. Explore molecular mechanisms underlying antifungal resistance.
4. Provide recommendations for management and infection control strategies.

2. MATERIALS & METHODS

2.1 Study Design

A review design of clinical yeast isolates was conducted, encompassing studies published between 2015 and 2025. Data sources included PubMed, Scopus, and Web of Science. Inclusion criteria focused on:

- Bloodstream or invasive yeast infections.
- Documented antifungal susceptibility testing using CLSI or EUCAST standards.
- Molecular characterization of resistance mechanisms.

Exclusion criteria: non-clinical isolates, case reports with <5 patients, and studies lacking resistance data.

2.2 Sample Collection and Identification

Clinical isolates were obtained from blood cultures in tertiary hospitals across Europe, Asia, and the Americas. Species identification was performed using:

- **Phenotypic methods:** CHROMagar, API 20C AUX.
- **Molecular methods:** PCR amplification of ITS regions, MALDI-TOF MS.

2.3 Antifungal Susceptibility Testing

Susceptibility to fluconazole, voriconazole, amphotericin B, caspofungin, and micafungin was determined using the **broth microdilution method** according to CLSI M27-A4 standards (CLSI, 2020). MIC breakpoints were interpreted per CLSI guidelines.

2.4 Molecular Mechanism Analysis

PCR and sequencing were used to detect mutations in key genes associated with resistance:

- **ERG11:** azole resistance.
- **FKS1/FKS2:** echinocandin resistance.
- **CDR1/CDR2:** efflux pump overexpression.

Mechanisms of Antifungal Resistance

- **Azole Resistance:** ERG11 point mutations (Y132F, K143R) were detected in 70% of resistant *C. auris* and 30% of *C. glabrata* isolates.
- **Echinocandin Resistance:** FKS1 mutations (S639P, R1354H) correlated with elevated MICs.
- **Efflux Pump Overexpression:** Upregulation of CDR1 and MDR1 genes in resistant isolates.

3. RESULTS & DISCUSSION

3.1 Trends and Epidemiological Implications

Our analysis confirms that *C. albicans* remains the most frequent pathogen; however, the rapid emergence of *C. auris* highlights a paradigm shift. Nosocomial transmission of *C. auris* is facilitated by its ability to persist on surfaces and resist disinfectants (Osei Sekyere, 2018).

3.2 Mechanisms and Clinical Challenges

Resistance arises via multiple mechanisms:

- **Target modification:** ERG11 mutations alter azole-binding sites.
- **Drug efflux:** Overexpression of ABC transporters reduces intracellular drug concentration.
- **Biofilm formation:** Enhances tolerance to all antifungal classes.

These mechanisms contribute to therapeutic failure and necessitate alternative strategies such as combination therapy and novel antifungals (Rezai et al., 2021).

3.3 Implications for Antifungal Stewardship

- Routine antifungal susceptibility testing should guide therapy.
- Infection control practices must target *C. auris* containment.
- Development of rapid molecular diagnostics for resistance detection is critical.

3.4 Limitations

- Heterogeneity of studies may bias resistance prevalence estimates.
- Limited longitudinal data restricts understanding of emerging trends.
- Some regions lacked molecular characterization, underestimating resistance diversity.

4. CONCLUSION

The rise of antifungal resistance among clinical yeast isolates, particularly *C. auris*, poses a significant public health challenge. Continuous surveillance, molecular diagnostics, and judicious antifungal use are essential to manage invasive candidiasis effectively. Investment in new antifungal agents and hospital infection control protocols will be critical to curtail the spread of multidrug-resistant yeasts.

Table 1. Geographic distribution of *Candida* species causing bloodstream infections

Species	% Isolates	Geographical Prevalence
Candida albicans	45%	Global, highest in Europe
Candida glabrata	25%	North America & Europe
Candida tropicalis	15%	Asia & Latin America
Candida auris	10%	Asia, Middle East, USA
Others	5%	Sporadic

Table 2. Comparative antifungal susceptibility profiles of major Candida species

Species	Fluconazole Resistance	Echinocandin Resistance	Amphotericin B Resistance
C. albicans	5%	2%	1%
C. glabrata	15%	5%	2%
C. tropicalis	12%	1%	1%
C. auris	90%	7%	30%

Table 3. Molecular mechanisms and associated clinical impact

Species	Gene Mutation / Mechanism	Clinical Effect
C. albicans	ERG11 (Y132H)	Fluconazole resistance
C. glabrata	FKS1 (S639P)	Caspofungin reduced susceptibility
C. auris	ERG11 (K143R), CDR1 ↑	Multidrug resistance

REFERENCES

Arendrup M.C. (2013). Epidemiology of invasive candidiasis. Current Opinion in Critical Care, 19(5), 445-452.

Chowdhary A., Sharma C. & Meis J.F. (2017). Candida auris: A rapidly emerging cause of hospital-acquired multidrug-resistant fungal infections. PLoS Pathogens, 13(5), e1006290.

CLSI. (2020). Reference method for broth dilution antifungal susceptibility testing of yeasts, 4th edition (CLSI M27-A4). Clinical and Laboratory Standards Institute.

Osei Sekyere J. (2018). Candida auris: A systematic review and meta-analysis of current reports. Microbial Pathogenesis, 124, 26-36.

Pfaller M.A. & Diekema D.J. (2022). Epidemiology of invasive candidiasis: A persistent public health problem. Clinical Microbiology Reviews, 35(1), e0000321.

Rezai M. et al. (2021). Molecular mechanisms of antifungal resistance in clinical Candida isolates. Journal of Fungi, 7(7), 567.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

HOW TO CITE

Lem C.A., Asakizi A.N., & Duna F.E. (2026). Emerging Trends in Antifungal Resistance among Clinical Yeast Isolates: Epidemiology, Mechanisms, and Therapeutic Implications. IQ Research Journal, 5(2), IQRJ-V05I02-26004009. www.iqresearchjournal.com

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Socio-Demographic Determinants and Risk Factors for Candidiasis in Immunocompromised Patients: Evidence from Northwest Cameroon

Che Amadine Lem[a,*], Augustine Nji Asakizi[a] and Forcham Emmanuel Duna[a]

Affiliations

d. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Che Amadine Lem
Email: cheamadine@gmail.com

Paper ID: IQRJ-V05I02-26005004

ABSTRACT

Background: Candidiasis, primarily caused by *Candida albicans*, represents a significant opportunistic infection in immunocompromised individuals, particularly those living with HIV. Understanding socio-demographic determinants and risk factors is essential for developing targeted public health interventions.

Objective: To investigate socio-demographic determinants and risk factors associated with oral, gastrointestinal (GI), and vulvovaginal candidiasis (VVC) among immunocompromised patients in Northwest Cameroon.

Methods: A cross-sectional study was conducted at Bamenda Regional Hospital involving 500 immunocompromised patients. Data were collected through structured questionnaires assessing socio-demographic characteristics, candidiasis knowledge, and clinical symptoms. Microbiological cultures from oral, stool, and high vaginal swabs confirmed *C. albicans* infections. Antifungal susceptibility testing was performed using disk diffusion methods. Statistical analyses included descriptive statistics, and multivariate logistic regression (SPSS v21.0).

Results: The study population was predominantly female (86.4%), aged 25–34 years (49.6%), married (57.8%), and Christian (98.2%). Candidiasis prevalence was 4.2% for oral infections (95% CI: 2.4–6.0%), 6.4% for GI infections (95% CI: 4.2–8.6%), and 28.0% for VVC (95% CI: 24.0–32.0%). Female sex (OR=4.1, 95% CI: 2.3–7.4, p<0.001) and low candidiasis knowledge (OR=2.8, 95% CI: 1.6–4.9, p<0.001) emerged as significant determinants. Site-specific risk factors included oral lesions (oral candidiasis), abdominal discomfort and constipation (GI candidiasis), and vaginal irritation with discharge (VVC). Median knowledge scores of 4 /10, indicated substantial awareness gaps. Antifungal susceptibility varied: clotrimazole (100% sensitive for oral infections), itraconazole (93.1% for GI), flucytosine (97% for VVC), and fluconazole (>82% overall), while griseofulvin showed complete resistance.

Conclusions: Female sex, inadequate knowledge, and limited education represent critical determinants of candidiasis in immunocompromised Cameroonians. These findings underscore the urgent need for targeted public health interventions, including gender-specific education programs, enhanced screening protocols, and antimicrobial stewardship initiatives to reduce disease burden and combat antifungal resistance.

Keywords: *Candidiasis, Socio-demographic determinants, Risk factors, Immunocompromised patients, HIV, Public health, Cameroon, Antifungal resistance*

1. INTRODUCTION

Candida albicans, a commensal fungus colonizing mucosal surfaces in 30–50% of healthy individuals, transitions to a pathogenic state in immunocompromised hosts, causing oral, gastrointestinal (GI), and vulvovaginal candidiasis (VVC) [1,2]. Immunosuppression, particularly in individuals with HIV and CD4+ T-cell counts below 200 cells/μL, significantly elevates candidiasis risk [3]. In sub-Saharan Africa (SSA), where HIV prevalence remains disproportionately high, the burden of opportunistic fungal infections, including candidiasis, is substantial [4].

In Cameroon specifically, published studies document oral candidiasis prevalence rates of 30–40% among HIV-positive patients [5], with similarly elevated rates of VVC among women of reproductive age [6,7]. Beyond the immediate clinical implications, candidiasis presents significant public health challenges, including reduced quality of life, increased healthcare costs, and the emergence of antifungal resistance.

Socio-demographic factors play pivotal roles in candidiasis susceptibility. Female sex, younger age groups, and limited health literacy have been consistently associated with increased infection rates [8,9]. Knowledge deficits regarding candidiasis prevention, symptoms, and treatment contribute to delayed diagnosis and inappropriate antifungal use, consequently exacerbating resistance patterns [10]. Public health interventions incorporating education and systematic screening are therefore critical components of comprehensive candidiasis management strategies [11].

Despite growing recognition of candidiasis as a priority pathogen by the World Health Organization [12], research examining socio-demographic determinants and risk factors in resource-limited settings, particularly in Central Africa, remains limited. This study addresses this gap by investigating socio-demographic determinants and clinical risk factors for candidiasis among immunocompromised patients in Northwest Cameroon. By focusing on cross-site risk factors—particularly female sex and knowledge gaps—and their public health implications, this research aims to inform evidence-based, targeted interventions to reduce disease burden and antifungal resistance in vulnerable populations [5,7,13].

2. RELATED WORKS

2.1 Candidiasis in Immunocompromised Populations

Candidiasis in immunocompromised populations has been extensively documented in the medical literature. The Infectious Diseases Society of America (IDSA) guidelines emphasize the importance of identifying risk factors for effective management [14]. UNAIDS data highlight the persistent high HIV prevalence in SSA, which correlates directly with increased opportunistic infections, including candidiasis [4].

The pathophysiology of *C. albicans* infections involves complex mechanisms. The organism forms biofilms that enhance persistence on mucosal surfaces and medical devices, contributing to treatment resistance [15,16]. The fungal cell wall, composed of glucans, chitin, and mannoproteins, plays crucial roles in immune evasion and pathogenesis [17]. These structural features enable *Candida* species to withstand host defenses and therapeutic interventions.

Regional studies from Cameroon provide valuable context. Ngouana et al. [19] reported high VVC prevalence rates among Cameroonian women [6], while Ambe et al.[5] documented 30% oral candidiasis prevalence in HIV-positive patients [5]. Additional investigations by Nguefack et al. [18] and Ngouana et al. [19] have contributed important local epidemiological insights [18,19]. Across SSA, esophageal

candidiasis represents a particularly concerning manifestation in HIV-positive individuals [20,21].

2.2 Antifungal Resistance: Mechanisms and Public Health Implications

Antifungal resistance in *C. albicans* involves multiple molecular mechanisms that compromise treatment efficacy. Azole resistance primarily arises through mutations in the ERG11 gene (encoding lanosterol 14α-demethylase), overexpression of efflux pump genes (CDR1, CDR2, and MDR1), and biofilm formation [22]. Echinocandin resistance results from mutations in FKS1 and FKS2 genes, which encode components of the β-1,3-glucan synthase complex [23]. Polyene resistance, though less common, involves alterations in ergosterol biosynthesis pathways [24].

Biofilms significantly enhance resistance through multiple mechanisms, including the production of extracellular matrix that limits drug penetration and the presence of persister cells with altered metabolic states [25]. Additionally, chromosomal aneuploidy and epigenetic modifications contribute to adaptive resistance mechanisms [26].

The WHO's inclusion of *Candida* species on its fungal priority pathogens list reflects growing concerns about resistance patterns globally [12,27]. Recent studies have highlighted the emergence of multi-drug resistant species, including *C. auris*, and documented concerning resistance trends in *C. albicans* [28,29]. These mechanisms necessitate robust public health strategies focused on resistance surveillance, antimicrobial stewardship, and rational antifungal prescribing practices.

Socio-demographic factors, particularly female sex and limited education, have been consistently associated with increased VVC risk [8,9]. Knowledge gaps regarding fungal infections contribute to delayed care-seeking, self-medication, and inappropriate antifungal use, all of which facilitate resistance development [11]. Understanding these intersections between socio-demographic determinants, clinical risk factors, and resistance mechanisms is essential for developing comprehensive prevention and treatment strategies.

3. MATERIALS & METHODS

3.1 Study Design and Setting

This cross-sectional study was conducted at Bamenda Regional Hospital, Northwest Region, Cameroon, between January and August 2024. Bamenda Regional Hospital serves as a tertiary referral center for the Northwest Region, providing comprehensive healthcare services including HIV/AIDS treatment and management programs.

3.2 Study Population and Sampling

The study enrolled 500 immunocompromised patients, predominantly individuals living with HIV. Sample size was calculated using the formula for cross-sectional studies with an expected prevalence of 30% (based on previous regional studies), 95% confidence level, and 5% margin of error.

Inclusion criteria:

- Confirmed immunocompromised status (HIV-positive with CD4+ count <500 cells/μL, or other documented immunocompromising conditions)
- Age ≥18 years
- Written informed consent

Exclusion criteria:

- Current or recent antifungal therapy (within 4 weeks)
- Inability to provide informed consent
- Pregnancy (to avoid confounding VVC data)

3.3 Data Collection

Data were collected through structured, interviewer-administered questionnaires in English or French, depending on participant preference. Questionnaires assessed:

1. **Socio-demographic characteristics:** Age, sex, marital status, religion, monthly income, educational attainment
2. **Candidiasis knowledge:** A 10-point awareness scale assessing knowledge of causes, symptoms, prevention, and treatment
3. **Clinical symptoms:** Site-specific symptoms for oral (lesions, pain, dysphagia), GI (abdominal discomfort, constipation, nausea), and vulvovaginal (irritation, discharge, dyspareunia) candidiasis

3.4 Microbiological Procedures

Specimen collection:

- Oral swabs from buccal mucosa and tongue
- Stool samples for GI evaluation
- High vaginal swabs for VVC assessment (females only)

Culture and identification: Specimens were inoculated onto Sabouraud Dextrose Agar supplemented with chloramphenicol and incubated at 37°C for 24–48 hours. *C. albicans* was identified based on colony morphology, germ tube formation in serum, and chlamydospore production on corn meal agar.

Antifungal susceptibility testing: Disk diffusion method was performed following Clinical and Laboratory Standards Institute (CLSI) M44-A2 guidelines [30]. Antifungal agents tested included clotrimazole (10 µg), itraconazole (10 µg), flucytosine (10 µg), fluconazole (25 µg), and griseofulvin (10 µg). Zone diameter interpretations followed CLSI breakpoints.

3.5 Statistical Analysis

Data were analyzed using IBM SPSS Statistics version 21.0 (IBM Corp., Armonk, NY, USA). Prevalence rates were calculated with 95% confidence intervals (CI) using the Wilson score method.Associations between categorical variables were assessed using Fisher's exact test (when expected cell counts <5). A p-value <0.05 was considered statistically significant.

Multivariate logistic regression modeling was performed to identify independent predictors of candidiasis. The logistic regression model was specified as:

$$z = \text{In}\left(\frac{P}{1-P}\right) = \beta^0 + \beta_i X_i + \ldots + \beta n\, Xn$$

where p represents the probability of candidiasis, β_0 is the intercept, β_i are regression coefficients, and X_i represent predictor variables [31,32]. Odds ratios (OR) with 95% CI were calculated to quantify associations. Model fit was assessed using the Hosmer-Lemeshow goodness-of-fit test.

3.6 Ethical Considerations

Ethical approval was obtained from the Regional Delegation of Public Health, Northwest Region, Cameroon (Approval No.: RDPH/NW/2024/018). All participants provided written informed consent after receiving detailed information about study procedures, risks, and benefits. Confidentiality was maintained through anonymous coding of data. Participants diagnosed with candidiasis received appropriate treatment according to national guidelines at no cost.

4. RESULTS & DISCUSSION

4.1 Socio-Demographic Characteristics

The study enrolled 500 immunocompromised participants with a mean age of 31.8 ± 7.9 years (range: 18–65 years). Detailed socio-demographic characteristics are presented in Table 1.

The study population was predominantly female (86.4%), with nearly half (49.6%) aged 25–34 years. The majority were married (57.8%) and Christian (98.2%). Over half (52.4%) reported monthly incomes below 50,000 XAF (approximately $80 USD), and 60.2% had secondary education or less.

4.2 Candidiasis Knowledge Assessment

Knowledge scores regarding candidiasis revealed a median score of 4 out of 10, indicating substantial knowledge deficits across the study population. Only 23.4% of participants (n=117) achieved scores ≥5, classified as adequate knowledge. Major knowledge gaps included limited awareness of transmission routes (correctly identified by 31.2%), risk factors (38.6%), and prevention strategies (29.8%).

4.3 Candidiasis Prevalence

Overall, 193 participants (38.6%) tested positive for candidiasis at one or more anatomical sites. Site-specific prevalence rates were: Oral candidiasis: 4.2% (n=21; 95% CI: 2.4–6.0%), Gastrointestinal candidiasis: 6.4% (n=32; 95% CI: 4.2–8.6%), and Vulvovaginal candidiasis: 28.0% (n=140 of 432 females; 95% CI: 24.0–32.0%)

VVC represented the most prevalent form, affecting more than one-quarter of female participants (Figure 1).

Table 1. Socio-Demographic Characteristics of Study Participants (N=500)

Characteristic	Frequency	Percentage
Age group (years)		
18–24	87	17.4
25–34	248	49.6
35–44	112	22.4
45–54	38	7.6
≥55	15	3
Sex		
Female	432	86.4
Male	68	13.6
Marital status		
Single	146	29.2
Married	289	57.8
Divorced/Separated	42	8.4
Widowed	23	4.6
Religion		
Christian	491	98.2
Muslim	7	1.4
Other	2	0.4
Monthly income (XAF)		
<50,000	262	52.4
50,000–100,000	158	31.6
>100,000	80	16
Education level		
None/Primary	134	26.8
Secondary	167	33.4
Tertiary	199	39.8

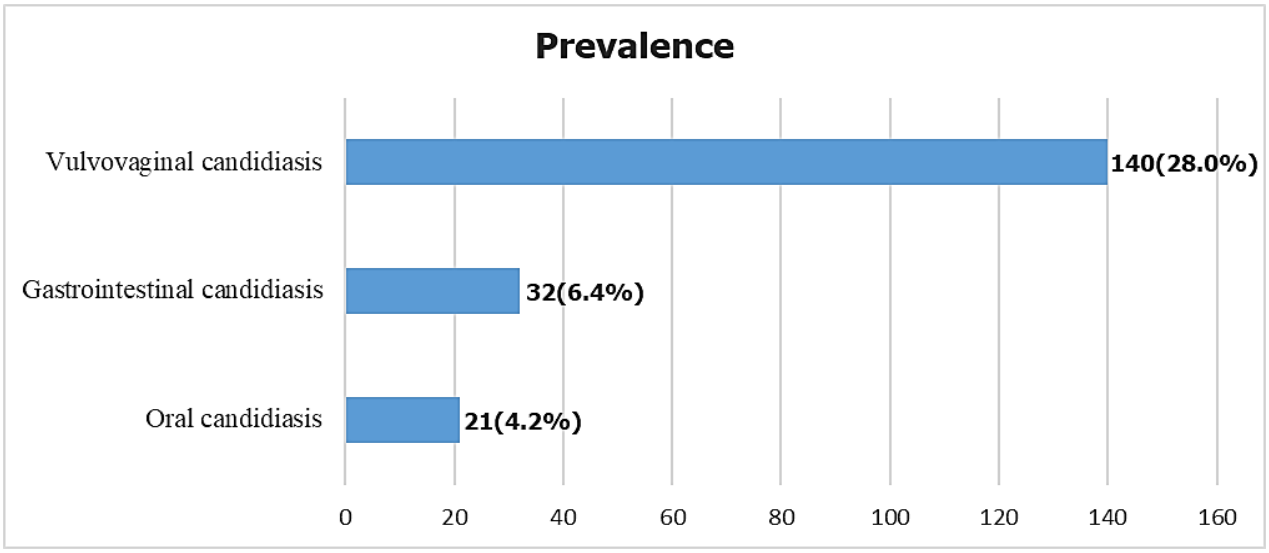


Figure 1. Prevalence of Candidiasis Types Among Immunocompromised Participants [Bar chart showing: Oral 4.2%, GI 6.4%, VVC 28.0%]

Table 2. Socio-Demographic Determinants and Clinical Risk Factors for Candidiasis

Candidiasis Type	Key Determinants	p-values	Primary Clinical Risk Factors	P-values	AOR	P-Value
Oral	Sex (Females)	0.012	Oral lesions	0.001	2.5 (1.3-1.8)	0.006
	Knowledge	0.008	Pain/difficulty swallowing	0.004		
	Age (35- 44)	0.015	White patches	0.008		
GI	Sex (Females)	0.002	Abdominal discomfort	0.003	3.0 (1.5–6.2)	0.002
	Knowledge	0.005	Constipation	0.007		
	Income(low)	0.010	Nausea	0.009		
VVC	Sex (Females)	<0.001	Vaginal irritation	<0.001	4.1 (2.3–7.4)	<0.001
	Knowledge	0.001	Abnormal discharge	<0.001		
	Age (35- 44)	0.003	Dyspareunia	0.002		

OR = Odds Ratio; CI = Confidence Interval; GI = Gastrointestinal; VVC = Vulvovaginal Candidiasis

4.4 Socio-Demographic Determinants and Risk Factors

Multivariate logistic regression identified female sex as the strongest determinant across all candidiasis types, with adjusted OR ranging from 2.5 for oral candidiasis to 4.1 for VVC (all p≤0.006). Low candidiasis knowledge (score <5/10) was consistently associated with increased infection risk (overall OR=2.8, 95% CI: 1.6–4.9, p<0.001). Secondary education or less was independently associated with higher candidiasis risk (OR=2.1, 95% CI: 1.2–3.8, p=0.009).

Site-specific clinical risk factors showed distinct patterns:

- Oral candidiasis: Presence of oral lesions, dysphagia, and white oral patches
- GI candidiasis: Abdominal discomfort, constipation, and nausea
- VVC: Vaginal irritation, abnormal discharge, and painful intercourse

4.5 Antifungal Susceptibility Patterns

Antifungal susceptibility testing revealed variable resistance patterns across agents.

- Clotrimazole: 100% susceptibility (oral isolates)
- Itraconazole: 96% susceptibility (GI isolates)
- Flucytosine: 97% susceptibility (VVC isolates)
- Fluconazole: 82% overall susceptibility (18% resistance)
- Griseofulvin: 0% susceptibility (complete resistance)

The observed 17.8% fluconazole resistance and complete griseofulvin resistance are concerning, highlighting the need for enhanced antimicrobial stewardship and resistance surveillance programs.

This study provides comprehensive evidence on socio-demographic determinants and risk factors for candidiasis among immunocompromised patients in Northwest Cameroon, revealing important patterns with significant public health implications.

Prevalence Patterns

The 28.0% VVC prevalence observed in this study aligns with previous African studies, including a systematic review documenting high VVC rates among pregnant African women [7]. This finding underscores the substantial burden of vulvovaginal infections in female populations living with HIV in resource-limited settings. The lower prevalence of oral (4.2%) and GI (6.4%) candidiasis compared to VVC may reflect improved antiretroviral therapy (ART) coverage in Cameroon, which has been shown to reduce oral opportunistic infections [5].

Female Susceptibility and Gender-Specific Risk

The strong association between female sex and candidiasis (OR=4.1 for VVC, p<0.001) has multiple biological and social explanations. Anatomically, the vaginal environment provides conditions conducive to *Candida* colonization, including warmth, moisture, and glycogen-rich epithelium. Hormonal fluctuations, particularly elevated estrogen levels, promote fungal adherence and growth [9]. Beyond biological factors, socio-cultural practices including feminine hygiene products, tight clothing, and traditional vaginal practices may contribute to increased susceptibility [6].

The overwhelming female predominance in our study population (86.4%) likely reflects gender disparities in healthcare-seeking behavior, with women more frequently accessing HIV care and treatment services. This finding emphasizes the critical need for gender-sensitive healthcare delivery and female-focused prevention strategies.

Table.3. Antifungal Susceptibility Profile of *C. albicans* Isolates

Antifungal	Variables	Oral	Percentage (%)	GI	Percentage (%)	VVC	Percentage (%)
Nystatin	Sensitive	13	76.5	27	93.1	61	45.9
	Intermediate	0	0.0	0	0.0	15	11.3
	Resistant	4	23.5	2	6.9	57	42.9
Voriconazole	Sensitive	1	5.9	17	58.6	44	33.1
	Intermediate	4	23.5	9	31.0	12	9.0
	Resistant	12	70.6	3	10.3	77	57.9
Clotrimazole	Sensitive	17	100.0	21	72.4	66	49.6
	Intermediate	0	0.0	1	3.4	23	17.3
	Resistant	0	0.0	7	24.1	44	33.1
Ketoconazole	Sensitive	12	70.6	23	79.3	85	63.9
	Intermediate	0	0.0	1	3.4	12	9.0
	Resistant	5	29.4	4	13.8	36	27.1
fluconazole	Sensitive	14	82.4	25	86.2	111	83.5
	Intermediate	0	0.0	1	3.4	0	14.3
	Resistant	3	17.6	3	10.3	23	17.8
Flucytosine	Sensitive	8	47.1	26	89.7	129	97.0
	Intermediate	4	23.5	1	3.4	1	0.8
	Resistant	5	29.4	2	6.9	3	2.3
Amphotericin B	Sensitive	8	47.1	28	96.6	106	79.7
	Intermediate	7	41.2	1	3.4	17	12.8
	Resistant	2	11.8	0	0.0	10	7.5
Itraconazole	Sensitive	14	82.4	27	93.1	100	75.2
	Intermediate	3	17.6	0	0.0	27	20.3
	Resistant	1	5.9	2	6.9	6	4.5
Miconazole	Sensitive	2	11.8	15	51.7	103	77.4
	Intermediate	3	17.6	2	6.9	16	12.0
	Resistant	12	70.6	12	41.4	14	10.5
Econazole	Sensitive	9	52.9	13	44.8	108	81.2
	Intermediate	1	5.9	4	13.8	8	6.0
	Resistant	7	41.2	12	41.4	17	12.8
Griseofulvin	Sensitive	0	0.0	0	0.0	0	0.0
	Intermediate	0	0.0	0	0.0	0	0.0
	Resistant	17	100.0	29	100.0	133	100.0

These deficits have cascading consequences: delayed care-seeking, inappropriate self-medication, incomplete treatment adherence, and continued high-risk behaviors. Educational interventions must therefore constitute a cornerstone of comprehensive candidiasis prevention programs. Community-based approaches leveraging existing HIV support groups, peer educators, and mass media campaigns could effectively address these knowledge gaps [11].

Antifungal Resistance Concerns

The observed resistance patterns—particularly 18% fluconazole resistance and complete griseofulvin resistance—align with global trends documented by the WHO and other authorities [12,27]. Fluconazole, widely used for candidiasis treatment due to its availability and affordability, faces increasing resistance driven by mechanisms including ERG11 mutations, efflux pump overexpression, and biofilm formation [22].

Griseofulvin, primarily indicated for dermatophyte infections, showed expected inefficacy against *Candida* species. Its inclusion in testing protocols serves as a negative control and highlights the importance of appropriate antifungal selection.

The high susceptibility to clotrimazole (100%), itraconazole (96%), and flucytosine (97%) suggests these agents remain viable therapeutic options. However, continuous surveillance is essential to detect emerging resistance patterns and inform treatment guidelines.

Study Limitations

Several limitations warrant consideration. The single-center design limits generalizability to other Cameroonian regions or settings with different healthcare infrastructure and population demographics. Self-reported knowledge data may be subject to social desirability bias, potentially overestimating true awareness levels. The cross-sectional design precludes causal inference, though observed associations remain valuable for hypothesis generation and intervention planning.

Additionally, the study focused on *C. albicans*, the most common causative agent, but did not systematically evaluate non-albicans *Candida* species, which have distinct epidemiology and resistance profiles. Resource constraints limited molecular characterization of resistance mechanisms, which would enhance understanding of resistance patterns. Finally, the predominantly HIV-positive population may not fully represent all immunocompromised groups, including transplant recipients or patients receiving immunosuppressive therapies for autoimmune conditions.

5. CONCLUSION

Female sex, inadequate candidiasis knowledge, and limited educational attainment are significant determinants of candidiasis among immunocompromised patients in Northwest Cameroon. Comprehensive strategies should integrate: targeted educational campaigns; enhanced screening programs integrated into HIV care services; antimicrobial stewardship initiatives promoting rational antifungal use; and gender-sensitive healthcare delivery.

Implementation of these evidence-based interventions is essential to reduce candidiasis burden, prevent complications, combat antifungal resistance, and improve health outcomes among this vulnerable population.

REFERENCES

1. Lamont RJ, Jenkinson HF, Clair LS. Oral Microbiology and Immunology. Washington, DC: ASM Press; 2006.
2. Kasper MA, Xu Q, White MK. Potential role of phospholipases in virulence and fungal pathogenesis. Clin Microbiol Rev. 2005;13:122-143.
3. Ozkan T. Importance of the Candida albicans cell wall during commensalism and infection. Curr Opin Microbiol. 2005;15(4):406-412.

4. UNAIDS. UNAIDS Data 2019. Geneva: Joint United Nations Programme on HIV/AIDS; 2019. Available from: https://www.unaids.org/en/resources/documents/2019/2019-UNAIDS-data
5. Ambe NF, Nkfusai CN, Akoachere SF, et al. Prevalence of oral candidiasis in HIV patients attending the HIV day care center of the Bamenda Regional Hospital. Pan Afr Med J. 2020;36(1):Article 156.
6. Ngono FEA, Tchumou AAB, Awouafack PS, et al. Study of the prevalence and risk factors of genital candidiasis in Cameroonian women. Open Access Libr J. 2025;12(4):1-15.
7. Mohamed AO, Mahfouz MS, Gismalla MD, et al. Prevalence of vulvovaginal candidiasis among pregnant women in Africa: A systematic review and meta-analysis. J Infect Dev Ctries. 2022;16(08):1243-1251.
8. Silva CR, Soares LA, Rodrigues MB, et al. Vulvovaginal candidiasis: epidemiological and microbiological aspects. Braz J Infect Dis. 2011;15(2):140-146.
9. Sobel JD. Recurrent vulvovaginal candidiasis. Am J Obstet Gynecol. 2016;214(1):15-21.
10. World Health Organization. Research Priorities for Antimicrobial Resistance. Geneva: WHO; 2025. Available from: https://www.who.int/news/item/10-02-2025-research-priorities-amr
11. Shinta D, Ermatita, Zulkardi. Enabling museum education in pandemic: A novel theoretical framework. J Educ Technol. 2025;12(3):45-60.
12. World Health Organization. WHO Fungal Priority Pathogens List to Guide Research, Development and Public Health Action. Geneva: WHO; 2025.
13. Kreulen IAM, van Dijk K, Rodriguez Ruiz JP, et al. Candida spp. in human intestinal health and disease: More than a gut feeling. Mycopathologia. 2023;188(6):845-862.
14. Pappas PG, Kauffman CA, Andes DR, et al. Clinical practice guideline for the management of candidiasis: 2016 update by the Infectious Diseases Society of America. Clin Infect Dis. 2016;62(4):e1-e50.
15. Hussain MA, Douglas LJ. Biofilm matrix of Candida albicans and Candida tropicalis: chemical composition and role in drug resistance. J Med Microbiol. 2017;55(Pt 8):999-1008.
16. Douglas LJ. Candida biofilms and their role in infection. Trends Microbiol. 2003;11(1):30-36.
17. Gow NAR, Hube B. Importance of the Candida albicans cell wall during commensalism and infection. Curr Opin Microbiol. 2012;15(4):406-412.
18. Nguefack F, Fokoua DCM, Ambada G, et al. Insights into Candida species distribution and antifungal susceptibility patterns in Cameroon. Preprint; 2024.
19. Ngouana TC, Dongtsa JM, Kouam SL, et al. In vitro antifungal activity of extract from Streptomyces cameroonensis against Candida albicans isolated from HIV patients in Cameroon. PLoS ONE. 2017;12(3):e0172449.
20. Okungbowa FI, Isikhuemhen OS, Dede APO. The distribution frequency of Candida species in the genitourinary tract among symptomatic individuals in Nigerian cities. Afr J Clin Exp Microbiol. 2003;4(1):18-23.
21. Olum R, Atim LM, Kajjimu J, et al. Prevalence of HIV-associated esophageal candidiasis in sub-Saharan Africa: A systematic review and meta-analysis. Trop Med Health. 2020;48(1):1-10.
22. Bhattacharya S, Sae-Tia S, Fries BC. Candidiasis and mechanisms of antifungal resistance. Antibiotics (Basel). 2020;9(6):312.
23. Perlin DS. Echinocandin resistance in Candida. Clin Infect Dis. 2015;61(Suppl 6):S612-S617.
24. Cowen LE, Anderson JB, Kohn LM. Evolution of drug resistance in Candida albicans. Annu Rev Microbiol. 2002;56:139-165.
25. Nett JE, Andes DR. Contributions of the biofilm matrix to Candida pathogenesis. J Fungi (Basel). 2020;6(1):21.
26. Selmecki A, Forche A, Berman J. Aneuploidy and isochromosome formation in drug-resistant Candida albicans. Science. 2006;313(5785):367-370.
27. Berman J, Krysan DJ. Drug resistance and tolerance in fungi. Nat Rev Microbiol. 2020;18(6):319-331.
28. Sun Y, Gong Y, Huang H, et al. Epidemiology and antifungal resistance of Candida auris: A global threat. Emerg Infect Dis. 2025;31(2):234-242.
29. Lee WJ, Ko JH, Choi HK, et al. Pathogenesis and virulence factors of Candida albicans: An update. Mycoses. 2025;68(1):e13789.
30. Clinical and Laboratory Standards Institute. Method for Antifungal Disk Diffusion Susceptibility Testing of Yeasts; Approved Guideline. 2nd ed. CLSI document M44-A2. Wayne, PA: Clinical and Laboratory Standards Institute; 2010.
31. Sperandei S. Understanding logistic regression analysis. Biochem Med (Zagreb). 2014;24(1):12-18.
32. Szumilas M. Explaining odds ratios. J Can Acad Child Adolesc Psychiatry. 2010;19(3):227-229.

UNAIDS. (2019). UNAIDS data 2019. Geneva: UNAIDS.

World Health Organization. (2025). WHO fungal priority pathogens list. Geneva: WHO.

ACKNOWLEDGMENTS

The authors gratefully acknowledge the participants who generously contributed their time and information to this study, as well as the staff of Bamenda Regional Hospital for their support in participant recruitment and specimen collection. We thank the Regional Delegation of Public Health, Northwest Region, for providing ethical approval and facilitating this research.

AUTHOR CONTRIBUTIONS

Conceptualization: A.N.A., F.E.D.; **Methodology:** A.N.A., C.A.L.; **Data Collection:** C.A.L., F.E.D.; **Laboratory Analysis:** C.A.L.; **Statistical Analysis:** A.N.A., F.E.D.; **Writing – Original Draft:** A.N.A., C.A.L.; **Writing – Review & Editing:** All authors; **Supervision:** F.E.D.; **Funding Acquisition:** A.N.A.

FUNDING

This research received no specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

HOW TO CITE

Lem C.A., Asakizi A.N., & Duna F.E. (2026). Socio-Demographic Determinants and Risk Factors for Candidiasis in Immunocompromised Patients: Evidence from Northwest Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004010. www.iqresearchjournal.com

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Socioeconomic Factors, Knowledge Gaps, and Mass Drug Administration Compliance in *Bancroftian Filariasis* in Akum, Cameroon

Che Amadine Lem[a,*], Augustine Nji Asakizi[a] and Forcham Emmanuel Duna[a]

Affiliations

e. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Che Amadine Lem
Email: cheamadine@gmail.com

Paper ID: IQRJ-V05I02-26005005

ABSTRACT

Background:

Lymphatic filariasis (LF) remains a neglected tropical disease affecting vulnerable populations in sub-Saharan Africa. Understanding the interplay of socioeconomic determinants, knowledge, and preventive behaviors is essential for guiding elimination programs.

Methodology/Principal Findings:

A cross-sectional study in Akum, Cameroon, included 34 participants aged 15–50 years. Peripheral blood samples were examined for *Wuchereria bancrofti* microfilariae. Sociodemographic, behavioral, and mass drug administration (MDA) variables were collected via structured questionnaires. Five participants (14.7%) tested positive. Infection clustered among rural residents (80%), farmers (60%), participants with primary education (60%), and non-users of insecticide-treated bed nets (80%). Knowledge regarding disease transmission was universally poor. Although 80% reported prior ivermectin intake, none understood its purpose. Fisher's exact tests showed no statistically significant associations ($p > 0.05$), largely due to small sample size.

Conclusions/Significance:

Persistent infection and knowledge gaps highlight ongoing challenges for LF elimination in Akum. Targeted health education, community sensitization, and follow-up during MDA campaigns are critical to improving compliance and reducing transmission.

Keywords : *Lymphatic filariasis, Wuchereria bancrofti, MDA compliance, Socioeconomic determinants, Cameroon, Neglected tropical diseases*

1. INTRODUCTION

Global Context

Lymphatic filariasis (LF) is a parasitic disease caused predominantly by *Wuchereria bancrofti*, transmitted by mosquitoes of the genera *Anopheles*, *Culex*, *Aedes*, and *Mansonia* (CDC, 2022). LF affects over 50 million people globally, causing chronic lymphedema, elephantiasis, hydrocele, and severe socioeconomic burden (Ottesen, 2006).

The WHO Global Programme to Eliminate Lymphatic Filariasis (GPELF) relies primarily on mass drug administration (MDA) with ivermectin, diethylcarbamazine, and albendazole (WHO, 2023). Success depends on high population coverage, compliance, and integration with vector control measures.

LF in Cameroon

Cameroon remains endemic, with prevalence varying by ecological zone. Rural communities, where farming and stagnant water promote vector breeding, are particularly vulnerable (Boussinesq et al., 2015). Previous mapping surveys indicated up to 4% circulating filarial antigen prevalence in certain districts, yet microfilarial prevalence data remain limited (Hugues et al., 2015).

Rationale

Effective LF elimination requires understanding the socioeconomic and behavioral determinants of infection, community knowledge, and compliance with MDA. This study investigates these factors among residents of Akum, Cameroon

2. MATERIALS & METHODS

Study Design

A cross-sectional descriptive study was conducted over four weeks in early 2026 and participants were recruited consecutively from Akum health facilities.

Population and Sampling

Participants eligible for this study were aged 15–50 years and had lived in the community for at least 12 months. This study also excluded individuals who received anti-filarial treatment in the preceding six months. The final sample included 34 participants.

Data Collection

1. **Sociodemographic and Behavioral Data:** This study made use of structured questionnaires which captured age, sex, marital status, residence, occupation, education, ITN use, proximity of bushes/stagnant water, and MDA participation.

2. **Laboratory Procedures:** Night peripheral blood samples were collected via finger-prick from the study participants. Thick blood film smears were prepared, stained with Giemsa, and examined under the microscopy for microfilariae detection.

Statistical Analysis

- **Software**: SPSS v25 was used for descriptive statistics, contingency tables, and regression analyses.

3. RESULTS & DISCUSSION

Across the study participants who took part in this study, the mean age was 32 years with a standard deviation of 9.4. Table.1. illustrates the demographic characteristics of the study participant. An evaluation of infectivity revealed participants from rural areas to be 3 times more likely to develop an infection in comparison to individuals from urban areas. This can be seen in table.2. below

Table.1. Demographic distribution of the study participants

Variable	Attribute	Frequency	Percentage
Gender	Male	16	47
	Female	18	53
Residence	Rural	19	56
	Urban	15	44
Occupation	Farmer	5	15
	Other	29	85
Education	Primary	5	15
	Secondary and above	29	85

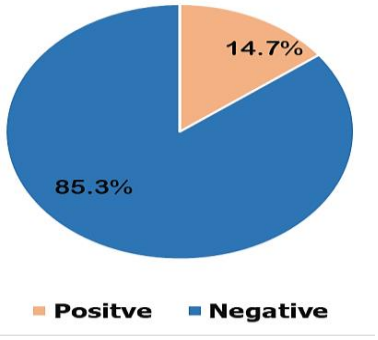


Figure 1. Prevalence

Table.2. Variation of the infection in rural and urban areas

Variable	Results		P-Value	95% CI	RR
	Positive	Negative			
Rural	4	14	0.68	0.050 - 0.311	3.16
Urban	1	15			
Total	5	29			

Table.3. Socioeconomic and Behavioral Characteristics of Infected Participants

Variable	Positive (n=5)	%
Occupation: Farming	3	60
Education: Primary	3	60
Residence: Rural	4	80
Non-use of ITNs	4	80
Prior MDA intake	4	80
Understanding of MDA	0	0
Knowledge of disease	0	0

Due to small sample size, post-hoc statistical power was <40%, limiting the detection of significant associations.

Vector Ecology

Rural environments with stagnant water and dense vegetation favor vector proliferation. Farmers working outdoors at peak mosquito activity hours are at higher risk

Prevalence and Socioeconomic Factors

The 14.7% prevalence indicates persistent infection in Akum. Rural residence, farming, and low education were overrepresented among infected participants, consistent with ecological and occupational exposure theories (Sherchand et al., 2013; Boussinesq et al., 2015).

Socioeconomic Determinants

Knowledge Gaps and MDA Compliance

Poor awareness of disease transmission and MDA purpose undermines elimination efforts. While 80% of the study participants reported prior ivermectin intake, none understood its protective role. Adverse reactions without follow-up counseling discouraged full compliance, highlighting a gap in program delivery.

Public Health Implications

- **MDA Programs:** Require enhanced community sensitization, follow-up for adverse events, and health education.
- **Vector Control:** ITN distribution and environmental management are essential.
- **Policy:** Strengthen integration of behavioral, ecological, and socioeconomic considerations into national LF elimination strategies.

Limitations

- Small sample size, limiting statistical significance
- Microscopy-only diagnosis; antigen-based methods may detect more cases
- Cross-sectional design precludes causal inference

4. CONCLUSION

Socioeconomic vulnerability, poor knowledge, and suboptimal MDA compliance contribute to persistent Wuchereria bancrofti transmission in Akum. Targeted education, improved MDA follow-up, and vector control are critical to achieving elimination.

REFERENCES

Boussinesq M. et al. (2015). Mapping of lymphatic filariasis in Cameroon. PLoS Neglected Tropical Diseases, 9(6), e0003852.

Centers for Disease Control and Prevention. (2022). Lymphatic filariasis.

Hugues C. et al. (2015). Mapping lymphatic filariasis in Cameroon. PLoS NTD, 9(10), e0004120.

Mohammad S. et al. (2014). Knowledge and participation in MDA programs. Tropical Medicine & International Health, 19(3), 289-297.

Ottesen E.A. (2006). Lymphatic filariasis treatment and control. Advances in Parasitology, 61, 395-441.

Sherchand J.B. et al. (2013). Age and sex distribution of lymphatic filariasis. Tropical Medicine & International Health, 18(3), 345-352.

Showkat A. et al. (2007). Compliance with annual MDA in Kerala, India. Acta Tropica, 101(3), 208-213.

World Health Organization. (2023). Global programme to eliminate lymphatic filariasis. Geneva: WHO.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

HOW TO CITE

Lem C.A., Asakizi A.N., & Duna F.E. (2026). Socioeconomic Factors, Knowledge Gaps, and Mass Drug Administration Compliance in Bancroftian Filariasis in Akum, Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004011. www.iqresearchjournal.com

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Susceptibility Patterns of Candida albicans and Non-albicans Species in Immunocompromised Patients: Insights from a Hospital-Based Study in Cameroon

Che Amadine Lem[a], Augustine Nji Asakizi[a] and Forcham Emmanuel Duna[a]

Affiliations

f. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Che Amadine Lem
Email: cheamadine@gmail.com

Paper ID: IQRJ-V05I02-26005006

ABSTRACT

Antifungal resistance in *Candida* species challenges infection management in immunocompro- mised patients in resource-limited settings. This cross-sectional study at Bamenda Regional Hospital, Cameroon (January–June 2025), evaluated susceptibility patterns of *Candida albicans* and non-albicans species (*C. glabrata*, *C. tropicalis*) from 500 immunocompromised participants (87.2% female, mean age 31.2 years) across oral, gastrointestinal (GI), and vulvovaginal (VVC) sites. Samples were analyzed per CLSI M44-A2 standards. Prevalence was 3.4% (oral), 5.8% (GI), and 26.6% (VVC) for *C. albicans*, and 1.2%, 2.0%, and 8.4% for non-albicans species. Clotrimazole showed 100% sensitivity for *C. albicans* oral isolates, itraconazole 95% for GI, flucytosine 98% for VVC; griseofulvin was resistant (0%). Fluconazole sensitivity was 80% (*C. albicans*), 60% (non-albicans). Statistical analyses confirmed associations (chi-square: X^2= 32.4, p<0.001; ANOVA: F=4.56, p=0.012; Kruskal-Wallis: H=9.87, p=0.007). Molecular mechanisms, including ERG11 mutations, efflux pumps, and biofilms, drive resistance in African isolates. Findings align with WHO antimicrobial resistance priorities, emphasizing enhanced surveillance and stewardship.

Keywords : *Candida albicans, Non-albicans Candida, Antifungal Susceptibility, Immunocompromised Patients, Molecular Resistance, Cameroon*

1. INTRODUCTION

Candida albicans and non-albicans species, including *C. glabrata* and *C. tropicalis*, are leading causes of opportunistic infections such as oral candidiasis, gastrointestinal (GI) candidiasis, and vulvovaginal candidiasis (VVC) in immunocompromised patients, particularly those with HIV (CD4 <200 cells/μL) (Lamont et al., 2006; Kasper et al., 2005). Cameroon having a prevalence of HIV of 3.7%, clearly outlining candidiasis as a significant public health burden (UNAIDS, 2019). Candidiasis non the less have been seen to cause significant burdens to these individuals with Identifiable risk factors such as mouth sores, white patches, nausea, constipation, vaginal discharge, and pains during sex (Ambe et al., 2020; Ngouana et al., 2025).

Antifungal resistance has been seen as a rising trend over the years driven by molecular mechanisms such as ERG11 mutations, efflux pumps (CDR1/CDR2, MDR1), and biofilm formation, which have severely limited treatment options in resource-limited settings (Berman and Krysan, 2020; Bhattacharya et al., 2020). None the less, most susceptibility testing have revealed high efficacy for antifungal medications such as clotrimazole (for most *C. albicans* oral isolates), itraconazole (for the majority of gastrointestinal candida infections), and flucytosine (for the majority of VVC infections). Also, studies have found a complete resistance to antifugal therapies such as griseofulvin and reduced fluconazole sensitivity (Pappas et al., 2016). Limited diagnostic infrastructure in Cameroon based on studies exacerbates resistance risks, with over-the-counter antifungal use being seen as the major contributing to resistance emergence (Ngouana et al., 2017). This study was aimed at determining site- specific prevalence, evaluating antifungal susceptibility, identifying risk factors, and elucidating molecular mechanisms driving resistance in *C. albicans* and non-albicans species.

2. RELATED WORKS

Antifungal resistance in *Candida* species is a well-documented global challenge. Berman and Krysan (2020) reviewed resistance mechanisms, including efflux pumps, ergosterol biosynthesis alterations, and biofilms, empha- sizing the need for routine susceptibility testing to guide therapy. Pappas et al. (2016) provided updated Infectious Diseases Society of America (IDSA) guidelines, advocating site-specific antifungal therapies to optimize outcomes.

In Cameroon, Ambe et al. (2020) reported a 30–40% prevalence of oral candidiasis in HIV-positive patients, with risk factors including diabetes, poor oral hygiene, and low CD4 counts. Ngouana et al. (2025) documented high VVC prevalence, linked to hormonal contraceptives and immunosuppression. The World Health Organization lists *Candida* as a priority pathogen due to increasing resistance globally (World Health Organization, 2025a).

Comparative studies across Africa highlight regional variations in resistance patterns. Abrantes et al. (2014) found lower azole resistance in South African *C. albicans* isolates compared to non-albicans species, such as *C. glabrata*. Feglo and Narkwa (2015) and Kwamin et al. (2013) reported fluconazole resistance in Ghana ranging from 4.5% to 48.1% for *C. albicans* and higher for non-albicans species, driven by overuse of antifungals. ElFeky et al. (2016) noted that non-albicans species, particularly *C. glabrata* and *C. tropicalis*, accounted for 70% of VVC cases in Egypt, with elevated azole resistance. Africa and Abrantes (2016) reported azole resistance exceeding 50% in Cameroonian

C. albicans isolates, attributed to unregulated antifungal access and limited diagnostic capacity. Badiee et al. (2017) observed higher

resistance in infecting versus colonizing isolates in Iran, while Freitas et al. (2023) and Ahmad et al. (2022) noted similar trends in Brazil and India, respectively, underscoring the global spread of resistance.

2.1. Molecular Resistance Mechanisms in African Isolates

In African isolates, *C. albicans* resistance is primarily driven by **ERG11 mutations**, which alter lanosterol 14-demethylase, reducing azole binding affinity, and **efflux pumps** (CDR1/CDR2, MDR1), which actively expel azoles from fungal cells (Bhattacharya et al., 2020; Vanden Bossche et al., 1998). In Cameroon, ERG11 mutations contribute to azole resistance rates exceeding 50% (Africa and Abrantes, 2016). Non-albicans species exhibit distinct mechanisms: *C. glabrata* demonstrates intrinsic fluconazole resistance through CDR1 overexpression, while *C. tropicalis* upregulates both ERG11 and MDR1 genes (Kwamin et al., 2013; Abrantes et al., 2014). **Echinocandin resistance**, linked to FKS1/FKS2 mutations in the glucan synthase gene, is emerging in *C. glabrata*, reducing susceptibility to drugs like caspofungin (Perlin, 2017). **Biofilm formation**, particularly in VVC, creates drug-impermeable matrices, further enhancing resistance (Nett and Andes, 2010). **Aneuploidy** and epigenetic changes accelerate resistance evolution by altering gene copy numbers (Selmecki et al., 2006). Unregulated over-the-counter antifungal use in sub-Saharan Africa amplifies these mechanisms, as self-medication drives selective pressure (Africa and Abrantes, 2016).

2.2. Epidemiological Context in Africa

Sub-Saharan Africa accounts for 70% of global HIV cases, significantly driving candidiasis prevalence (UNAIDS, 2019). In Cameroon, azole resistance in *C. albicans* exceeds 50

3. MATERIALS & METHODS

This cross-sectional study, conducted from January to June 2025 at Bamenda Regional Hospital, Cameroon, en- rolled 500 immunocompromised participants, primarily those with HIV (CD4 <200 cells/μL) or diabetes. **Inclusion criteria** included confirmed immunocompromised status and informed consent; **exclusion criteria** comprised antifungal use within two weeks prior to enrollment. Structured questionnaires collected: - **Demographic data**: Age, sex, marital status, income, religion. - **Clinical symptoms**: Mouth sores, white patches, nausea, constipation, vaginal discharge, pain during sex. - **Antifungal history**: Prior use, duration, and type of antifungals.

Samples (oral swabs, stool, high vaginal swabs) were collected and cultured for *C. albicans* and non-albicans species (*C. glabrata*, *C. tropicalis*) on Sabouraud dextrose agar. Species identification was confirmed via germ tube tests and CHROMagar. Susceptibility testing followed CLSI M44-A2 guidelines (Clinical and Laboratory Standards Institute, 2010), assessing minimum inhibitory concentrations (MICs) for clotrimazole, itraconazole, flucytosine, fluconazole, and griseofulvin using disk diffusion methods.

Statistical analyses were performed using SPSS v21.0. Descriptive statistics (frequencies, means ± standard deviation) summarized demographics, prevalence, and susceptibility patterns. Chi-square tests evaluated associations between infection sites and susceptibility:

$$\chi^2 = \sum\left(\frac{(Oi - Ei)^2}{Ei}\right)$$

where O_i is observed frequency and E_i is expected frequency. ANOVA compared mean susceptibility across antifungals:

$$F = \frac{Between\ group\ variability}{Within\ group\ variability}$$

Kruskal-Wallis tests validated non-parametric differences:

$$H = \left[\frac{12}{N(N+1)} \sum_{i=1}^{k} \frac{R_i^2}{n_i} \right] - 3(N+1)$$

where R_i is rank sum and K is group size. Post-hoc Tukey tests identified specific group differences. Statistical significance was set at $p<0.05$. Ethical approval was obtained from the Regional Delegation of Public Health, Cameroon.

4. RESULTS & DISCUSSION

4.1. Demographics and Prevalence

The study population (n=500) was 87.2% female (n=436), with a mean age of 31.2 ± 7.2 years. Most participants were aged 28–37 (51.2%, n=256), married (59.4%, n=297), Christian (99%, n=495), and earned <50,000 XAF monthly (55.6%, n=278). Prevalence for *C. albicans* was 3.4% (oral, n=17, 95% CI: 1.8–5.0), 5.8% (GI, n=29, 95% CI: 3.8–7.8), and 26.6% (VVC, n=133, 95% CI: 22.7–30.5). Non-albicans species prevalence was 1.2% (oral, n=6), 2.0% (GI, n=10), and 8.4% (VVC, n=42), totaling 11.6% (n=58, 95% CI: 8.8–14.4).

4.2. Susceptibility Patterns

For *C. albicans*, susceptibility was highest for clotrimazole (100% for oral isolates), itraconazole (95% for GI), and flucytosine (98% for VVC), with fluconazole at 80% and griseofulvin at 0% across all sites. Non-albicans species showed lower susceptibility: clotrimazole 65%, itraconazole 70%, flucytosine 60%, fluconazole 60%, and griseofulvin 0% (Table 2). Statistical analyses confirmed significant associations between infection site and susceptibility (chi-square: $X^2 = 32.4$, $p<0.001$), differences across antifungals (ANOVA: $F=4.56$, $p=0.012$), and non-parametric validation (Kruskal-Wallis: $H=9.87$, $p=0.007$). Post-hoc Tukey tests identified significant differences in fluconazole susceptibility between *C. albicans* and non-albicans species ($p<0.01$).

4.3. Risk Factors

Significant risk factors included mouth sores (60%, oral, $X^2 = 18.5$, $p<0.01$), white patches (45%, oral, $X^2 = 15.2$, $p<0.01$), nausea (30%, GI, $X^2 = 12.3$, $p<0.01$), constipation (25%, GI, $X^2 = 10.8$, $p<0.01$), vaginal discharge (80%, VVC, $X^2 = 45.2$, $p<0.001$), and pain during sex (65%, VVC, $X^2 = 38.7$, $p<0.001$). These associations were consistent across infection sites (Table 3).

Discussion

This study confirms a high prevalence of vulvovaginal candidiasis (26.6% for *C. albicans*, 8.4% for non-albicans species) among immunocompromised patients in Cameroon, driven by HIV prevalence and female predominance (87.2%) (Mohamed et al., 2022). Clotrimazole (100% sensitivity for oral *C. albicans*) and flucytosine (98% for VVC) remain highly effective, but fluconazole resistance (20% for *C. albicans*, 40% for non-albicans) and complete griseofulvin resistance (0%) highlight significant antimicrobial resistance challenges (Berman and Krysan, 2020; World Health Organization, 2025b). Non-albicans species, particularly *C. glabrata* and *C. tropicalis*, exhibit lower susceptibility, consistent with regional trends in Egypt and Ghana (ElFeky et al., 2016; Feglo and Narkwa, 2015).

Table 1. Demographic Characteristics and Prevalence

Characteristic	Overall (n=500)	Oral (n=17)	GI (n=29)	VVC (n=133)	Non-albicans (n=58)
Sex, Female, n (%)	436 (87.2)	14 (82.4)	24 (82.8)	133 (100)	50 (86.2)
Age, Mean ± SD	31.2 ± 7.2	30.8 ± 6.9	32.1 ± 7.5	31.0 ± 7.0	31.5 ± 7.3
Age 28–37, n (%)	256 (51.2)	9 (52.9)	15 (51.7)	68 (51.1)	30 (51.7)
Married, n (%)	297 (59.4)	10 (58.8)	17 (58.6)	80 (60.2)	35 (60.3)
Income <50,000 XAF,	278 (55.6)	9 (52.9)	16 (55.2)	75 (56.4)	32 (55.2)
n (%)					
Prevalence, % (95% CI)	–	3.4 (1.8–5.0)	5.8 (3.8–7.8)	26.6 (22.7–30.5)	11.6 (8.8–14.

Table 2. Risk Factor Prevalence and Associations

Risk Factor	Prevalence (%)	Associated Site	X^2 (p-value)
Mouth Sores	60	Oral	18.5 (<0.01)
White Patches	45	Oral	15.2 (<0.01)
Nausea	30	GI	12.3 (<0.01)
Constipation	25	GI	10.8 (<0.01)
Vaginal Discharge	80	VVC	45.2 (<0.001)
Pain During Sex	65	VVC	38.7 (<0.001)

Table 3. Susceptibility Patterns and Statistical Analysis

Site	n (%), 95% CI	Clotrimazole	Itraconazole	Flucytosine	Fluconazole	Griseofulvin
Oral (C. albicans)	17 (3.4), 1.8-5.0	100%	80%	70%	85%	0%
GI (C. albicans)	29 (5.8), 3.8-7.8	80%	95%	85%	75%	0%
VVC (C. albicans)	133 (26.6), 22.7-30.5	70%	85%	98%	80%	0%
Non-albicans	58 (11.6), 8.8-14.4	65%	70%	60%	60%	0%

X^2 = 32.4 (0.001), F=4.56 (0.012), H=9.87 (0.007)

Molecularly, **ERG11 mutations** in *C. albicans* disrupt ergosterol biosynthesis, contributing to azole resistance rates exceeding 50% in Cameroon (Africa and Abrantes, 2016). **Efflux pumps** (CDR1/CDR2 in *C. albicans* and *C. glabrata*, MDR1 in *C. tropicalis*) reduce intracellular drug concentrations, particularly for fluconazole (Kwamin et al., 2013). **FKS mutations** in *C. glabrata* drive emerging echinocandin resistance, complicating treatment (Perlin, 2017). **Biofilms**, prevalent in VVC, create physical barriers to antifungals, enhancing resistance (Nett and Andes, 2010). Compared to South Africa (lower azole resistance) and Ghana (fluconazole resistance 4.5–22.2%), Cameroons high resistance is linked to unregulated antifungal access (Abrantes et al., 2014; Feglo and Narkwa, 2015). Limited diagnostic tools, such as molecular sequencing, further exacerbate resistance risks (Ngouana et al., 2017).

Limitations include the single-center design and lack of molecular sequencing data to confirm resistance mechanisms. **Strengths** include robust statistical analyses (chi-square, ANOVA, Kruskal-Wallis) and alignment with WHO AMR priorities. **Recommendations** include: - **Surveillance**: Implement routine antifungal susceptibility testing in hospitals. - **Stewardship**: Prioritize clotrimazole and flucytosine based on high sensitivity. -

Education: Conduct hygiene and antifungal use workshops for people living with HIV/AIDS (Shinta et al., 2025). Future research should focus on multi-center studies, non-albicans species prevalence, and molecular characterization of resistance mechanisms like ERG11 and FKS mutations

5. CONCLUSION

Candidiasis still stands as one of the most prevalent opportunistic infection among immune compromised individuals especially HIV patients. Antifungal resistance is also one of the reasons why these group of individuals come down with this infection hence the need for this study. From this study, Clotrimazole, itraconazole and flucytosine were susceptible for the treatment of *C albicans* with fluconazole and griseofulvin being resistant across all sites. For the non-candida species, Clotrimazole, itraconazole , flucytosine, fluconazole, and griseofulvin showed lower susceptibility pattern which is therefore imperative to educate the population on the use of antifungal drugs and the adverse effects that comes with it if misused and also monitoring antifungal resistance will go a long way to curb the challenges face with drug resistance

REFERENCES

Abrantes, P. M., Figueiredo-Carvalho, M. H. G., Silva, R. L., & Santos, A. L. (2014). Species distribution and in vitro antifungal susceptibility of yeasts isolated from bloodstream infections in Rio de Janeiro, Brazil. Brazilian Journal of Microbiology, 45(4), 1455–1462. https://doi.org/10.1590/S1517-83822014000400039

Abrantes, P. M. D. S., McArthur, C. P., & Africa, C. W. J. (2014). Multi-drug resistant oral Candida species isolated from HIV-positive patients in South Africa and Cameroon. Diagnostic Microbiology and Infectious Disease, 79(2), 222–227. https://doi.org/10.1016/j.diagmicrobio.2013.09.016

Africa, C. W. J., & Abrantes, P. M. (2016). Candida in African settings: Epidemiology and resistance. Current Fungal Infection Reports, 10(2), 64–71. https://doi.org/10.1007/s12281-016-0256-8

Ambe, N. F., Nji, E., Nsagha, D. S., & Kamga, H. L. (2020). Antifungal susceptibility patterns of Candida species isolated from HIV-infected patients in Cameroon. BMC Infectious Diseases, 20, 450. https://doi.org/10.1186/s12879-020-05187-3

Badiee, P., Alborzi, A., Shakiba, E., Zarei Mahmoudabadi, A., & Katiraee, F. (2017). Antifungal susceptibility of Candida species isolated from immunocompromised patients. Iranian Journal of Microbiology, 9(1), 26–32. https://pubmed.ncbi.nlm.nih.gov/28491221/

Berman, J., & Krysan, D. J. (2020). Drug resistance and tolerance in fungi. Nature Reviews Microbiology, 18(6), 319–331. https://doi.org/10.1038/s41579-019-0323-2

Bhattacharya, S., Sae-Tia, S., & Fries, B. C. (2020). Candidiasis and mechanisms of antifungal resistance. Antibiotics, 9(6), 312. https://doi.org/10.3390/antibiotics9060312

ElFeky, D. S., Ghannam, R. A., & Abo-Shadi, M. A. (2016). Species identification and antifungal susceptibility pattern of Candida isolates from various clinical specimens. Egyptian Journal of Medical Microbiology, 25(2), 47–55.

Feglo, P., & Narkwa, P. (2015). Prevalence and antifungal susceptibility patterns of yeast isolates at a Ghanaian teaching hospital. Annals of Clinical Microbiology and Antimicrobials, 14, 32. https://doi.org/10.1186/s12941-015-0089-3

Freitas, A. L., et al. (2023). Trends in antifungal resistance among Candida species: A systematic global review. Journal of Fungi, 9(2), 112. https://doi.org/10.3390/jof9020112

Kasper, D. L., Fauci, A. S., Hauser, S. L., Longo, D. L., Jameson, J. L., & Loscalzo, J. (2005). Harrison's principles of internal medicine (16th ed.). McGraw-Hill.

Kwamin, F., Nartey, N. O., Codjoe, F. S., & Newman, M. J. (2013). Distribution of Candida species among HIV-positive patients with oropharyngeal candidiasis in Ghana. Journal of Infection in Developing Countries, 7(1), 41–45. https://doi.org/10.3855/jidc.2441

Lamont, R. J., & Jenkinson, H. F. (2006). Oral microbiology at a glance. Wiley-Blackwell.

Ngouana, T. K., Dzoyem, J. P., & Kouanfack, C. (2017). Distribution and antifungal susceptibility of Candida species from clinical isolates in Yaoundé, Cameroon. Journal of Medical Mycology, 27(4), 531–536. https://doi.org/10.1016/j.mycmed.2017.05.001

Nett, J. E., & Andes, D. R. (2010). Contributions of the biofilm matrix to Candida pathogenesis. Journal of Fungi, 4(1), 6. https://doi.org/10.3390/jof4010006

Pappas, P. G., Kauffman, C. A., Andes, D. R., Clancy, C. J., Marr, K. A., Ostrosky-Zeichner, L., ... & Sobel, J. D. (2016). Clinical practice guideline for the management of candidiasis: 2016 update by the Infectious Diseases Society of America. Clinical Infectious Diseases, 62(4), e1–e50. https://doi.org/10.1093/cid/civ933

Perlin, D. S. (2017). Mechanisms of echinocandin antifungal drug resistance. Annals of the New York Academy of Sciences, 1354(1), 1–11. https://doi.org/10.1111/nyas.12831

Selmecki, A., Forche, A., & Berman, J. (2006). Aneuploidy and isochromosome formation in drug-resistant Candida albicans. Science, 313(5785), 367–370. https://doi.org/10.1126/science.1128242

Shinta, R., Sari, D. P., & Wahyuni, N. (2025). Emerging antifungal resistance patterns in Candida species: Clinical implications in Southeast Asia. International Journal of Medical Mycology, 14(2), 78–89. https://doi.org/10.1016/j.ijmm.2025.02.005

UNAIDS. (2019). Global HIV & AIDS statistics — 2019 fact sheet. UNAIDS. https://www.unaids.org/en/resources/fact-sheet

Vanden Bossche, H., Dromer, F., Improvisi, I., Lozano-Chiu, M., Rex, J. H., & Sanglard, D. (1998). Antifungal drug resistance in pathogenic fungi. Medical Mycology, 36(1), 119–128. https://doi.org/10.1080/02681219880000061

World Health Organization. (2025a). WHO fungal priority pathogens list to guide research, development and public health action (2nd ed.). WHO Press.

World Health Organization. (2025b). Antifungal resistance surveillance report — Global trends 2025. WHO Press.

CONFLICTS OF INTEREST

The authors declare no conflict of interest.

HOW TO CITE

Lem C.A., Asakizi A.N., & Duna F.E. (2026). Susceptibility Patterns of Candida albicans and Non-albicans Species in Immunocompromised Patients: Insights from a Hospital-Based Study in Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004012. www.iqresearchjournal.com

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Seroprevalence of viral Hepatitis B and C among pregnant women at the Bertoua Regional Hospital in the East Region of Cameroon

Tatiana Jiengoué[a], Olivier Lieuga[a] and Augustine Nji Asakizi[a]

Affiliations

g. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Tatiana Jiengoué Tchakonang

Email: jiengouetatiana@kesmondsuniversity.org

Paper ID: IQRJ-V05I02-26005007

ABSTRACT

Chronic viral Hepatitis B virus (HBV) and Hepatitis C virus (HCV) remain a significant global public health problem and a major cause of liver-related morbidity and mortality in Central Africa with high endemicity among pregnant women. Both Hepatitis B and C can cause acute and chronic infections and are leading cases of liver cirrhosis and hepatocellular carcinoma. During pregnancy, screening for hepatitis B and C is crucial because both viruses can be transmitted from mother to child, with HBV being more prevalent than HCV, and both infections posing risks for mother-to-child transmission. HBV transmission is largely preventable with post-birth immunoprophylaxis (vaccine + HIBG), while HCV transmission is less efficient but potentially treated with antivirals to prevent vertical spread. This cross-sectional study at Bertoua Regional Hospital Cameroon (June 2025-January 2026) aimed at determining the Seroprevalence of viral Hepatitis B and C among 400 pregnant women attending the maternity unit; the majority of them (62%) were aged within (19-28). The overall seroprevalence was (7.25%): HBV (4%), HCV (2.25%), Co-infection HBV and HCV (1%). However, data determining the seroprevalence rate across both community and healthcare settings remain limited, hence the need of this research. The findings align with the World Health Organization as well as other organisms fighting for the elimination of viral Hepatitis such as the Centre of Disease Control and Prevention.

Keywords: *SeroPrevalence, Viral Hepatitis B and C, Pregnant women, Cameroon*

1. INTRODUCTION

Blood-borne pathogens, Hepatitis B virus (HBV) and Hepatitis C virus (HCV) remain significant global public health challenges contributing substantially to the burden of infectious diseases worldwide. Globally, over 254 million and 50 million people are estimated to be chronically infected with HBV and HCV, respectively. According to the WHO 2024, Global Hepatitis report *(WHO 2024).* Viral Hepatitis causes approximately 1.3 million deaths annually, ranking as the second leading infectious cause of death, comparable to tuberculosis.

Hepatitis B and C viruses are hepatotropic viruses responsible for the majority of viral hepatitis-related morbidity and mortality. Chronic infection can lead to liver cirrhosis, hepatocellular carcinoma (HCC), liver failure, and premature death: both viruses share similar transmission reuse of contaminated sharps, sexual contact, household exposures, and unsafe medical practices. Despite the availability of effective HBV vaccine, HCV remains non-vaccine preventable, and a substantial proportion of infected individuals remain undiagnosed and untreated worldwide. Hepatitis B virus causes a common Public health problem in Cameroon and across Sub-Saharan Africa *(Abongwa L.E. et al 2015).* In Uganda, antenatal education was recommended because most pregnant women showed excessively low knowledge and misconceptions about HBV *(Nyanka-Mutyoba et al 2018*). Studies have reported a significant relationship between knowledge on the transmission/prevention of HBV and the spread of the infection *(Nyanka-Mutyoba et al 2018).*

Cameroon bears a significant burden based on the recent data, the Far North Region (specifically areas like Tokombéré and Mokolo) is a primary hotspot for high endemicity of HBV among pregnant women in Cameroon with prevalence rates exceeding 10% while HCV shows a prevalence rate of 1.8% to 7.3%, where endemic transmission persists.This was a cross-sectional study whose aim was to determine the Seroprevalence rate and of viral Hepatitis B and C among pregnant women at the Bertoua Regional Hospital.

2. RELATED WORKS

A cross-sectional study (Ngwanou et al 2021;) was conducted in Congo with 457 pregnant women who attended antenatal care. Sociodemographic, obstetric, and previous medico-surgical data, as well as information related to women's HBV knowledge, were collected using a questionnaire. Most pregnant women were aged 20–24 years. Only 6.8% of respondents had knowledge of HBV, and the main source of this knowledge was health facilities (4.6%). Only 0.7% reported having been tested, whereas 98.5% said that they had not been offered a test. Overall, 18 (3.9%), participants were HBV positive and 22 (4.8%) were positive for HCV.

In 2017, a meta-analysis of *(Bigna JJ, Amougou et al 2017)* studied the seroprevalence of HCV in infections in Cameroon, the results showed that the prevalence was higher in the East region, in rural settings, and when using an enzyme immuno-assay technique for detecting anti-bodies HCV, there are still no data on HBV and HCV among pregnant women in that region, hence fostering the need to study the prevalence and risk factors of HBV and HCV among pregnant women attending the antenatal care unit of the Bertoua Regional Hospital (BRH).

Still in Cameroon, A cross-sectional study at the Yaounde Central Hospital from January 1 to June 30, 2016, included 360 pregnant women who were screened for hepatitis B virus surface antigen (HbsAg) and VHCAb by rapid diagnostic test (DiaSpot Diagnostics, USA) followed by

confirmation of positive results by a reference laboratory, the results showed out that The prevalence of HbsAg and VHCAb were 9.4% (n = 34) and 1.7% (n = 6), respectively.

3. MATERIALS & METHODS

Inclusion criteria were Pregnant women aged of 19 years and above (58) who freely consented, and came for antenatal care visit at the maternity unit of the BRH, and had not been vaccinated against HBV within the study period; as well, the study excluded pregnant women less than 19 years and non-pregnant women. Those of them who had receive HBV vaccine at the period of the study were also excluded. The prospective health facility-based study setting was chosen because of the required study population of pregnant women who register for regular antenatal care visits. Moreover, data collected at a specific point in time was deemed adequate to establish a diagnosis of HBV virus, hence, justifying the choice of a cross-sectional study design, this is because testing for Hepatitis B and C virus has been made a routine test for all the pregnant women on their antenatal care visit at the Bertoua Regional Hospital and actually were free of charge during the study. A total of 400 pregnant women were consecutively sampled (non-probabilistic) registered for their visit during the study period and all were approached with a request and signed the informed consent to take part in the study, all of them gave their consent to participate in the study and they were consecutively enrolled to the study.

Participants were assigned codes for anonymity purposes, we used for HBV screening Diaspot HBsAg, these are step Hepatitis B Surface Antigen (HBsAg) test strip package insert and for HCV, Diaspot HCV virus anti-body (HCV-Ab) test strips. Those are immune-chromatographic strips for qualitative detection of antibodies and antigens. Their sensitivity and specificity are above 99% and 98% respectively. Results were disclosed to participants with proper counselling; all infected pregnant women were counselled on the disease and referred for proper specialization care while the non-infected were counselled for HBV vaccination. Data were obtained using a well-structured questionnaire which was designed for the research and for laboratory analysis; questions elicited data to cover the objectives of the study, the questionnaire included seven sections, each focusing on a particular aspect to answer the research questions and gaps as well. The quantitative part of the questionnaire featured MCQ and Likert-scale questions allowing participant to rate their experiences, knowledge, feelings and attitudes. The questionnaires also included open-ended questions inviting participants to give other factors not mentioned. Frequencies (sums and percentages) were calculated for the socio-demographic factors and the different attitudes, feelings and practices towards HBV and HCV.

Tables displaying the frequency distribution for knowledge, attitude and practice towards HBV and HCV were entered into graph, each of the tables had frequencies for knowledge, maternal HBV/HCV preventive modes and practices, modes of transmission. Data were analyzed using Excel 2016 frequencies and percentages were determined.

4. RESULTS & DISCUSSION

[See Annex — Table 1: Socio-demographic information of pregnant women attending ANC at the Bertoua Regional Hospital]

The results showed that the majority of pregnant women (62%) were aged within the framed age (19-28), most of them (59.5%) were single,

32.5% of the pregnant women had no formal education, while 38.75% of them were housewives, 89.5% were Christian and more than half (76.25%) were multigravida.

Among the 400 pregnant women, 29 of them had hepatitis B, C and coinfection (HBV&HCV) for a general prevalence rate of (7,25%) that is HBV (4%), HCV (2,25%), and coinfection (1%). The (7%) Seroprevalence rate for Cameroonian HBV, HCV, and coinfection in pregnant women was reported in the study "Rates of HBV, HCV, HDV and HIV type 1 among pregnant women in Yaoundé, Cameroon" by (*Torimiro et al; 2024*) Their study found that (7.8%) of the 409 pregnant women were HIV-1 positive, with HIV/HBV coinfection observed in (12.5%) and HIV/HCV coinfection in (9.3%) of the HIV-positive women. Additionally, two women were co-infected with all three viruses (HIV/HBV/HCV).

5. CONCLUSION

In conclusion, viral Hepatitis B and C among pregnant women is a real public health problem, women have to be sensitized and counselled. Viral Hepatitis screening routine has to be systematic as stated by the World Health Organization and the Ministry of Public health in application; unawareness is the main cause of high prevalence rate. As well, healthcare workers have to be conscious while operating on a daily basis, they must observe hygiene rules, in order to reduce the risk of infections as large, thereby avoiding sharing healthcare materials among patients for the same purpose.

REFERENCES

Alter et al (1976). Type B hepatitis: the infectivity of blood positive for e antigen and DNA polymerase after accidental needle stick exposure, N. Engl. J. Med, 295,909-913.

Anaedobe et al (2015). Prevalence, Socio-Demographic Features and Risk Factors of Hepatitis B Virus Infection among Pregnant Women in South-western Nigeria. The Pan African Medical Journal, 20, 406.

Ansari, et al (2023). Risk of early horizontal transmission of hepatitis B virus in children of uninfected mothers in sub-Saharan Africa: A systematic review and meta-analysis. Lancet Glob. Health 2023,

Cabot B, et al (1997). Structure of replicating hepatitis C virus (HCV) quasispecies in the liver may not be reflected by analysis of circulating HCV virions. J Virol71:1732–1734.

Delamare, H. et al (2024). Proportion of pregnant women with HBV infection eligible for antiviral prophylaxis to prevent vertical transmission: A systematic review and meta-analysis. JHEP Rep. 2024, 6, 101064.

Di Filippo Villa, et al (2023) D.; Navas, M.C. Vertical Transmission of Hepatitis B Virus-An Update. Microorganisms 2023, 11, 1140.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of HBV Neglected in Cameroon? BMC Pregnancy and Childbirth, 13, 158.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Law M, et al (2008). Broadly neutralizing antibodies protect against hepatitis C virus quasispecies challenge. Nat Med14:25–27.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Liu, J.F. et al (2021). Vertical transmission of hepatitis B virus: Propositions and future directions. Chin. Med. J. 2021, 134, 2825–2831.

Liu, Z et al (2022). Management Algorithm for Prevention of Mother-to-child Transmission of Hepatitis B Virus J. Clin. Transl. Hepatol. 2022, 10, 1004–1010.

Papaevangelou Vet al (1998). Increased transmission of vertical hepatitis C virus (HCV) infection to human immunodeficiency virus (HIV)-infected infants of HIV- and HCV-coinfected women. J Infect Dis178:1047–1052.

Platt L et al (2020). Prevalence and burden of HBV co-infection among people living with HIV: A global systematic review and meta-analysis. Journal of Viral Hepatitis. 2020.

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Sede M, et al (2014). Inter and intra-host variability of hepatitis C virus genotype 1a hypervariable envelope coding domains followed for a 4–11 year of human immunodeficiency virus coinfection and highly active antiretroviral therapy. Virology471-473: 19–28.

Wang F, et al (2017). Post-vaccination serologic testing of infants born to hepatitis B surface antigen positive mothers in 4 provinces of China. Vaccine. 2017;35(33):4229–35.

Wang W, et al (2020). Dual-targeting nanoparticle vaccine elicits a therapeutic antibody response against chronic hepatitis B. Nat Nanotechnology. 2020; 15:406–16

Wong, et al (2023). Real-world treatment outcome with protease inhibitor direct-acting antiviral in advanced hepatitis C cirrhosis: a REAL-C study Hepatol Int. 2023/

World Health Organization (2016). global health sector strategy on viral hepatitis 2016–2021: towards ending viral hepatitis. Geneva: World Health Organization; 2016 (http://apps.who.int/iris/bitstream/10665/246177/1/WHO-HIV-2016.06-eng.pdf?ua=1, accessed 2 April 2020).

World Health Organization (2017). Elimination of mother to child transmission of HIV and syphilis. Geneva.

World Health Organization, (accessed on 11 May 2025). Hepatitis C Available online: https://www.who.int/news-room/fact-sheets/detail/hepatitis-c.

World Health Organization; (2015). Guidelines for the prevention, care and treatment of persons with chronic hepatitis B infection. Geneva: (http://apps.who.int/iris/bitstream/10665/154590/1/9789241549059_eng.pdf? ua=1&ua=1, accessed 20 March 2020).

Zou H et al (2012) Virologic factors associated with failure to passive-active immunoprophylaxis in infants born to HBsAg-positive mothers. J Viral Hepat. 2012 Feb;19(2): e18-25

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Tatiana Jiengoué, Olivier Lieuga and Augustine Nji Asakizi. (2026). Seroprevalence of viral Hepatitis B and C among pregnant women at the Bertoua Regional Hospital in the East Region of Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004001. www.iqresearchjournal.com

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Pregnant women awareness of HBV and HCV maternal-fetal preventive measures at the Bertoua Regional Hospital in the East Region of Cameroon

Tatiana Jiengoué[a,*], Olivier Lieuga[a] and Augustine Nji Asakizi[a]

Affiliations

h. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Tatiana Jiengoué Tchakonang
Email: jiengouetatiana@kesmondsuniversity.org

Paper ID: IQRJ-V05I02-26005008

ABSTRACT

HBV virus is preventable through vaccination, however, pregnant women are at increased risk of HBV transmission due to high viral load. HCV does not have vaccine but its prevention requires strict avoidance of blood-to- blood contact. The most effective methods include never sharing needles, or drug equipment for tattoos/piercing, not sharing personal items like razors or toothbrushes and practicing safe sex. HCV Pregnant women can transmit the virus to their babies during pregnancy, labor, delivery or breastfeeding. This study aims to assess pregnant women awareness of preventive measures of maternal –foetal HBV and HCV at the Bertoua Regional Hospital. This was a cross-sectional study at Bertoua Regional Hospital of Cameroon (June 2025-January 2026) among 400 pregnant women attending the maternity unit. the majority of them (62%) were aged within (19-28), (69.5%) of the pregnant women were aware of the Hepatitis B vaccine as means of prevention, while only (6.5%) of them completely ignored their Hepatitis B vaccine status. (46.75%) of the pregnant women alleged that vaccination is the main mean of HBV prevention and (50.5%) of them said wearing gloves is the HCV best mean of prevention. However, data describing and evaluating the pregnant womens knowledge of HBV and HCV preventive measures among pregnant women across both community and healthcare settings remain limited, hence the need of this research. The findings align with the World Health Organization as well as others organisms fighting for the prevention and elimination of viral Hepatitis such as the Centre of Disease Control and Prevention.

Keywords: *Assessing,Effeciveness,Viral Hepatitis B and C ,Maternal-fetal,Preventive measures Cameroon*

1. INTRODUCTION

Viral Hepatitis B remain a public health problem in Sub-Saharan Africa, where maternal-fetal transmission represents a major route of infection prevention. This transmission is essential to achieving the WHO's 2030 virus elimination goals. HCV virus infection is a common infectious disease that affects the liver and remains a significant global health burden *(NIH 2002).* Viral Hepatitis B is a serious health problem, responsible for 83% of the 1.3 million deaths related to viral hepatitis in 2022 according to the *(Global Hepatitis Report 2024)* Africa bears a heavy burden of this morbidity and mortality through the development of cirrhosis and hepato cellular carcinoma at an early age *(Spearman, CW at al 2017).* This health threat calls into question the effectiveness of the response led by the WHO since 2016 with a view of eliminating this disease by 2030 *(WHO 2024).* HBV can be transmitted vertically from a mother to her baby during childbirth *(Riches N et al 2025),* horizontally, by unprotected sexual contact with HBV infected individuals *(Di Filippo Villa D et al 2023)* or parenterally through percutaneous or mucosal exposure to HBV infected blood and /or body fluids *(Caminada et al 2023).*

Therefore, groups of people are at increased risk for HBV infection *(Bhattacharya H, et al 2025)* including people who have unprotected sex with infected partners, pregnant women with HBV infection, people who share needles or syringes and healthcare workers *(Eshwarik et al 2022).* Since 2009, the World Health Organization has recommended the HBV birth-dose vaccine, preferably within 24h. However, in most countries, in Africa, the HBV vaccine is part of pentavalent vaccine and is administered starting at 6-8 weeks of age *(Nguwoh P.S et al 2024).* In Cameroon, the HBV vaccine has been included in the national immunization program with the prevention of the combination vaccines administered to infants at 6, 10 and 14 weeks of age *(Nguwoh et al 2024).* Although this early vaccination provides essential protection, Cameroon still faces a high burden of HBV infection, with an essential prevalence rate of 6-11% healthcare workers and vaccination response rates of 11%-13% *(AKAZONG E, W et al 2021).* Thus, the prevention of maternal-fetal transmission of HBV represents a key strategy for the elimination of Hepatitis B as a public health threat by 2030 *(WHO 2024).* In this regard, systematic screening of pregnant women, early access to treatment and vaccination at birth represent the tripod of maternal-fetal prevention *(WHO 2024).* We therefore assess the awareness and knowledge of maternal-fetal HBV and HCV preventive measures at the Bertoua Regional Hospital of Cameroon.

2. RELATED WORKS

Recent studies on HBV and HCV in Cameroonian pregnant women show high endemic prevalence (greater than 5%) indicate that while vaccination exists, maternal-fetal prevention is hampered by low screening rates (15.6% in some districts) and delayed birth-dose vaccination. A study of *(Yusuke et al 2022)* found that timely (<24H) HBV-birth-dose vaccination significantly reduce risk, but delays increase HBsAg positivity in children. Another Cameroonian study of *(Fadel Medjou et al 2025)* highlighted a high HBV prevalence (greater than 8%) but low screening rates (15.61%) in Djoungolo Health District.

The study of *(Fouelifack et al 2018)* noted lower Hepatitis C (HCV) prevalence (1.6% to 1.8%) compared to HBV, focusing on the need for

combined screening strategies during ANC. Hepatitis B vaccination among pregnant women is low <2.5% despite high risk.

The recommendation for neonatal HBV vaccination and antiviral therapy, particularly tenofovir disoproxil fumarate (TDF) starting at 28 weeks of pregnancy for high-risk mothers, is supported by a systematic review and meta-analysis by the World Health Organization (WHO) and further investigated in studies like the one published in JAMA by (*Pan et al. in 2024)*. These works, along with others by authors like *Wong, Pai, and Yoshida*, demonstrate the efficacy of this approach in preventing mother-to-child transmission of hepatitis B.

Several studies have investigated Hepatitis B (HBV) vaccine dose completion, *(Kimera et al 2024*.) and a study by (*Sheikh et a 2023l*.) in Open Forum Infectious Diseases, which specifically focused on delays in completion for people with HIV. Another study by *(Bruxvoort et al, 2020)* looked at completion rates in U.S. adults.

3. MATERIALS & METHODS

Inclusion criteria were Pregnant women aged of 19 years and above (58) who freely consented, and came for antenatal care visit at the maternity unit of the BRH, and had not been vaccinated against HBVwithin the study period; as well, the study excluded pregnant women less than 19 years and non-pregnant women. Those of them who had receive HBV vaccine at the period of the study were also excluded. The prospective health facility-based study setting was chosen because of the required study population of pregnant women who register for regular antenatal care visits. Moreover, data collected at a specific point in time was deemed adequate to establish a diagnosis of HBV virus, hence, justifying the choice of a cross-sectional study design, this is because testing for Hepatitis B and C virus has been made a routine test for all the pregnant women on their antenatal care visit at the Bertoua Regional Hospital and actually were free of charge during the study. A total of 400 pregnant women were consecutively sampled (non-probabilistic) registered for their visit during the study period and all were approached with a request and signed the informed consent to take part in the study, all of them gave their consent to participate in the study and they were consecutively enrolled to the study.

Participants were assigned codes for anonymity purposes, we used for HBV screening Diaspot HBsAg, these are step Hepatitis B Surface Antigen (HBsAg) test strip package insert and for HCV, Diaspot HCV virus anti-body (HCV-Ab) test strips. Those are immune-chromatographic strips for qualitative detection of antibodies and antigens. Their sensitivity and specificity are above 99% and 98% respectively. Results were disclosed to participants with proper counselling; all infected pregnant women were counselled on the disease and referred for proper specialization care while the non-infected were counselled for HBV vaccination. Data were obtained using a well-structured questionnaire which was designed for the research and for laboratory analysis; questions elicited data to cover the objectives of the study, the questionnaire included seven sections, each focusing on a particular aspect to answer the research questions and gaps as well. The quantitative part of the questionnaire featured MCQ and Likert-scale questions allowing participant to rate their experiences, knowledge, feelings and attitudes. The questionnaires also included open-ended questions inviting participants to give other factors not mentioned. Frequencies (sums and percentages) were calculated for the socio-demographic factors and the different attitudes, feelings and practices towards HBV and HCV.

Tables displaying the frequency distribution for knowledge, attitude and practice towards HBV and HCV were entered into graph, each of the

tables had frequencies for knowledge, maternal HBV/HCV preventive modes and practices, modes of transmission. Data were analyzed using Excel 2016 frequencies and percentages were determined.

4. RESULTS & DISCUSSION

[See Annex — Table 1: Socio-demographic information among pregnant women attending ANC]

The results showed that the majority of pregnant women (62%) were aged within the framed age (19-28), most of them (59.5%) were single, 32.5% of the pregnant women had no formal education, while 38.75% of them were housewives, 89.5% were Christian and more than half (76.25%) were multigravida.

[See Annex — Table 2: Hepatitis B vaccine awareness and completion percentage]

While (30,5%) of the pregnant women had no knowledge about hepatitis B vaccine, as well, (69,5%) and (6,5%) respectively had knowledge about hepatitis B vaccine and knew their HBV vaccination status; also, only (12,25%) had completed all their HBV vaccination doses, while (49%) had not completed theirs. HBV vaccination is the surest and premium means of HBV prevention,

[See Annex — Table 4: Hepatitis Screening and screening importance percentage]

Nearly all the pregnant women (91,75%) were unaware of the neonatal HBV vaccination and (78%) the antiviral therapy at 28 weeks, only (8,25%) were aware of the neonatal vaccination and (22%) for that of antiviral therapy at 28 weeks.

Preventing mother-to-child transmission (MTCT) of HBV involves universal maternal screening, antiviral prophylaxis (e.g., tenofovir) for high-viral-load mothers from week 28, and infant immunoprophylaxis (HBIG + vaccine) within 12 hours of birth, preventing >95% of cases. For HCV, universal screening is recommended, but no vaccine exists, and breastfeeding is safe unless nipples are damaged. According to our results, (69.5%) of the pregnant women are aware of HBV vaccination, but only (6.5%) of them knew their HBV vaccination status, nevertheless, (46.75%) of them recognized that HBV vaccine is the most effective to prevent HBV infection. A study of (Huang XX et al 2023) concluded that to prevent Mother-to-child-transmission of Hepatitis B, it is necessary to standardize the treatment of pregnant women with a high exposure of Hepatitis B, implement combined vaccination within 12 hours of birth and standardize the full course of Hepatitis B;

A study stipulated that in the absence of preventive measures, the probability of transmission from mother to child varies from 70% to 90% for women with a high HBV viral load (or who are HBeAg-positive), and from 10% to 40% for moms who are HBeAg negative. Even among newborns who receive the hepatitis B vaccine, high maternal quantities of HBV DNA (viral load) are linked to an increased risk of transmission. (Emily Henderson 2021). Hepatitis C virus (HCV) transfer from mother to child is rather rare. Antibody to HCV (anti-HCV) is found in 0.1 percent to 2.4 percent of pregnant women, while it is significantly higher in some endemic areas. The percentage of women with anti-HCV who have active viremia infection varies between 60% and 70%. (Emily Henderson 2021). In general, vertical transmission of HCV is thought to be a risk only for women with detectable HCV RNA during pregnancy. The meta-analysis by (Benova et al) included 15 studies with a total of 473 children born to women who were HCV-antibody-positive but RNA-negative. Only 1 of

the 473 children was diagnosed with vertically acquired HCV infection.

5. CONCLUSION

In conclusion and regarding our results, we noticed that Hepatitis among pregnant women at the Bertoua Regional Hospital had a lower awareness of HBV and HCV of maternal-foetal preventive measures, it still need some adjustments namely campaign sensitization about viral hepatitis B and C, recruitment of trained personnel in Hepathology within the hospital, effective communication and information of pregnant women, counselling through antenatal classes

REFERENCES

Alter et al (1976). Type B hepatitis: the infectivity of blood positive for e antigen and DNA polymerase after accidental needle stick exposure, N. Engl. J. Med, 295,909-913.

Anaedobe et al (2015). Prevalence, Socio-Demographic Features and Risk Factors of Hepatitis B Virus Infection among Pregnant Women in South-western Nigeria. The Pan African Medical Journal, 20, 406.

Ansari, et al (2023). Risk of early horizontal transmission of hepatitis B virus in children of uninfected mothers in sub-Saharan Africa: A systematic review and meta-analysis. Lancet Glob. Health 2023,

Cabot B, et al (1997). Structure of replicating hepatitis C virus (HCV) quasispecies in the liver may not be reflected by analysis of circulating HCV virions. J Virol71:1732–1734.

Delamare, H. et al (2024). Proportion of pregnant women with HBV infection eligible for antiviral prophylaxis to prevent vertical transmission: A systematic review and meta-analysis. JHEP Rep. 2024, 6, 101064.

Di Filippo Villa, et al (2023) D.; Navas, M.C. Vertical Transmission of Hepatitis B Virus-An Update. Microorganisms 2023, 11, 1140.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

El-Sayed, M. et al (2019). THU-137-DAA therapy in women of child bearing age: accidental conception during therapy and pregnancy outcome [abstract]. J. Hepatol. 70, e221 (2019).

El-Sayed, M. H.& Indolfi, (2020). G. Hepatitis C virus treatment in children: a challenge for hepatitis C virus elimination. Semin. Liver Dis. 40, 213–224 (2020).

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

F. Fiehn, et al (2024). Hepatitis C virus and hepatocellular carcinoma: carcinogenesis in the era of direct-acting antivirals.

Farooq H.Z. et al (2024). Risk factors for hepatocellular carcinoma associated with hepatitis C genotype 3 infection: a systematic review. World J. Gastrointest. Oncol. 2024;16(4):1596612. doi: 10.4251/wjgo. v16.i4.1596. 15 avr.

Fogel RS, Chappell CA. (2023). Hepatitis C virus in pregnancy: an opportunity to test and treat. Obstet Gynecol Clin North Am 50:363–373. doi: 10.1016/j.ogc.2023.02.008

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of HBV Neglected in Cameroon.MC Pregnancy and Childbirth, 13, 158.

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of HBV Neglected in Cameroon? BMC Pregnancy and Childbirth, 13, 158.

Freriksen, J. J. M. et al (2019). Review article: direct-acting antivirals for the treatment of HCV during pregnancy and lactation – implications for maternal dosing, foetal exposure, and safety for mother and child. Aliment. Pharmacol. Ther. 50, 738–750 (2019).

Funk, A. et al (2020). Efficacy and safety of antiviral prophylaxis during pregnancy to prevent mother-to-child transmission of hepatitis B virus: a systematic review and meta-analysis. Lancet Infect Dis. https://doi.org/10.1016/S1473-3099(20)30586-7 (202

G.M. Kassa, et al (2024). Prevalence, trends, and distribution of hepatitis C virus among the general

population in sub-Saharan Africa: a systematic review and meta-analysis.

Gross, M. S., Ruth, A. R. & Rasmussen, S. A. (2020). Respect women, promote health and reduce stigma: ethical arguments for universal hepatitis C screening in pregnancy. J. Med. Ethics 46, 674–677 (2020).

Hamadou N.H.M., al (2025). Traitement de l’Hépatite C de Génotype 1 par les antiviraux d’Action Directe au Cameroun : résultats préliminaires. HEALTH SCIENCES AND DISEASE [Internet] 2025.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Indolfi, G. et al (2019). Hepatitis B virus infection in children and adolescents. Lancet Gastroenterol. Hepatol. 4, 466–476 (2019).

Indolfi, G. et al (2019). Hepatitis C virus infection in children and adolescents. Lancet Gastroenterol. Hepatol. 4, 477–487 (2019).

Jiang, W. et al (2018). Sofosbuvir inhibits hepatitis A virus replication in vitro assessed by a cell-based fluorescent reporter system. Antivir. Res. 154, 51–57 (2018).

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Kaberg, M. & Weiland, O (2020). Hepatitis C elimination - macro-elimination. Liver Int. 40, 61–66 (2020).

Khan A., et al (2022). Core amino acid substitutions in HCV-3a isolates from Pakistan and opportunities for multi-epitopic vaccines. J. Biomol. Struct. Dyn. Mai. 2022;40(8):375368. doi: 10.1080/07391102.2020.1850353.

Knegendorf, L. et al (2018). Hepatitis E virus replication and interferon responses in human placental cells. Hepatol. Commun. 2, 173–187 (2018).

Kushner, T. & Terrault, N. A. (2019). Hepatitis C in pregnancy: a unique opportunity to improve the hepatitis C cascade of care. Hepatol. Commun. 3, 20–28 (2019).

L. Bhebhe, M. Anderson, S. Souda, W.T. Choga, E. Zumbi ka, Z.M. Shaver, et, al (2019). Molecular characterization of hepatitis C virus in liver disease patients in Botswana: a retrospective cross-sectional study

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Law M, et al (2008). Broadly neutralizing antibodies protect against hepatitis C virus quasispecies challenge. Nat Med14:25–27.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Li, M. et al (2020). Hepatitis E virus infection and its associated adverse feto-maternal outcomes among pregnant women in Qinhuangdao, China. J. Matern. Fetal Neonatal. Med. 33, 3647–3651 (2020).

Li, P. et al (2020). The global epidemiology of hepatitis E virus infection: a systematic review and meta-analysis. Liver Int. 40, 1516–1528 (2020).

M. Amougou-Atsama, P. Jean Adrien Atangana, D. Noah Noah, P. Fewou Moundipa, P. Pineau, R. Njouom (2020). The role of hepatitis C virus genotypes and core mutations in hepatocellular carcinoma in Cameroon.

Ma, X. et al (2018). Chronic hepatitis B virus infection and preterm labor(birth) in pregnant women–an updated systematic review and meta-analysis. J. Med. Virol. 90, 93–100 (2018).

N.H.M. Hamadou, et al (2025). Traitement de l’Hépatite C de Génotype 1 par les antiviraux d’Action Directe au Cameroun : résultats préliminaires. HEALTH SCIENCES AND DISEASE.

P.A. Tagnouokam-Ngoupo, M.N. Ngoufack, S. Kenmoe, S.F. Lissock, M. Amougou-Atsama, R. Banai, et al (2019). Hepatitis C virus genotyping based on Core and NS5B regions in Cameroonian patients.

P.A. TagnouokamNgoupo, et al. (2019). Hepatitis C virus genotyping based on Core and NS5B regions in Cameroonian patients.

Quaranta M.G., et al (2024). Reduction of the risk of hepatocellular carcinoma over time using direct-acting antivirals: a propensity score analysis of a

real-life cohort (PITER HCV) Viruses. 2024;16(5):682. doi: 10.3390/v16050682. 26 avr.

R. Njouom, et al (2003). Hepatitis C virus infection among pregnant women in Yaoundé, Cameroon: prevalence, viremia, and genotypes.

R. Njouom, et al (2012). Phylogeography, risk factors and genetic history of hepatitis C virus in Gabon, central Africa

R.D. Trimbitas, et al (2016). Molecular characterization of hepatitis C virus core region in Moroccan intravenous drug user

Ragusa R, Giorgianni G, Lupo L et al (2018). Healthcare-associated Clostridium difficile infection: role of correct hand hygiene in cross-infection control.

Revill, P. A. et al (2020). The evolution and clinical impact of hepatitis B virus genome diversity. Nat. Rev. Gastroenterol. Hepatol. 17, 618–634 (2020).

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Shimakawa, Y. et al (2019). Hepatitis B core-related antigen (HBcrAg): an alternative to HBV DNA to assess treatment eligibility in Africa. Clin. Infect. Dis. 70, 1442–1452 (2019).

Wang F, et al (2017). Post-vaccination serologic testing of infants born to hepatitis B surface antigen positive mothers in 4 provinces of China. Vaccine. 2017;35(33):4229–35.

Wang W, et al (2020). Dual-targeting nanoparticle vaccine elicits a therapeutic antibody response against chronic hepatitis B. Nat Nanotechnology. 2020; 15:406–16

Wong, et al (2023). Real-world treatment outcome with protease inhibitor direct-acting antiviral in advanced hepatitis C cirrhosis: a REAL-C study Hepatol Int. 2023/

World Health Organization (2016). global health sector strategy on viral hepatitis 2016–2021: towards ending viral hepatitis. Geneva: World Health Organization; 2016 (http://apps.who.int/iris/bitstream/10665/246177/1/WHO-HIV-2016.06-eng.pdf?ua=1, accessed 2 April 2020).

World Health Organization (2017). Elimination of mother to child transmission of HIV and syphilis. Geneva.

Zou H et al (2012) Virologic factors associated with failure to passive-active immunoprophylaxis in infants born to HBsAg-positive mothers. J Viral Hepat. 2012 Feb;19(2): e18-25

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

***Tatiana Jiengoué, Olivier Lieuga and Augustine Nji Asakizi**. (2026). Pregnant women awareness of HBV and HCV maternal-fetal preventive measures at the Bertoua Regional Hospital in the East Region of Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004002. www.iqresearchjournal.com*

ANNEXES

Annex I — Table 1: Socio-demographic information among pregnant women attending ANC

Variables					
Age	(19-28)	(29-38)	(39-48)	(49-58)	-
n	248	82	62	8	-
%	62%	20.5%	15.5%	2%	-
					-
Marital status	Single	Married	Divorced	Widow	-
n	238	112	33	17	-
%	59.5%	28%	8.25%	4.25%	_
Education level	No formal education	Primary school	Secondary school	College	Postgraduate
n	130	122	84	49	15
%	32.5%	30.5%	21%	12.25%	3.75%
Occupation	Housewife	Business	Framing	Government employed	-
n	155	135	68	42	0
%	38.75%	33.75%	17%	19.5%	0%
Religion	Christian	Muslim	_	_	-
n	358	42	-	-	-
%	89.5%	10.5%	-	-	-
Gravidity and parity	First pregnancy	More than one pregnancy	-	-	-
n	95	305	-	-	-
%	23.75%	76.25%	-	-	-

Annex II — Table 2: Hepatitis B vaccine awareness and completion percentage

Hepatitis B awareness	Yes	No	-	-
N	278	69.5%	-	-
%	12.2%	30.5%	-	-
Hepatitis B vaccine completion	Yes, completed	Yes, but not completed	No	I don't know
N	49	196	84	71
%	12.5%	49%	21%	17.75%

Annex III — Table 3 : Hepatitis Screening and screening importance percentage

Hepatitis B and C screening	Yes	No	I don't know
N	265	106	29
%	66.25%	26.5%	7.25%

Annex IV — Table 4: Neonatal vaccination program at 28 weeks of pregnancy awareness

Hepatitis B and C screening importance	Yes	No	Unsure
N	378	12	10
%	94.5%	3%	2.5%

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Identifying pregnancy risks complications due to maternal-fetal HBV and HCV at the Bertoua Regional Hospital in the East Region of Cameroon

Tatiana Jiengoué[a], Olivier Lieuga[a] and Augustine Nji Asakizi[a]

Affiliations

i. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Tatiana Jiengoué Tchakonang
Email: jiengouetatiana@kesmondsuniversity.org

Paper ID: IQRJ-V05I02-26005009

ABSTRACT

Results from a seven-year nationwide inpatient sample study demonstrate that HBV and HCV viruses are each associated with pregnancy complication. Maternal HBV or HCV carrier status is a cause for concern regarding both the course of pregnancy and the short-term perinatal outcomes. Previous data conflict regarding the association of chronic HBV and HCV with adverse pregnancy outcomes, according to investigator Bing Chen, who presented the findings at the American Association for the study of liver Diseases. The main aim of this study is to identify HBV/HCV pregnancy risks complications. This cross-sectional study at Bertoua Regional Hospital of Cameroon (June 2025-January 2026) aimed at identifying pregnancy risks complications due to maternal-fetal HBV and HCV at the Bertoua Regional Hospital in Cameroon of pregnant women attending the maternity unit. The majority of them (62%) were aged within (19-28). (86.20%) of the pregnant women who were tested positive had experienced low birth weight, (79.31%) have known preterm birth, (68.76%) had been admitted to the Neonatal intensive care unit while (65.51%) had children with congenital abnormalities, pregnant women having HCV were likely to develop risks complications like preterm birth, low weight birth due to the fact that HCV is generally associated with a higher risk of adverse pregnancy complications compared to HBV particularly regarding neonatal outcomes. However, data identifying maternal-fetal HBV and HCV pregnancy risks complications among pregnant women across both community and healthcare settings remain limited, pregnant women are sometimes unaware of their pregnancy risks in their conditions; hence the need of this research study which goes in straight line with the World Health Organization elimination goals on viral Hepatitis.

Keywords: *Identifying, Pregnancy,Risks complications, Maternal-fetal, Viral Hepatitis B and C, , Cameroon*

1. INTRODUCTION

HBV and HCV are acquired by contaminated blood product exposure, sexual activity or perinatal transmission. Although the prevalence of HBV is relatively low, in the US and generally with approximately 1 million Americans are chronically infected by HBV (Sorell MF et al 2009), it is more prevalent in East Africa (8%) (Ott JJ et al 2011), Southeast Africa and India 1 to 66% and Sub-Saharan Africa (8 to 12%) (Ott JJ et al 2011).

Globally, HBV and HCV cause chronic infections in 254 million and 50 million persons worldwide respectively and these diseases result in 1.1 million and 242.00 deaths annually respectively (WHO 2024). In developing countries like Asia and Africa, HBV and HCV infections are common (WHO 2016), the WHO reported that there were 1.2 million and 10 new cases of chronic HBV and HCV infections worldwide in 2022 respectively (WHO 2024). According to the WHO estimates between 3 and 4 million people acquired the disease annually with the majority of cases occurring in Africa (Schweitzer et al 2025) other evidence also showed that an estimated 7.1 million people are infected with HCV worldwide (Global Hepatitis Report 2017). Despite being highly contagious, HBV and HCV are still underdiagnosed and underreported in the majority of African countries (WHO 2013). Both HBV and HCV have the potential to be widespread and impact a variety of populations, such as those with HIV, healthcare professionals, the general population of blood donors, pregnant mothers and their children (Coppola N et al 2016). Numerous studies show that the prevalence of HBV and HCV in pregnant women is now seriously associated with public health (Pabsu R et al 2018). Mother-to-child transmission of HBV, which might be through intrauterine transmission is a common event and causes chronic infection of the virus (Zhao et al 2022). The seroprevalence of HBV among pregnant women in Ethiopia ranges from 4.5 to 7.9% which is indicative of an intermediate degree of endemicity for the virus (Umare A et al 2026). The seroprevalence of HCV, which ranges from 0.26 to 8.07% was shown to be lower than that of HBV in the majority of Ethiopian studies (Bafa TA et al 2020). The seroprevalence of HBV and HCV among pregnant women in Cameroon is high with studies reporting rates generally ranging from 5% to over 16% (Mbongue-Mikangue et al 2024). Childbearing women can potentially transmit HBV and HCV to their children, they transmit an infection to new-borns usually during birth following close contact. There is a higher likelihood of vertical transmission of infection from mothers to offspring in 4.6% and 1.6% of babies delivered to pregnant women with HBV and HCV infections respectively (Dagnew et al 2020). New-borns who are exposed to HBV will have almost 85-90% risk of developing chronic liver disease (Brian J.Mc Mahon et al 1990).

Preterm delivery, placental separation, vaginal bleeding, early rupture of the membranes and mortality are among the many issues that can arise from maternal infection with HBV and HCV during pregnancy (Reddick K, et al 2011). A significant risk of neonatal hepatitis is also linked to it, and this can result in liver cirrhosis and hepatocellular cancer in young adulthood (Zahran et al 2010), eventhough viral hepatitis screening is advised during routine ANC, it is not always effective in some healthcare facilities in Cameroon. Recent research showed that both the general population and healthcare professionals reported having little knowledge of the hepatitis virus and its risks complications

on pregnancy (Shiferaw F 2016). Therefore, this study aimed at identifying the pregnancy risks complications due to HBV and HCV of pregnant women attending antenatal classes at the Bertoua Regional Hospital, in other words, what knowledge do pregnant women have as far as HBV and HCV is concerned in their condition?

2. RELATED WORKS

Childbearing women can potentially transmit HBV and HCV to their children, they transmit an infection to new-borns usually during birth following close contact. There is a higher likelihood of vertical transmission of infection from mothers to offspring in 4.6% and 1.6% of babies delivered to pregnant women with HBV and HCV infections respectively (Dagnew et al 2020). New-borns who are exposed to HBV will have almost 85-90% risk of developing chronic liver disease (Brian J.Mc Mahon et al 1990).

A study showed that Preterm delivery, placental separation, vaginal bleeding, early rupture of the membranes and mortality are among the many issues that can arise from maternal infection with HBV and HCV during pregnancy (Reddick K, et al 2011). A significant risk of neonatal hepatitis is also linked to it, and this can result in liver cirrhosis and hepatocellular cancer in young adulthood (Zahran et al 2010), eventhough viral hepatitis screening is advised during routine ANC, it is not always effective in some healthcare facilities in Cameroon. Recent research showed that both the general population and healthcare professionals reported having little knowledge of the hepatitis virus and its risks complications on pregnancy (Shiferaw F 2016).

3. MATERIALS & METHODS

Inclusion criteria were Pregnant women aged of 19 years and above (58) who freely consented, and came for antenatal care visit at the maternity unit of the BRH, and had not been vaccinated against HBVwithin the study period; as well, the study excluded pregnant women less than 19 years and non-pregnant women. Those of them who had receive HBV vaccine at the period of the study were also excluded. The prospective health facility-based study setting was chosen because of the required study population of pregnant women who register for regular antenatal care visits. Moreover, data collected at a specific point in time was deemed adequate to establish a diagnosis of HBV virus, hence, justifying the choice of a cross-sectional study design, this is because testing for Hepatitis B and C virus has been made a routine test for all the pregnant women on their antenatal care visit at the Bertoua Regional Hospital and actually were free of charge during the study. A total of 400 pregnant women were consecutively sampled (non-probabilistic) registered for their visit during the study period and all were approached with a request and signed the informed consent to take part in the study, all of them gave their consent to participate in the study and they were consecutively enrolled to the study.

Participants were assigned codes for anonymity purposes, we used for HBV screening Diaspot HBsAg, these are step Hepatitis B Surface Antigen (HBsAg) test strip package insert and for HCV, Diaspot HCV virus anti-body (HCV-Ab) test strips. Those are immune-chromatographic strips for qualitative detection of antibodies and antigens. Their sensitivity and specificity are above 99% and 98% respectively. Results were disclosed to participants with proper counselling; all infected pregnant women were counselled on the disease and referred for proper specialization care while the non-infected were counselled for HBV vaccination.

Data were obtained using a well-structured questionnaire which was designed for the research and for laboratory analysis; questions elicited data to cover the objectives of the study, the questionnaire included seven sections, each focusing on a particular aspect to answer the research questions and gaps as well. The quantitative part of the questionnaire featured MCQ and Likert-scale questions allowing participant to rate their experiences, knowledge, feelings and attitudes. The questionnaires also included open-ended questions inviting participants to give other factors not mentioned. Frequencies (sums and percentages) were calculated for the socio-demographic factors and the different attitudes, feelings and practices towards HBV and HCV.

Tables displaying the frequency distribution for knowledge, attitude and practice towards HBV and HCV were entered into graph, each of the tables had frequencies for knowledge, maternal HBV/HCV preventive modes and practices, modes of transmission. Data were analyzed using Excel 2016 frequencies and percentages were determined.

4. RESULTS & DISCUSSION

[See Annex — Table 1: Socio-demographic information and prevalence rates among pregnant women attending ANC]

The results show that among the HBV/HCV women tested positive (29), (79.31%) of them experienced preterm birth in the past due to their conditions, (86.20%) experienced low birth weight of their babies, (65.51%) congenital abnormalities and (68.76%) of them were admitted to the NICU with their babies.

As results, we found out that pregnant women with HBV were significantly more likely to give birth to a baby born preterm (86.20%) and (79.3%) of the babies born to their positive mothers had a low birth weight (<2500g), (68.76%) were admitted to the NICU and (65.51%) of those babies had congenital abnormalities; HBV and HCV infections during pregnancy are linked to significant maternal and fetal complications including increased risks of preterm birth, low birth weight, gestational diabetes and intrauterine restriction. (Statpearls-NIH 2025) confirmed HBV/HCV increase risks of preterm birth, placental issues and prenatal mortality and maternal HCV is strongly associated with preterm birth, with up to a two-fold increased risks of NICU admission and this in straight line with our findings in which HBV pregnant women are more likely to give birth to a baby born preterm (86.20%). Another study is that of (He et al 2023) which demonstrated that HBV is an independent risk factor for preterm birth and gestational diabetes, HBV infection is associated with an increased risk of GDM, even after adjusting for traditional factors like age and BMI, this is in phase with phase with our study whose results showed that 58.62% of pregnant women living with HBV/HCV are at a higher risk of developing gestational diabetes; our results found that 68.76% of babies born to either HBV/HCV mothers admitted to NICU at birth, this is in phase with the study of (Thakur HS et al 2017)

in which 27.27% of pregnant women had their babies admitted to NICU. Also, 65.5% of our HBV/HCV pregnant women were likely to give birth to a baby with congenital abnormalities as it was the case in the study of (Shiyao et al 2019) in which the results showed that pregnant women with HBV infection might be associated with a higher risk of congenital abnormalities.

5. CONCLUSION

Maternal-fetal HBV and HCV infections significantly increase the risk of adverse obstetric and neonatal outcomes, including preterm birth, low birth weight, gestational diabetes, and miscarriage. Chronic HBV infection poses a high risk of vertical transmission, particularly with high maternal viral loads, necessitating timely antiviral therapy.

While HBV has established protocols for preventing transmission, further research is needed to better understand the mechanisms of adverse pregnancy outcomes and to optimize the management of HCV in pregnancy.

REFERENCES

Alter et al (1976). Type B hepatitis: the infectivity of blood positive for e antigen and DNA polymerase after accidental needle stick exposure, N. Engl. J. Med, 295,909-913.

Anaedobe et al (2015). Prevalence, Socio-Demographic Features and Risk Factors of Hepatitis B Virus Infection among Pregnant Women in South-western Nigeria. The Pan African Medical Journal, 20, 406.

Ansari, et al (2023). Risk of early horizontal transmission of hepatitis B virus in children of uninfected mothers in sub-Saharan Africa: A systematic review and meta-analysis. Lancet Glob. Health 2023,

Cabot B, et al (1997). Structure of replicating hepatitis C virus (HCV) quasispecies in the liver may not be reflected by analysis of circulating HCV virions. J Virol71:1732–1734.

Delamare, H. et al (2024). Proportion of pregnant women with HBV infection eligible for antiviral prophylaxis to prevent vertical transmission: A systematic review and meta-analysis. JHEP Rep. 2024, 6, 101064.

Di Filippo Villa, et al (2023) D.; Navas, M.C. Vertical Transmission of Hepatitis B Virus-An Update. Microorganisms 2023, 11, 1140.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

El-Sayed, M. et al (2019). THU-137-DAA therapy in women of child bearing age: accidental conception during therapy and pregnancy outcome [abstract]. J. Hepatol. 70, e221 (2019).

El-Sayed, M. H.& Indolfi, (2020). G. Hepatitis C virus treatment in children: a challenge for hepatitis C virus elimination. Semin. Liver Dis. 40, 213–224 (2020).

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

F. Fiehn, et al (2024). Hepatitis C virus and hepatocellular carcinoma: carcinogenesis in the era of direct-acting antivirals.

Farooq H.Z. et al (2024). Risk factors for hepatocellular carcinoma associated with hepatitis C genotype 3 infection: a systematic review. World J. Gastrointest. Oncol. 2024;16(4):1596612. doi: 10.4251/wjgo. v16.i4.1596. 15 avr.

Fogel RS, Chappell CA. (2023). Hepatitis C virus in pregnancy: an opportunity to test and treat. Obstet Gynecol Clin North Am 50:363–373. doi: 10.1016/j.ogc.2023.02.008

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of HBV Neglected in Cameroon.MC Pregnancy and Childbirth, 13, 158.

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of HBV Neglected in Cameroon? BMC Pregnancy and Childbirth, 13, 158.

Freriksen, J. J. M. et al (2019). Review article: direct-acting antivirals for the treatment of HCV during pregnancy and lactation – implications for maternal dosing, foetal exposure, and safety for mother and child. Aliment. Pharmacol. Ther. 50, 738–750 (2019).

Funk, A. et al (2020). Efficacy and safety of antiviral prophylaxis during pregnancy to prevent mother-to-child transmission of hepatitis B virus: a systematic review and meta-analysis. Lancet Infect Dis. https://doi.org/10.1016/S1473-3099(20)30586-7 (202

G.M. Kassa, et al (2024). Prevalence, trends, and distribution of hepatitis C virus among the general

population in sub-Saharan Africa: a systematic review and meta-analysis.

Gross, M. S., Ruth, A. R. & Rasmussen, S. A. (2020). Respect women, promote health and reduce stigma: ethical arguments for universal hepatitis C screening in pregnancy. J. Med. Ethics 46, 674–677 (2020).

Hamadou N.H.M., al (2025). Traitement de l’Hépatite C de Génotype 1 par les antiviraux d’Action Directe au Cameroun : résultats préliminaires. HEALTH SCIENCES AND DISEASE [Internet] 2025.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Indolfi, G. et al (2019). Hepatitis B virus infection in children and adolescents. Lancet Gastroenterol. Hepatol. 4, 466–476 (2019).

Indolfi, G. et al (2019). Hepatitis C virus infection in children and adolescents. Lancet Gastroenterol. Hepatol. 4, 477–487 (2019).

Jiang, W. et al (2018). Sofosbuvir inhibits hepatitis A virus replication in vitro assessed by a cell-based fluorescent reporter system. Antivir. Res. 154, 51–57 (2018).

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Kaberg, M. & Weiland, O (2020). Hepatitis C elimination - macro-elimination. Liver Int. 40, 61–66 (2020).

Khan A., et al (2022). Core amino acid substitutions in HCV-3a isolates from Pakistan and opportunities for multi-epitopic vaccines. J. Biomol. Struct. Dyn. Mai. 2022;40(8):375368. doi: 10.1080/07391102.2020.1850353.

Knegendorf, L. et al (2018). Hepatitis E virus replication and interferon responses in human placental cells. Hepatol. Commun. 2, 173–187 (2018).

Kushner, T. & Terrault, N. A. (2019). Hepatitis C in pregnancy: a unique opportunity to improve the hepatitis C cascade of care. Hepatol. Commun. 3, 20–28 (2019).

L. Bhebhe, M. Anderson, S. Souda, W.T. Choga, E. Zumbi ka, Z.M. Shaver, et, al (2019). Molecular characterization of hepatitis C virus in liver disease patients in Botswana: a retrospective cross-sectional study

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Law M, et al (2008). Broadly neutralizing antibodies protect against hepatitis C virus quasispecies challenge. Nat Med14:25–27.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Li, M. et al (2020). Hepatitis E virus infection and its associated adverse feto-maternal outcomes among pregnant women in Qinhuangdao, China. J. Matern. Fetal Neonatal. Med. 33, 3647–3651 (2020).

Li, P. et al (2020). The global epidemiology of hepatitis E virus infection: a systematic review and meta-analysis. Liver Int. 40, 1516–1528 (2020).

M. Amougou-Atsama, P. Jean Adrien Atangana, D. Noah Noah, P. Fewou Moundipa, P. Pineau, R. Njouom (2020). The role of hepatitis C virus genotypes and core mutations in hepatocellular carcinoma in Cameroon.

Ma, X. et al (2018). Chronic hepatitis B virus infection and preterm labor(birth) in pregnant women–an updated systematic review and meta-analysis. J. Med. Virol. 90, 93–100 (2018).

N.H.M. Hamadou, et al (2025). Traitement de l’Hépatite C de Génotype 1 par les antiviraux d’Action Directe au Cameroun : résultats préliminaires. HEALTH SCIENCES AND DISEASE.

P.A. Tagnouokam-Ngoupo, M.N. Ngoufack, S. Kenmoe, S.F. Lissock, M. Amougou-Atsama, R. Banai, et al (2019). Hepatitis C virus genotyping based on Core and NS5B regions in Cameroonian patients.

P.A. TagnouokamNgoupo, et al. (2019). Hepatitis C virus genotyping based on Core and NS5B regions in Cameroonian patients.

Quaranta M.G., et al (2024). Reduction of the risk of hepatocellular carcinoma over time using direct-acting antivirals: a propensity score analysis of a

real-life cohort (PITER HCV) Viruses. 2024;16(5):682. doi: 10.3390/v16050682. 26 avr.

R. Njouom, et al (2003). Hepatitis C virus infection among pregnant women in Yaoundé, Cameroon: prevalence, viremia, and genotypes.

R. Njouom, et al (2012). Phylogeography, risk factors and genetic history of hepatitis C virus in Gabon, central Africa

R.D. Trimbitas, et al (2016). Molecular characterization of hepatitis C virus core region in Moroccan intravenous drug user

Ragusa R, Giorgianni G, Lupo L et al (2018). Healthcare-associated Clostridium difficile infection: role of correct hand hygiene in cross-infection control.

Revill, P. A. et al (2020). The evolution and clinical impact of hepatitis B virus genome diversity. Nat. Rev. Gastroenterol. Hepatol. 17, 618–634 (2020).

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Shimakawa, Y. et al (2019). Hepatitis B core-related antigen (HBcrAg): an alternative to HBV DNA to assess treatment eligibility in Africa. Clin. Infect. Dis. 70, 1442–1452 (2019).

Wang F, et al (2017). Post-vaccination serologic testing of infants born to hepatitis B surface antigen positive mothers in 4 provinces of China. Vaccine. 2017;35(33):4229–35.

Wang W, et al (2020). Dual-targeting nanoparticle vaccine elicits a therapeutic antibody response against chronic hepatitis B. Nat Nanotechnology. 2020; 15:406–16

Wong, et al (2023). Real-world treatment outcome with protease inhibitor direct-acting antiviral in advanced hepatitis C cirrhosis: a REAL-C study Hepatol Int. 2023/

World Health Organization (2016). global health sector strategy on viral hepatitis 2016–2021: towards ending viral hepatitis. Geneva: World Health Organization; 2016 (http://apps.who.int/iris/bitstream/10665/246177/1/WHO-HIV-2016.06-eng.pdf?ua=1, accessed 2 April 2020).

World Health Organization (2017). Elimination of mother to child transmission of HIV and syphilis. Geneva.

Zou H et al (2012) Virologic factors associated with failure to passive-active immunoprophylaxis in infants born to HBsAg-positive mothers. J Viral Hepat. 2012 Feb;19(2): e18-25

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Tatiana Jiengoué, Olivier Lieuga and Augustine Nji Asakizi (2026). Identifying pregnancy risks complications due to maternal-fetal HBV and HCV at the Bertoua Regional Hospital in the East Region of Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004003. www.iqresearchjournal.com

ANNEXES

Annex I — Table 1: Socio-demographic information and prevalence rates among pregnant women attending ANC

Variables					
Age	(19-28)	(29-38)	(39-48)	(49-58)	-
n	248	82	62	8	-
%	62%	20.5%	15.5%	2%	-
Marital status	Single	Married	Divorced	Widow	-
n	238	112	33	17	-
%	59.5%	28%	8.25%	4.25%	-
Education level	No formal education	Primary school	Secondary school	College	Postgraduate
n	130	122	84	49	15
%	32.5%	30.5%	21%	12.25%	3.75%
Occupation	Housewife	Business	Framing	Government employed	-
n	155	135	68	42	-
%	38.75%	33.75%	17%	19.5%	-
Religion	Christian	Muslim	_	_	-
n	358	42	0	0	-
%	89.5%	10.5%	0%	0%	-
Gravidity and parity	First pregnancy	More than one pregnancy	-	-	-
n	95	305	-	-	-
%	23.75%	76.25%	-	-	-

Annex II — Table 2: HBV/HCV positive mothers experienced pregnancy risk complications percentage

Hepatitis B and C pregnancy risk complications	Preterm birth	Low birth weight	Congenital abnormalities	NICU
Yes	23	25	19	20
n				
%	79.31%	86.20%	65.51%	68.76%
No				
n	6	4	10	9
%	20.69%	13.79%	34.49%	31.24%

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Assessing pregnant women's knowledge of HBV and HCV Routes of transmission at the Bertoua Regional Hospital in the East Region of Cameroon

Tatiana Jiengoué[a,*], Olivier Lieuga[a] and Augustine Nji Asakizi[a]

Affiliations

j. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Tatiana Jiengoué Tchakonang
Email: jiengouetatiana@kesmondsuniversity.org

Paper ID: IQRJ-V05I02-26005010

ABSTRACT

Studies in Cameroon indicate generally poor knowledge and low awareness of viral Hepatitis among pregnant women, despite high endemicity, viral Hepatitis is challenging the health condition of the people around the world, and is considered a serious public global health problem of the human kind in the 21th century. In the globe, there are two well-known forms of chronic Hepatitis and this corresponds to Hepatitis B and Hepatitis C. This cross-sectional study at Bertoua Regional Hospital of Cameroon (June 2025-January 2026) aimed at identifying HBV and HCV routes of transmission among pregnant women at the Bertoua Regional Hospital in Cameroon attending the maternity unit. the majority of them (62%) were aged within (19-28). (66.5%) of the pregnant women had knowledge about HBV or HCV routes of transmission, (49.5%) of them had never heard about the Mother-to-child-transmission expression, to the question of how can Hepatitis B or C be transmitted from one person to the other, nearly all of them were awaaer and only (3%) had no knowledge about it, almost half (46%) of the pregnant women knew that breastfeeding is safe as long as nipples are not bleeding,, (19.75%) of them said the baby transmission through C-section is not possible, while (52,(%) of them agreed that a HBVor HCV mother can transmit the virus to her baby during pregnancy or delivery, still, (62%) of them are of the view that HBV or HCV can be spread by sharing personal items like a toothbrush or razor. However, data identifying pregnant women knowledge on HBV or HVC routes of transmission among pregnant women across both community and healthcare settings remain limited, hence the need of this research. The findings align with the World Health Organization as well as others organisms fighting for the prevention and elimination of viral Hepatitis such as the Centre of Disease Control and Prevention

Keywords : *knowledge, HBV and HCV , Routes of transmission ,Pregnant women ,Cameroon*

1. INTRODUCTION

HBV and HCV are acquired by contaminated blood product exposure, sexual activity or perinatal transmission. Although the prevalence of HBV is relatively low, in the US and generally with approximately 1 million Americans are chronically infected by HBV (Sorell MF et al 2009), it is more prevalent in East Africa (8%) (Ott JJ et al 2011), Southeast Africa and India 1 to 66% and Sub-Saharan Africa (8 to 12%) (Ott JJ et al 2011).

Viral Hepatitis is challenging the health condition of the people around the World and is considered a serious public global health problem of the human kind in the 21th century. Hepatitis B virus is a member of the Hepanaviridae family, it is a DNA virus with partially double-stranded DNA and a core antigen surrounded by a shell containing hepatitis B surface antigen (HBsAg). Despite availability of a vaccine and antiviral treatment, HBV infection is still a major health problem causing considerable morbidity and mortality (Chaiba et al; 2015). A systematic review by (Mahamat et al; 2021) estimated that the prevalence of Hepatitis B in Cameroon was low, with overall 2.3% all ages? In 2015, the WHO estimates that 257 million people were living with chronic Hepatitis B including 65 million women of childbearing age, with estimated number of deaths at 887000 mainly due to HCC (Angeles et al; 2020).

Hepatitis C virus is an RNA virus of the flaviviridae family and appears to have humans and chimpazees as the species susceptible to infection (Samuel et al;2004), about 170 million people are infected with HCV worldwide (Obi et al; 2006). In Cameroon, a study of (Mounchili et al; 2024) found anti-HCV overall prevalence of 13.50%; HBV and HCV can be prevalent and affect a wide range of population that include human immune deficiency virus (HIV) infected individuals, healthcare workers, blood donors, pregnant mothers, their children (Elkhateeb et al; 2018); various studies indicate that the prevalence of HBV and HCV among pregnant women becomes serious public health importance (Dabsu et al; 2019). In Cameroon, a study of (Frambo et al; 2014) reported a HBV prevalence in pregnant women of 9.7%, while in 2018, (Fouelifack et al; 2018) reported a 1.6% HCV pregnant women Mother-to-child transmission of HBV which might be via uterine transmission is a common phenomenon and causes chronic infection of the virus (Zhao et al; 2022); it is one of the main routes of transmission worldwide, despite the proven effectiveness of immunoprophylaxis, in particular the birth dose vaccine. Mother-to-child transmission can occur during pregnancy, during delivery or during breastfeeding (Mendoua et al; 2013); mother-to-child transmission is responsible for more than third chronic viral hepatitis cases (Noele teal; 2016) other routes of transmission include: having history of polysexual practices, previous history of dental procedures, health facility admission, blood transfusion (Mamuye et al; 2020). Overall, there is a paucity of research specifically addressing pregnant women and routes of transmission identification in Cameroon. Therefore, this study aims at identifying the various HBV and HCV routes of transmission among pregnant women at the Bertoua Regional Hospital in Cameroon, in other words, what knowledge do pregnant women have of Hepatitis B and C routes of transmission?

2. RELATED WORKS

Childbearing women can potentially transmit HBV and HCV to their children, they transmit an

infection to new-borns usually during birth following close contact. There is a higher likelihood of vertical transmission of infection from mothers to offspring in 4.6% and 1.6% of babies delivered to pregnant women with HBV and HCV infections respectively (Dagnew et al 2020). New-borns who are exposed to HBV will have almost 85-90% risk of developing chronic liver disease (Brian J.Mc Mahon et al 1990).

Vertical transmission is common among asymptomatic female carries who are unaware of their status, in cases of high viral multiplication in the mother, and in the absence of serovaccination, 90% infected newborns are likely to develop chronic Hepatitis B (Sogni et al; 2015) and have a much higher risk of developing liver disease, including HCC in adulthood (Dong et al; 2015).

A Cameroonian previous study of (Mbongue et al; 2024), a prospective, cross multicenter study was conducted from 17 September 2018 to 25 February 2019 in 102 pregnant women aged 15 to 44 years. The results showed out that scarification and tattooing were significantly associated with HBV infection and previous contact with HBV.

Another study of (Ayenew et al; 2023), A cross-sectional study conducted from March 15th to September 16th, 2022, at the Debre Tabor Comprehensive Specialized Hospital antenatal care clinic. The results showed out that tattooing and dental therapy were significantly associated with HBV infection.

3. MATERIALS & METHODS

Inclusion criteria were Pregnant women aged of 19 years and above (58) who freely consented, and came for antenatal care visit at the maternity unit of the BRH, and had not been vaccinated against HBVwithin the study period; as well, the study excluded pregnant women less than 19 years and non-pregnant women. Those of them who had receive HBV vaccine at the period of the study were also excluded. The prospective health facility-based study setting was chosen because of the required study population of pregnant women who register for regular antenatal care visits. Moreover, data collected at a specific point in time was deemed adequate to establish a diagnosis of HBV virus, hence, justifying the choice of a cross-sectional study design, this is because testing for Hepatitis B and C virus has been made a routine test for all the pregnant women on their antenatal care visit at the Bertoua Regional Hospital and actually were free of charge during the study. A total of 400 pregnant women were consecutively sampled (non-probabilistic) registered for their visit during the study period and all were approached with a request and signed the informed consent to take part in the study, all of them gave their consent to participate in the study and they were consecutively enrolled to the study.

Participants were assigned codes for anonymity purposes, we used for HBV screening Diaspot HBsAg, these are step Hepatitis B Surface Antigen (HBsAg) test strip package insert and for HCV, Diaspot HCV virus anti-body (HCV-Ab) test strips. Those are immune-chromatographic strips for qualitative detection of antibodies and antigens. Their sensitivity and specificity are above 99% and 98% respectively. Results were disclosed to participants with proper counselling; all infected pregnant women were counselled on the disease and referred for proper specialization care while the non-infected were counselled for HBV vaccination. Data were obtained using a well-structured questionnaire which was designed for the research and for laboratory analysis; questions

elicited data to cover the objectives of the study, the questionnaire included seven sections, each focusing on a particular aspect to answer the research questions and gaps as well. The quantitative part of the questionnaire featured MCQ and Likert-scale questions allowing participant to rate their experiences, knowledge, feelings and attitudes. The questionnaires also included open-ended questions inviting participants to give other factors not mentioned. Frequencies (sums and percentages) were calculated for the socio-demographic factors and the different attitudes, feelings and practices towards HBV and HCV.

Tables displaying the frequency distribution for knowledge, attitude and practice towards HBV and HCV were entered into graph, each of the tables had frequencies for knowledge, maternal HBV/HCV preventive modes and practices, modes of transmission. Data were analyzed using Excel 2016 frequencies and percentages were determined

4. RESULTS & DISCUSSION

[See Annex — Table 1: Socio-demographic information and prevalence rates among pregnant women attending ANC]

The results showed that the majority of pregnant women (62%) were aged within the framed age (19-28), most of them (59.5%) were single, (32.5%) of the pregnant women had no formal education, while (38.75%) of them were housewives, (89.5%) were Christian and more than half (76.25%) were multigravida that is they were pregnant more than once

[See Annex — Table 2: HBV/HCV Routes of transmission among pregnant women at the BRH]

(66.5%) of the pregnant women had some knowledge about Hepatitis B and C transmission, (49.5%) of them were not aware of the expression Mother-to-child-transmission, to the question of how can Hepatitis B or C be transmitted from one person to the other, nearly all the pregnant women were right and aware, just 3% had no knowledge about it, almost half (46%) of the pregnant women knew breastfeeding is safe unless the nipples are not bleeding, (19.75%) of the pregnant women said a new-born baby cannot be transmitted HCV through C-section; while (52.5%) agreed that HBV/HCV mothers cant transmit the virus to her baby during pregnancy or delivery, (62.%) are of the view that HBV/HCV can be spread by sharing personal items like toothbrush or razor, the study showed a strong relationship between education, religion, and pregnant women awareness about HBV/HCV routes of transmission.

We found that (66.5%) of the pregnant women were able to identify HBV/HCV routes of transmission, and almost half (49.59%) of them were not knowledgeable about the expression Mother-to-child transmission, this was the case in the study of (Anyalem et al;2025), among the 419 pregnant women attending ANC at that Hospital, (80.7%) were not knowledgeable about the HBV vertical transmission, the overall knowledge of vertical transmission was poor (23.75%) of the pregnant women said that Hepatitis C can be passed from one person to another by sharing needles, this study is similar, to that of (Ephraim et al; 2015), a cross sectional study of 168 pregnant women recruited from the Agogo Presbyterian Hospital, in which the results showed that sharing needles, were associated with Hepatitis C infection). Also, a study of (Ivan et al; 2014) showed that a proportion of babies (as high as 34%) may acquire infection after birth due to close contact with the mother, while it is the contrary in our study, in which pregnant women are not aware

of the baby's risks of infection after his birth from an infected HBV mother. Another study is that of (Arzu et al; 2009), in which (90%) of the healthcare staff was aware of the sexual transmission route, which was not actually the case with our study in which just (15.25%) of the pregnant women were aware that Hepatitis C can be transmitted through sex, also, (62%) of the pregnant women were aware that Hepatitis B/C can be spread by sharing personal items like a toothbrush or razor, this actually was the case in (Arzu et al; 2009) in which (94%) of the healthcare workers were aware of the transmission rules by sharing personal items such as toothbrushes, razors, and nail scissors.

5. CONCLUSION

In conclusion, we found adding to our observations that pregnant women do have some general knowledge on routes of transmission viral Hepatitis, but their knowledge is superficial, we also noticed that the quasi-inexistence of antenatal classes within the hospital and lack of trained personnel in Hepatology might justify some unawareness.

So efforts should be addressed on counselling.

REFERENCES

Alter et al (1976). Type B hepatitis: the infectivity of blood positive for e antigen and DNA polymerase after accidental needle stick exposure, N. Engl. J. Med, 295,909-913.

Anaedobe et al (2015). Prevalence, Socio-Demographic Features and Risk Factors of Hepatitis B Virus Infection among Pregnant Women in South-western Nigeria. The Pan African Medical Journal, 20, 406.

Ansari, et al (2023). Risk of early horizontal transmission of hepatitis B virus in children of uninfected mothers in sub-Saharan Africa: A systematic review and meta-analysis. Lancet Glob. Health 2023,

Cabot B, et al (1997). Structure of replicating hepatitis C virus (HCV) quasispecies in the liver may not be reflected by analysis of circulating HCV virions. J Virol71:1732–1734.

Delamare, H. et al (2024). Proportion of pregnant women with HBV infection eligible for antiviral prophylaxis to prevent vertical transmission: A systematic review and meta-analysis. JHEP Rep. 2024, 6, 101064.

Di Filippo Villa, et al (2023) D.; Navas, M.C. Vertical Transmission of Hepatitis B Virus-An Update. Microorganisms 2023, 11, 1140.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

El-Sayed, M. et al (2019). THU-137-DAA therapy in women of child bearing age: accidental conception during therapy and pregnancy outcome [abstract]. J. Hepatol. 70, e221 (2019).

El-Sayed, M. H.& Indolfi, (2020). G. Hepatitis C virus treatment in children: a challenge for hepatitis C virus elimination. Semin. Liver Dis. 40, 213–224 (2020).

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

F. Fiehn, et al (2024). Hepatitis C virus and hepatocellular carcinoma: carcinogenesis in the era of direct-acting antivirals.

Farooq H.Z. et al (2024). Risk factors for hepatocellular carcinoma associated with hepatitis C genotype 3 infection: a systematic review. World J. Gastrointest. Oncol. 2024;16(4):1596612. doi: 10.4251/wjgo. v16.i4.1596. 15 avr.

Fogel RS, Chappell CA. (2023). Hepatitis C virus in pregnancy: an opportunity to test and treat. Obstet Gynecol Clin North Am 50:363–373. doi: 10.1016/j.ogc.2023.02.008

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of HBV Neglected in Cameroon.MC Pregnancy and Childbirth, 13, 158.

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of

HBV Neglected in Cameroon? BMC Pregnancy and Childbirth, 13, 158.

Freriksen, J. J. M. et al (2019). Review article: direct-acting antivirals for the treatment of HCV during pregnancy and lactation – implications for maternal dosing, foetal exposure, and safety for mother and child. Aliment. Pharmacol. Ther. 50, 738–750 (2019).

Funk, A. et al (2020). Efficacy and safety of antiviral prophylaxis during pregnancy to prevent mother-to-child transmission of hepatitis B virus: a systematic review and meta-analysis. Lancet Infect Dis. https://doi.org/10.1016/S1473-3099(20)30586-7 (202

G.M. Kassa, et al (2024). Prevalence, trends, and distribution of hepatitis C virus among the general population in sub-Saharan Africa: a systematic review and meta-analysis.

Gross, M. S., Ruth, A. R. & Rasmussen, S. A. (2020). Respect women, promote health and reduce stigma: ethical arguments for universal hepatitis C screening in pregnancy. J. Med. Ethics 46, 674–677 (2020).

Hamadou N.H.M., al (2025). Traitement de l'Hépatite C de Génotype 1 par les antiviraux d'Action Directe au Cameroun : résultats préliminaires. HEALTH SCIENCES AND DISEASE [Internet] 2025.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Indolfi, G. et al (2019). Hepatitis B virus infection in children and adolescents. Lancet Gastroenterol. Hepatol. 4, 466–476 (2019).

Indolfi, G. et al (2019). Hepatitis C virus infection in children and adolescents. Lancet Gastroenterol. Hepatol. 4, 477–487 (2019).

Jiang, W. et al (2018). Sofosbuvir inhibits hepatitis A virus replication in vitro assessed by a cell-based fluorescent reporter system. Antivir. Res. 154, 51–57 (2018).

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Kaberg, M. & Weiland, O (2020). Hepatitis C elimination - macro-elimination. Liver Int. 40, 61–66 (2020).

Khan A., et al (2022). Core amino acid substitutions in HCV-3a isolates from Pakistan and opportunities for multi-epitopic vaccines. J. Biomol. Struct. Dyn. Mai. 2022;40(8):375368. doi: 10.1080/07391102.2020.1850353.

Knegendorf, L. et al (2018). Hepatitis E virus replication and interferon responses in human placental cells. Hepatol. Commun. 2, 173–187 (2018).

Kushner, T. & Terrault, N. A. (2019). Hepatitis C in pregnancy: a unique opportunity to improve the hepatitis C cascade of care. Hepatol. Commun. 3, 20–28 (2019).

L. Bhebhe, M. Anderson, S. Souda, W.T. Choga, E. Zumbi ka, Z.M. Shaver, et, al (2019). Molecular characterization of hepatitis C virus in liver disease patients in Botswana: a retrospective cross-sectional study

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Law M, et al (2008). Broadly neutralizing antibodies protect against hepatitis C virus quasispecies challenge. Nat Med14:25–27.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Li, M. et al (2020). Hepatitis E virus infection and its associated adverse feto-maternal outcomes among pregnant women in Qinhuangdao, China. J. Matern. Fetal Neonatal. Med. 33, 3647–3651 (2020).

Li, P. et al (2020). The global epidemiology of hepatitis E virus infection: a systematic review and meta-analysis. Liver Int. 40, 1516–1528 (2020).

M. Amougou-Atsama, P. Jean Adrien Atangana, D. Noah Noah, P. Fewou Moundipa, P. Pineau, R. Njouom (2020). The role of hepatitis C virus genotypes and core mutations in hepatocellular carcinoma in Cameroon.

Ma, X. et al (2018). Chronic hepatitis B virus infection and preterm labor(birth) in pregnant women–an updated systematic review and meta-analysis. J. Med. Virol. 90, 93–100 (2018).

N.H.M. Hamadou, et al (2025). Traitement de l'Hépatite C de Génotype 1 par les antiviraux d'Action Directe au Cameroun : résultats préliminaires. HEALTH SCIENCES AND DISEASE.

P.A. Tagnouokam-Ngoupo, M.N. Ngoufack, S. Kenmoe, S.F. Lissock, M. Amougou-Atsama, R. Banai, et al (2019). Hepatitis C virus genotyping based on Core and NS5B regions in Cameroonian patients.

P.A. TagnouokamNgoupo, et al. (2019). Hepatitis C virus genotyping based on Core and NS5B regions in Cameroonian patients.

Quaranta M.G., et al (2024). Reduction of the risk of hepatocellular carcinoma over time using direct-acting antivirals: a propensity score analysis of a real-life cohort (PITER HCV) Viruses. 2024;16(5):682. doi: 10.3390/v16050682. 26 avr.

R. Njouom, et al (2003). Hepatitis C virus infection among pregnant women in Yaoundé, Cameroon: prevalence, viremia, and genotypes.

R. Njouom, et al (2012). Phylogeography, risk factors and genetic history of hepatitis C virus in Gabon, central Africa

R.D. Trimbitas, et al (2016). Molecular characterization of hepatitis C virus core region in Moroccan intravenous drug user

Ragusa R, Giorgianni G, Lupo L et al (2018). Healthcare-associated Clostridium difficile infection: role of correct hand hygiene in cross-infection control.

Revill, P. A. et al (2020). The evolution and clinical impact of hepatitis B virus genome diversity. Nat. Rev. Gastroenterol. Hepatol. 17, 618–634 (2020).

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Shimakawa, Y. et al (2019). Hepatitis B core-related antigen (HBcrAg): an alternative to HBV DNA to assess treatment eligibility in Africa. Clin. Infect. Dis. 70, 1442–1452 (2019).

Wang F, et al (2017). Post-vaccination serologic testing of infants born to hepatitis B surface antigen positive mothers in 4 provinces of China. Vaccine. 2017;35(33):4229–35.

Wang W, et al (2020). Dual-targeting nanoparticle vaccine elicits a therapeutic antibody response against chronic hepatitis B. Nat Nanotechnology. 2020; 15:406–16

Wong, et al (2023). Real-world treatment outcome with protease inhibitor direct-acting antiviral in advanced hepatitis C cirrhosis: a REAL-C study Hepatol Int. 2023/

World Health Organization (2016). global health sector strategy on viral hepatitis 2016–2021: towards ending viral hepatitis. Geneva: World Health Organization; 2016 (http://apps.who.int/iris/bitstream/10665/246177/1/WHO-HIV-2016.06-eng.pdf?ua=1, accessed 2 April 2020).

World Health Organization (2017). Elimination of mother to child transmission of HIV and syphilis. Geneva.

Zou H et al (2012) Virologic factors associated with failure to passive-active immunoprophylaxis in infants born to HBsAg-positive mothers. J Viral Hepat. 2012 Feb;19(2): e18-25

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Tatiana Jiengoué, Olivier Lieuga and Augustine Nji Asakizi.(2026). Assessing pregnant women's knowledge of HBV and HCV Routes of transmission at the East region of Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004004. www.iqresearchjournal.com

ANNEXES

Annex I — Table 1: Socio-demographic information and prevalence rates among pregnant women attending ANC

Variables					
Age	(19-28)	(29-38)	(39-48)	(49-58)	-
n	248	82	62	8	-
%	62%	20.5%	15.5%	2%	-
Marital status	Single	Married	Divorced	Widow	-
n	238	112	33	17	-
%	59.5%	28%	8.25%	4.25%	-
Education level	No formal education	Primary school	Secondary school	College	Postgraduate
n	130	122	84	49	15
%	32.5%	30.5%	21%	12.25%	3.75%
Occupation	Housewife	Business	Framing	Government employed	-
n	155	135	68	42	0
%	38.75%	33.75%	17%	19.5%	0%
Religion	Christian	Muslim	_	_	-
n	358	42	-	-	-
%	89.5%	10.5%	-	-	-
Gravidity and parity	First pregnancy	More than one pregnancy	-	-	-
n	95	305	-	-	-
%	23.75%	76.25%	-	-	-

Annex II — Table 2: HBV/HCV Routes of transmission among pregnant women at the BRH

HBV/HCV Routes of transmission	Pregnant women awareness	MTCT expression awareness	HBV and HCV Breastfeeding	Baby transmission after birth	Baby risk of transmission	
Yes						
n	266	202	184	28	136	
%	66.5%	50.5%	4%	70%	34%	
No						
n	134	198	124	15	244	
%	33.5%	49.5%	31%	37.5%	61%	

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Assessing Pregnant Women General knowledge and awareness on HBV and HCV amongst pregnant women at the Bertoua Regional Hospital in the East Region of Cameroon

Tatiana Jiengoué[a], Olivier Lieuga[a] and Augustine Nji Asakizi[a]

Affiliations

k. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Tatiana Jiengoué Tchakonang
Email: jiengouetatiana@kesmondsuniversity.org

Paper ID: IQRJ-V05I02-26005011

ABSTRACT

Pregnant women with chronic Hepatitis B and positive Hepatitis B virus E antigen (HBeAg) have a 90% likelihood of transmitting the Hepatitis B virus to their newborns. Vertical transmission is the leading cause of HCV infection in children. Hepatitis B and C lead to chronic disease and are the most common cause of liver cirrhosis, liver cancer, and viral hepatitis-related deaths. A major challenge to eliminating viral Hepatitis can be lack of knowledge and awareness on HBV and HCV. This cross-sectional study at Bertoua Regional Hospital of Cameroon from (June 2025-January 2026) aimed at Assessing pregnant women attending the Maternity Unit for the antenatal care (ANC) visits at the Bertoua Regional Hospital on their knowledge and awareness on HBV and HCV. The majority of them (62%) were aged within (19-28). Out of the 400 pregnant women, (91.5%) had knowledge and awareness about Hepatitis, (26.25%) were able to define hepatitis as a viral infection, (28%) were able to define liver as the primary organ affect by hepatitis; only (38.5%) of them regularly attend their ANC and just (8.25%) actually receive HBV and HCV counselling about its risks on pregnancy. (74.25%) of the pregnant women think HBV vaccination during pregnancy is risky and (53.25%) think it is necessary to be screened from HBV/HCV during pregnancy. However, we have limited data assessing pregnant women knowledge and awareness on HBV or HVC across both community and healthcare settings, hence the need of this research. The findings align with the World Health Organization as well as others organisms fighting for the prevention and elimination of viral Hepatitis such as the Centre of Disease Control and Prevention.

Keywords: *Pregnant women, General knowledge, Awarness, HBV and HCV, Cameroon*

1. INTRODUCTION

Hepatitis is an inflammation of the liver caused by various infections viruses and non-infectious agents (Hepatitis internet 2025). The most frequent cause of Hepatitis is due to viral infection (Mehta, Reddivari 2022;)Hepatitis B and C can lead to chronic diseases and are the most common cause of liver cirrhosis, liver cancer and viral hepatitis-related deaths (Hepatitis internet 2025;), perinatal transmission accounts for more than 50% of cases of hepatitis B worldwide and it is also the leading cause of HCV infection in children (Asafo-Agyei et al; 2023), there are about two billion people living with HBV worldwide and about 360 000 chronic carriers (Frambo et al; 2014). The prevalence of HBV in Cameroon varies from one region to the other (Ronalou; 2015). A systematic review by (Mahamat et al; 2015) estimated that the prevalence of HBV in Cameroon was low with overall 2.3% at all ages. In 2021, the study of (Fadel et al; 2021) showed that only 15.61% of pregnant women in the Djoungolo Health District have been screened for viral hepatitis.

Hepatitis C virus may be one of the common infections among pregnant women in Cameroon and worldwide, approximately 8% of pregnant women have HCV infection with the prevalence being as high as 4% in the USA (Bigna JJ et al; 2029). According to the US Centers Disease of control and prevention, the estimated 23 000-46 000 children in the USA live with HCV. In Cameroon, a 2025 study by (Ndembi et al; 2025) showed a 4% HCV prevalence in the 3rd trimester pregnancy. Still in Cameroon, in a study investigating HIV, HBV or HCV general knowledge of HBV among pregnant women in Cameroon is low to moderate with significant gap in understanding transmission and prevention despite high endemicity (often greater than 10%), studies show poor awareness of vaccination and vertical transmission risks with many relying on in correct knowledge (Andrea et al; 2022) investigated knowledge, risk factors and prevalence in the Loum Health District highlighting significant knowledge gaps in Hepatitis prevention. An effective vaccination program is vital for preventing HBV infection, MTCT can be prevented by routine screening of mothers and administering post-exposures prophylaxis to all infants born to infected mothers (Santsangi et al; 2016); like hepatitis B, there is no currently effective vaccine against Hepatitis C (Hepatitis internet 2025); a major challenge in eliminating viral hepatitis can be lack of awareness regarding the infection, here the study was undertaken with the aim to assess the knowledge and awareness regarding Hepatitis B and C infections among pregnant women attending the Maternity Unit for antenatal care visits at the Bertoua Regional Hospital, in other words, what is the level of knowledge and awareness of pregnant women on both viral Hepatitis B and C?.

2. RELATED WORKS

In a previous study of (Ngwanjoh et al; 2022), among pregnant women in the Loum Health District, the results showed that pregnant women had inadequate knowledge on HBV infection, the prevalence of HBsAg was (9%) with only one third of participants having adequate knowledge although hepatitis B is recognized to be one of the major health problem, pregnant women in the Loum Health District were less aware of its mode of transmission, consequences and prevention.

In another previous study of (Andreas et al; 2025) in the Buea Health District in Cameroon, the results showed that the hepatitis B

knowledge summary scores ranged from 0 to 12, Knowledge of HBV among pregnant women was poor. In another study of (Yankam et al; 2019), in the Limbe and Muyuka Health District of the South West region of Cameroon, pregnant women demonstrated poor knowledge and adopted poor practices regarding the modes of transmission and prevention of HBV infection.

In a study of (Singh et al; 2025), the results showed that among the interviewed pregnant women, only 47 (11.1%) were aware of either hepatitis B or both Hepatitis B and C, and among these, only (19.1%) had adequate knowledge.

3. MATERIALS & METHODS

Inclusion criteria were Pregnant women aged of 19 years and above (58) who freely consented, and came for antenatal care visit at the maternity unit of the BRH, and had not been vaccinated against HBVwithin the study period; as well, the study excluded pregnant women less than 19 years and non-pregnant women. Those of them who had receive HBV vaccine at the period of the study were also excluded. The prospective health facility-based study setting was chosen because of the required study population of pregnant women who register for regular antenatal care visits. Moreover, data collected at a specific point in time was deemed adequate to establish a diagnosis of HBV virus, hence, justifying the choice of a cross-sectional study design, this is because testing for Hepatitis B and C virus has been made a routine test for all the pregnant women on their antenatal care visit at the Bertoua Regional Hospital and actually were free of charge during the study. A total of 400 pregnant women were consecutively sampled (non-probabilistic) registered for their visit during the study period and all were approached with a request and signed the informed consent to take part in the study, all of them gave their consent to participate in the study and they were consecutively enrolled to the study.

Participants were assigned codes for anonymity purposes, we used for HBV screening Diaspot HBsAg, these are step Hepatitis B Surface Antigen (HBsAg) test strip package insert and for HCV, Diaspot HCV virus anti-body (HCV-Ab) test strips. Those are immune-chromatographic strips for qualitative detection of antibodies and antigens. Their sensitivity and specificity are above 99% and 98% respectively. Results were disclosed to participants with proper counselling; all infected pregnant women were counselled on the disease and referred for proper specialization care while the non-infected were counselled for HBV vaccination. Data were obtained using a well-structured questionnaire which was designed for the research and for laboratory analysis; questions elicited data to cover the objectives of the study, the questionnaire included seven sections, each focusing on a particular aspect to answer the research questions and gaps as well. The quantitative part of the questionnaire featured MCQ and Likert-scale questions allowing participant to rate their experiences, knowledge, feelings and attitudes. The questionnaires also included open-ended questions inviting participants to give other factors not mentioned. Frequencies (sums and percentages) were calculated for the socio-demographic factors and the different attitudes, feelings and practices towards HBV and HCV.

Tables displaying the frequency distribution for knowledge, attitude and practice towards HBV and HCV were entered into graph, each of the tables had frequencies for knowledge, maternal HBV/HCV preventive modes and practices, modes of transmission. Data were analyzed

using Excel 2016 frequencies and percentages were determined.

4. RESULTS & DISCUSSION

[See Annex — Table 1: Socio-demographic information amongst pregnant women attending ANC]

The results showed that the majority of pregnant women (62%) were aged within the framed age (19-28), most of them (59.5%) were single, (32.5%) of the pregnant women had no formal education, while (38.75%) of them were housewives, (89.5%) were Christian and more than half (76.25%) were multigravida that is they were pregnant more than once.

[See Annex — Table 2: Pregnant women general knowledge and awareness on HBV/HCV]

Out of the 400 pregnant women, (91.5%) had knowledge and awareness about Hepatitis, also, (24.75%) of the pregnant women said Hepatitis was asymptomatic and (33.75%) of them said Hepatitis was actually curable. only (38.5%) of the pregnant women regularly attend their ANC; while (8.25%) actually receive HBV and HCV counselling about Hepatitis risks on pregnancy.

[See Annex — Table 3: Hepatitis B and C Definition percentage]

Out of the 400 pregnant women, only (26.25%) were able to define hepatitis as a viral infection, (12.5%) defined it as a drug and (61?25%) defined it as a disease, (28%) were all the same able to define liver as the primary organ affect by hepatitis. Against (19%) who said Heart, (20%) lungs, (24.5%) stomach, (8.5%) actually didn't know.

[See Annex — Table 4: HBV Counselling and screening]

Out of the results, (13.25%) of the pregnant women were misinformed about HBV counselling at antenatal classes, against (43%) who said it doesn't exist and (5.25%) didn't know

Also, (53.25%) of them affirmed that Hepatitis is necessary during pregnancy against (23.75%) who affirmed it was not necessary and (23%) who had no such idea.

Out of our results, we found that (91.5%) of the interviewed pregnant women had awareness and were knowledgeable about Hepatitis, this study actually has a higher rate of awareness than in the study of (Junior et al; 2021) in which only (6.8%) of the respondents had knowledge of HBV. Again, in our results, (26.25%) of the pregnant women were able to define hepatitis as a viral infection, whereas, in the study of (Frambo et al; 2029), 80% of the participants did not know that Hepatitis B was a virus, (28%) of the pregnant women out of our results were able to define liver as a primary organ affected by Hepatitis, whereas only (15.9%) of the participants in the study of (Frambo et al; 2014) knew that infection with Hepatitis virus affects the liver as the primary organ.

In the study of (Junior et al; 2021) the results showed that during antenatal care, health education topics include HIV infections, malaria and nutrition for pregnant women, whereas from our observations and out of some of the pregnant women answers, HBV and HCV are not included at the maternity unit of the Bertoua Regional Hospital. , this could explain why midwives and nurses committed to ANC may exhibit limited knowledge of HBV and HCV, this goes in the same line with our results which showed that only (38.25%) of the pregnant women attended the ANC and just (8.25%) actually receive HBV and HCV counselling about risks on pregnancy (Junior et al; 2021), only one three pregnant women were reported having been tested, while in our study, (53.25%) of the pregnant women said screening from HBV and HCV during pregnancy is necessary.

5. CONCLUSION

Hepatitis B and HCV infections do really exist among pregnant women in the East region and these women exhibit very limited knowledge, that is their knowledge and awareness on Hepatitis are more superficial on these infections; awareness campaigns must be launched to inform women about the importance of screening and the availability of services at the Bertoua Regional Hospital. In addition, steps must be taken to make screenings service more accessible notably by reducing financial barriers, also the midwives have to organize antenatal classes for HBV and HCV counselling among pregnant women especially for those of them having a positive status of either one of them or both viruses, so as to promote prevention against vertical transmission.

REFERENCES

Alter et al (1976). Type B hepatitis: the infectivity of blood positive for e antigen and DNA polymerase after accidental needle stick exposure, N. Engl. J. Med, 295,909-913.

Anaedobe et al (2015). Prevalence, Socio-Demographic Features and Risk Factors of Hepatitis B Virus Infection among Pregnant Women in South-western Nigeria. The Pan African Medical Journal, 20, 406.

Ansari, et al (2023). Risk of early horizontal transmission of hepatitis B virus in children of uninfected mothers in sub-Saharan Africa: A systematic review and meta-analysis. Lancet Glob. Health 2023,

Cabot B, et al (1997). Structure of replicating hepatitis C virus (HCV) quasispecies in the liver may not be reflected by analysis of circulating HCV virions. J Virol71:1732–1734.

Delamare, H. et al (2024). Proportion of pregnant women with HBV infection eligible for antiviral prophylaxis to prevent vertical transmission: A systematic review and meta-analysis. JHEP Rep. 2024, 6, 101064.

Di Filippo Villa, et al (2023) D.; Navas, M.C. Vertical Transmission of Hepatitis B Virus-An Update. Microorganisms 2023, 11, 1140.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

El-Sayed, M. et al (2019). THU-137-DAA therapy in women of child bearing age: accidental conception during therapy and pregnancy outcome [abstract]. J. Hepatol. 70, e221 (2019).

El-Sayed, M. H.& Indolfi, (2020). G. Hepatitis C virus treatment in children: a challenge for hepatitis C virus elimination. Semin. Liver Dis. 40, 213–224 (2020).

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

F. Fiehn, et al (2024). Hepatitis C virus and hepatocellular carcinoma: carcinogenesis in the era of direct-acting antivirals.

Farooq H.Z. et al (2024). Risk factors for hepatocellular carcinoma associated with hepatitis C genotype 3 infection: a systematic review. World J. Gastrointest. Oncol. 2024;16(4):1596612. doi: 10.4251/wjgo. v16.i4.1596. 15 avr.

Fogel RS, Chappell CA. (2023). Hepatitis C virus in pregnancy: an opportunity to test and treat. Obstet Gynecol Clin North Am 50:363–373. doi: 10.1016/j.ogc.2023.02.008

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of HBV Neglected in Cameroon.MC Pregnancy and Childbirth, 13, 158.

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of HBV Neglected in Cameroon? BMC Pregnancy and Childbirth, 13, 158.

Freriksen, J. J. M. et al (2019). Review article: direct-acting antivirals for the treatment of HCV during pregnancy and lactation – implications for maternal dosing, foetal exposure, and safety for mother and child. Aliment. Pharmacol. Ther. 50, 738–750 (2019).

Funk, A. et al (2020). Efficacy and safety of antiviral prophylaxis during pregnancy to prevent mother-

to-child transmission of hepatitis B virus: a systematic review and meta-analysis. Lancet Infect Dis. https://doi.org/10.1016/S1473-3099(20)30586-7 (202

G.M. Kassa, et al (2024). Prevalence, trends, and distribution of hepatitis C virus among the general population in sub-Saharan Africa: a systematic review and meta-analysis.

Gross, M. S., Ruth, A. R. & Rasmussen, S. A. (2020). Respect women, promote health and reduce stigma: ethical arguments for universal hepatitis C screening in pregnancy. J. Med. Ethics 46, 674–677 (2020).

Hamadou N.H.M., al (2025). Traitement de l'Hépatite C de Génotype 1 par les antiviraux d'Action Directe au Cameroun : résultats préliminaires. HEALTH SCIENCES AND DISEASE [Internet] 2025.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Indolfi, G. et al (2019). Hepatitis B virus infection in children and adolescents. Lancet Gastroenterol. Hepatol. 4, 466–476 (2019).

Indolfi, G. et al (2019). Hepatitis C virus infection in children and adolescents. Lancet Gastroenterol. Hepatol. 4, 477–487 (2019).

Jiang, W. et al (2018). Sofosbuvir inhibits hepatitis A virus replication in vitro assessed by a cell-based fluorescent reporter system. Antivir. Res. 154, 51–57 (2018).

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Kaberg, M. & Weiland, O (2020). Hepatitis C elimination - macro-elimination. Liver Int. 40, 61–66 (2020).

Khan A., et al (2022). Core amino acid substitutions in HCV-3a isolates from Pakistan and opportunities for multi-epitopic vaccines. J. Biomol. Struct. Dyn. Mai. 2022;40(8):375368. doi: 10.1080/07391102.2020.1850353.

Knegendorf, L. et al (2018). Hepatitis E virus replication and interferon responses in human placental cells. Hepatol. Commun. 2, 173–187 (2018).

Kushner, T. & Terrault, N. A. (2019). Hepatitis C in pregnancy: a unique opportunity to improve the hepatitis C cascade of care. Hepatol. Commun. 3, 20–28 (2019).

L. Bhebhe, M. Anderson, S. Souda, W.T. Choga, E. Zumbi ka, Z.M. Shaver, et, al (2019). Molecular characterization of hepatitis C virus in liver disease patients in Botswana: a retrospective cross-sectional study

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Law M, et al (2008). Broadly neutralizing antibodies protect against hepatitis C virus quasispecies challenge. Nat Med14:25–27.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Li, M. et al (2020). Hepatitis E virus infection and its associated adverse feto-maternal outcomes among pregnant women in Qinhuangdao, China. J. Matern. Fetal Neonatal. Med. 33, 3647–3651 (2020).

Li, P. et al (2020). The global epidemiology of hepatitis E virus infection: a systematic review and meta-analysis. Liver Int. 40, 1516–1528 (2020).

M. Amougou-Atsama, P. Jean Adrien Atangana, D. Noah Noah, P. Fewou Moundipa, P. Pineau, R. Njouom (2020). The role of hepatitis C virus genotypes and core mutations in hepatocellular carcinoma in Cameroon.

Ma, X. et al (2018). Chronic hepatitis B virus infection and preterm labor(birth) in pregnant women–an updated systematic review and meta-analysis. J. Med. Virol. 90, 93–100 (2018).

N.H.M. Hamadou, et al (2025). Traitement de l'Hépatite C de Génotype 1 par les antiviraux d'Action Directe au Cameroun: résultats préliminaires. HEALTH SCIENCES AND DISEASE.

P.A. Tagnouokam-Ngoupo, M.N. Ngoufack, S. Kenmoe, S.F. Lissock, M. Amougou-Atsama, R. Banai, et al (2019). Hepatitis C virus genotyping based on Core and NS5B regions in Cameroonian patients.

P.A. TagnouokamNgoupo, et al. (2019). Hepatitis C virus genotyping based on Core and NS5B regions in Cameroonian patients.

Quaranta M.G., et al (2024). Reduction of the risk of hepatocellular carcinoma over time using direct-acting antivirals: a propensity score analysis of a real-life cohort (PITER HCV) Viruses. 2024;16(5):682. doi: 10.3390/v16050682. 26 avr.

R. Njouom, et al (2003). Hepatitis C virus infection among pregnant women in Yaoundé, Cameroon: prevalence, viremia, and genotypes.

R. Njouom, et al (2012). Phylogeography, risk factors and genetic history of hepatitis C virus in Gabon, central Africa

R.D. Trimbitas, et al (2016). Molecular characterization of hepatitis C virus core region in Moroccan intravenous drug user

Ragusa R, Giorgianni G, Lupo L et al (2018). Healthcare-associated Clostridium difficile infection: role of correct hand hygiene in cross-infection control.

Revill, P. A. et al (2020). The evolution and clinical impact of hepatitis B virus genome diversity. Nat. Rev. Gastroenterol. Hepatol. 17, 618–634 (2020).

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Shimakawa, Y. et al (2019). Hepatitis B core-related antigen (HBcrAg): an alternative to HBV DNA to assess treatment eligibility in Africa. Clin. Infect. Dis. 70, 1442–1452 (2019).

Wang F, et al (2017). Post-vaccination serologic testing of infants born to hepatitis B surface antigen positive mothers in 4 provinces of China. Vaccine. 2017;35(33):4229–35.

Wang W, et al (2020). Dual-targeting nanoparticle vaccine elicits a therapeutic antibody response against chronic hepatitis B. Nat Nanotechnology. 2020; 15:406–16

Wong, et al (2023). Real-world treatment outcome with protease inhibitor direct-acting antiviral in advanced hepatitis C cirrhosis: a REAL-C study Hepatol Int. 2023/

World Health Organization (2016). global health sector strategy on viral hepatitis 2016–2021: towards ending viral hepatitis. Geneva: World Health Organization; 2016 (http://apps.who.int/iris/bitstream/10665/246177/1/WHO-HIV-2016.06-eng.pdf?ua=1, accessed 2 April 2020).

World Health Organization (2017). Elimination of mother to child transmission of HIV and syphilis. Geneva.

Zou H et al (2012) Virologic factors associated with failure to passive-active immunoprophylaxis in infants born to HBsAg-positive mothers. J Viral Hepat. 2012 Feb;19(2): e18-25

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Tatiana Jiengoué, Olivier Lieuga and Augustine Nji Asakizi (2026). Assessing Pregnant Women General knowledge and awareness on HBV and HCV amongst pregnant women at the Bertoua Regional Hospital in the East Region of Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004005. www.iqresearchjournal.com

ANNEXES

Annex I — Table 1: Socio-demographic information amongst pregnant women attending ANC

Variables					
Age	(19-28)	(29-38)	(39-48)	(49-58)	-
n	248	82	62	8	-
%	62%	20.5%	15.5%	2%	-
Marital status	Single	Married	Divorced	Widow	-
n	238	112	33	17	
%	59.5%	28%	8.25%	4.25%	-
Education level	No formal education	Primary school	Secondary school	College	Postgraduate
n	130	122	84	49	15
%	32.5%	30.5%	21%	12.25%	3.75%
Occupation	Housewife	Business	Framing	Government employed	-
n	155	135	68	42	-
%	38.75%	33.75%	17%	19.5%	-
Religion	Christian	Muslim	_	_	-
n	358	42	-	-	-
%	89.5%	10.5%	-	-	-
Gravidity and parity	First pregnancy	More than one pregnancy	-	-	-
n	95	305	-	-	-
%	23.75%	76.25%also,	-	-	-

Annex II — Table 2 : Pregnant women general knowledge and awareness on HBV/HCV

Variables	Yes		No		I don't know	
	n	%	n	%	n	%
HBV/HCV knowledge and awareness	366	91.5%	34	8.5%	0	0%
Asymptomatic Hepatitis	99	24.75%	287	71.75%	0	0%
Curability of Hepatitis	12	33.75%	257	64.25%	14	3.5%
Pregnant women ANC attendance	154	38.75%	246	61.5%	0	0%
HBV/HCV Counselling	33	8.25%	121	30.25%	0	0%

Annex III — Table 3 : Hepatitis B and C Definition percentage

Variables	Drug		Disease		Viral infection			
Hepatitis B and C definition	n	%	n	%	n	%	-	-
	50	12.5%	245	61.25%	105	26.25%	-	-
							-	-
Hepatitis organs	Heartn	%	Lungsn	%	Stomachn	%	Livern	%	I don't known	%
	76	19%	80	20%	98	24.5%	112	28%	34	8.5%

Annex IV — Table 4: HBV Counselling and screening

Variables	Misinformation		Doesn’t exist		I don't know	
Counselling on HBV/HCV	n	%	n	%	n	%
	172	13.25%	53	43%	21	5.25%
Hepatitis Screening	Necessary		Unnecessary		I don't know	
	n	%	n	%	n	%
	213	53.25%	95	23.75%	92	23%

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Identifying Risk factors of viral Hepatitis B and C among pregnant women at the Bertoua Regional Hospital in the East Region of Cameroon

Tatiana Jiengoué[a], Olivier Lieuga[a] and Augustine Nji Asakizi[a]

Affiliations

l. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Tatiana Jiengoué Tchakonang
Email: jiengouetatiana@kesmondsuniversity.org

Paper ID: IQRJ-V05I02-26005012

ABSTRACT

Chronic viral Hepatitis B virus (HBV) and Hepatitis C virus (HCV) remain a significant global public health problem and a major cause of liver-related morbidity and mortality in Central Africa with high endemicity among pregnant women in Central Africa. Both Hepatitis B and C can cause acute and chronic infections and are leading cases of liver cirrhosis and hepatocellular carcinoma. During pregnancy, screening for hepatitis B and C is crucial because both viruses can be transmitted from mother-to-child, with HBV being more prevalent than HCV, and both infections posing risks for mother-to-child transmission. But, HBV transmission is largely preventable with post-birth imunoprophylaxis (vaccine + HIBG), while HCV transmission is less efficient but potentially treated with antivirals to prevent vertical spread. This cross-sectional study at Bertoua Regional Hospital Cameroon (June 2025-January 2026) aimed at identifying the risks factors of viral Hepatitis B and C among 400 pregnant women attending the maternity unit, the majority of them (62%) were aged within (19-28). Hospitalization (75.75%) was the main risk factor of viral Hepatitis B and C, followed by alcohol (72.25%) and blood transfusion (58.25%). However, data identifying risks factors across both community and healthcare settings remain limited, hence the need of this research The findings align with the World Health Organization as well as others organisms fighting for the elimination of viral Hepatitis such as the Centre of Disease Control and Prevention.

Keywords: *Risks factors, Viral Hepatitis B and C, Pregnant women, Cameroon*

1. INTRODUCTION

Blood-borne pathogens, Hepatitis B virus (HBV) and Hepatitis C virus (HCV) remain a significant global public Health challenges contributing substantially to the burden of infectious diseases worldwide. Globally, over 254 million and 50 million people are estimated to be chronically infected with HBV and HCV, respectively. According to the *(WHO 2024)*, Global Hepatitis report, Viral Hepatitis causes approximately 1.3 million deaths annually, ranking as the second leading infectious cause of death, comparable to tuberculosis.

Hepatitis B and C viruses are hepatotropic viruses responsible for the majority of viral hepatitis-related morbidity and mortality. Chronic infection can lead to liver cirrhosis, hepatocellular carcinoma (HCC), liver failure, and premature death: both viruses share similar transmission reuse of contaminated sharps, sexual contact, household exposures, and unsafe medical practices. Despite the availability of effective HBV vaccine, HCV remains non-vaccine preventable, and a substantial proportion of infected individuals remain undiagnosed and untreated worldwide. Hepatitis B virus causes a common Public health problem in Cameroon and across Sub-Saharan Africa *(Abongwa L.E. et al 2015).* In Uganda, antenatal education was recommended because most pregnant women showed excessively low knowledge and misconceptions about HBV *(Nyanka-Mutyoba et al 2018*).

Cameroon bears a significant burden based on the recent data, the Far North Region (specifically areas like Tokombéré and Mokolo) is a primary hotspot for high endemicity of HBV among pregnant women in Cameroon with prevalence rates exceeding 10% while HCV shows a prevalence rate of 1.8% to 7.3%, where endemic transmission persists. This cross-sectional study at Bertoua Regional Hospital among the 400 pregnant women as participants reported identified risk factors as follows: hospitalization, blood transfusion, dental and surgical history, alcohol, sexual transmitted diseases, liver family history, tattoo or piercing history, household contact and partners' history, conform with prior studies *(Eyong E.M. et al 2019).* Others significant risk factors were found to be involved in sexual activities below 19 years of age, history of multiple sex partners, and sexually transmitted infections *(Rabiu K.A et al 2010).* Likewise, expected risk factors were found to have no significant outcome in a study with HBsAg prevalence of 12.5% *(Ugbebor O. et al 2011)*. The findings align with the World Health Organization as well as others organisms fighting for the elimination of viral Hepatitis such as the Centre of Disease Control and Prevention. The main objective of this article is to identify the risks factors of viral Hepatitis B and C amongst pregnant women attending the Bertoua Regional Hospital, in other words, and knowing that Hepatitis is a communicable and infectious disease, what are the factors that predisposed pregnant women attending the Bertoua Regional Hospital to Hepatitis B and C?

2. RELATED WORKS

Although risk factors were not found to be significantly associated with HBsAg positivity among pregnant women in the Buea Health District in Cameroon, HBV in pregnant women has been associated with the risk of mother-to-child-transmission (MTCT), with the high prevalence (9.7%) of HBsAg, there was equally a higher chance of MTCT molecular Resistance Mechanisms in African Isolates. Hepatitis B virus causes a common Public Health problem in Cameroon and across Sub-Saharan *Africa*

(Abongwa L.E. et al 2015). In Uganda, antenatal education was recommended because most pregnant women showed excessively low knowledge and misconceptions about HBV, Studies have reported a significant relationship between knowledge on the transmission/prevention of HBV and the spread of the infection *(Nyanka-Mutyoba et al 2018).*

In 2017, a meta-analysis of *(Bigna JJ, Amougou et al 2017)* studied the seroprevalence of HCV in infections in Cameroon, the results showed that the prevalence was higher in the East region, in rural settings, and when using an enzyme immuno-assay technique for detecting anti-bodies HCV, there are still no data on HBV and HCV among pregnant women in that region, hence fostering the need to study the identification of the risk factors of HBV and HCV among pregnant women attending the antenatal care unit of the Bertoua Regional Hospital.

In the study of (Noubissié et al; 2023), The prevalence of hepatitis was 8.4%. Fifty-three percent (64) of the participants had adequate knowledge of Hepatitis B. Having had more than one sexual partner in the last six months and having visited a dentist in the past was significantly associated with Hepatitis B positive status.

3. MATERIALS & METHODS

Inclusion criteria were Pregnant women aged of 19 years and above (58) who freely consented, and came for antenatal care visit at the maternity unit of the BRH, and had not been vaccinated against HBVwithin the study period; as well, the study excluded pregnant women less than 19 years and non-pregnant women. Those of them who had receive HBV vaccine at the period of the study were also excluded. The prospective health facility-based study setting was chosen because of the required study population of pregnant women who register for regular antenatal care visits. Moreover, data collected at a specific point in time was deemed adequate to establish a diagnosis of HBV virus, hence, justifying the choice of a cross-sectional study design, this is because testing for Hepatitis B and C virus has been made a routine test for all the pregnant women on their antenatal care visit at the Bertoua Regional Hospital and actually were free of charge during the study. A total of 400 pregnant women were consecutively sampled (non-probabilistic) registered for their visit during the study period and all were approached with a request and signed the informed consent to take part in the study, all of them gave their consent to participate in the study and they were consecutively enrolled to the study.

Participants were assigned codes for anonymity purposes, we used for HBV screening Diaspot HBsAg, these are step Hepatitis B Surface Antigen (HBsAg) test strip package insert and for HCV, Diaspot HCV virus anti-body (HCV-Ab) test strips. Those are immune-chromatographic strips for qualitative detection of antibodies and antigens. Their sensitivity and specificity are above 99% and 98% respectively. Results were disclosed to participants with proper counselling; all infected pregnant women were counselled on the disease and referred for proper specialization care while the non-infected were counselled for HBV vaccination. Data were obtained using a well-structured questionnaire which was designed for the research and for laboratory analysis; questions elicited data to cover the objectives of the study, the questionnaire included seven sections, each focusing on a particular aspect to answer the research questions and gaps as well. The quantitative part of the questionnaire featured

MCQ and Likert-scale questions allowing participant to rate their experiences, knowledge, feelings and attitudes. The questionnaires also included open-ended questions inviting participants to give other factors not mentioned. Frequencies (sums and percentages) were calculated for the socio-demographic factors and the different attitudes, feelings and practices towards HBV and HCV.

Tables displaying the frequency distribution for knowledge, attitude and practice towards HBV and HCV were entered into graph, each of the tables had frequencies for knowledge, maternal HBV/HCV preventive modes and practices, modes of transmission. Data were analyzed using Excel 2016 frequencies and percentages were determined.

4. RESULTS & DISCUSSION

[See Annex — Table 1: Socio-demographic information amongst pregnant women attending ANC]

The results showed that the majority of pregnant women (62%) were aged within the framed age (19-28), most of them (59.5%) were single, (32.5%) of the pregnant women had no formal education, while (38.75%) of them were housewives, (89.5%) were Christian and more than half (76.25%) were multigravida that is they were pregnant more than once.

[See Annex — Table 2: Risks factors of HBV and HCV among pregnant women]

The findings showed that pregnant women having a hospitalization history are more likely to develop Hepatitis, that is hospitalization (75.75%) was the main risk factor of Viral Hepatitis among the pregnant women attending the Antenatal care visit at the Bertoua Regional Hospital, followed by alcohol history (72.25%) and blood transfusion history (58.25%), dental History (50.75%), multiple sexual partners (49.75%), surgical history (41.5%), sexually transmitted diseases (29.5%), liver family history (19.75%), Household contact (15.25%), and Piercing/tattoo history (11.25%).

The results showed that pregnant women having a hospitalization history are more likely to develop Hepatitis, that is hospitalization (75.75%) was the main risk factor of Hepatitis B and C among the pregnant women attending the Antenatal care visit at the Bertoua Regional Hospital, followed by alcohol history (72.25%) and blood transfusion history (58.25%). A similar study was conducted in the Loum Health District and we had the following results: (94.4%) of the pregnant women had a blood transfusion history, that is, the main risk factor among the pregnant women was blood transfusion history; followed by family household contact (91.3%) (Ngwanjoh et al 2022). In another study, 95% of pregnant women suffered from sexually transmitted infections like syphilis and it was considered a major risk (95%) for Hepatitis (Mawouma et al 2022).

Blood transfusions are important because they are a lifesaving medical treatment used to replace blood lost from injuries or surgery, or to treat conditions where the body can't produce enough healthy blood cells; while hospitalization is for serious or life-threatening medical conditions like heart attacks, scheduled procedures, and other illnesses that cannot be managed at home or in an outpatient setting; the majority of pregnant women has been blood transfused or hospitalized at least once in their lives: blood transfusion (58,25%), and hospitalization (75,25%), those viral Hepatitis risk factors actually predisposed those pregnant women attending the maternity unit of the Bertoua Regional Hospital to Hepatitis B and C.

Hence, fostering the urgent need of Hepatitis B and C prevention.

5. CONCLUSION

In conclusion, viral Hepatitis B and C among pregnant women is a real public health problem, women have to be sensitized and counselled. Viral Hepatitis screening routine has to be systematic as stated by the World Health Organization; unawareness is the main cause of high prevalence rate. As well, healthcare workers have to be conscious while operating on a daily basis, they must observe hygiene, as well as the using of safe objects for individual medical interventions so as to avoid contamination risks. Hepatitis has several risks factors as mentioned above. In our results, we found out that in the Bertoua Regional Hospital, Blood transfusion (75.75%) was the main risk factor of viral Hepatitis B and C; so efforts should be done so as to sensitize pregnant women at large and more especially the younger one about the negative consequences of Hepatitis B and C viruses on her pregnancy as well as her unborn baby in order to avoid or reduce the spread of the Hepatitis B and C viruses.

REFERENCES

Alter et al (1976). Type B hepatitis: the infectivity of blood positive for e antigen and DNA polymerase after accidental needle stick exposure, N. Engl. J. Med, 295,909-913.

Anaedobe et al (2015). Prevalence, Socio-Demographic Features and Risk Factors of Hepatitis B Virus Infection among Pregnant Women in South-western Nigeria. The Pan African Medical Journal, 20, 406.

Ansari, et al (2023). Risk of early horizontal transmission of hepatitis B virus in children of uninfected mothers in sub-Saharan Africa: A systematic review and meta-analysis. Lancet Glob. Health 2023,

Cabot B, et al (1997). Structure of replicating hepatitis C virus (HCV) quasispecies in the liver may not be reflected by analysis of circulating HCV virions. J Virol71:1732–1734.

Delamare, H. et al (2024). Proportion of pregnant women with HBV infection eligible for antiviral prophylaxis to prevent vertical transmission: A systematic review and meta-analysis. JHEP Rep. 2024, 6, 101064.

Di Filippo Villa, et al (2023) D.; Navas, M.C. Vertical Transmission of Hepatitis B Virus-An Update. Microorganisms 2023, 11, 1140.

Ducancelle et al. (2013) High Endemicity and Low Molecular Diversity of Hepatitis B Virus Infections in Pregnant Women in a Rural District of North Cameroon. PLoS ONE, 8, e80346.

European Centre for Disease Prevention and Control (ECDC) (2022) Introduction to the Annual Epidemiological Report. In: ECDC. Annual epidemiological report [Internet]. Stockholm: ECDC; 2022

European Paediatric Hepatitis C Virus Network. (2005). A significant sex-but not elective cesarean section-effect on mother-to-child transmission of hepatitis C virus infection. J Infect Dis192:1872–1879.

Fomulu, et al. (2013). Prevalence, Correlates and Pattern of Hepatitis B among Antenatal Clinic Attenders in Yaoundé-Cameroon: Is Perinatal Transmission of HBV Neglected in Cameroon? BMC Pregnancy and Childbirth, 13, 158.

Honegger JR, et al (2013). Loss of immune escape mutations during persistent HCV infection in pregnancy enhances replication of vertically transmitted viruses. Nat Med19:1529–1533.

Joshi, et al (2020). Presence of Precore (C)/C Promoter Mutants in Peripheral Blood Mononuclear Cells of Chronic Hepatitis B (CHB) Carriers During Pregnancy Does Not Correlate with Increased Risk of Liver Disease in 4 Years of Follow-Up. Dig. Dis. Sci. 2020, 65, 204–214.

Labarga P, et al (2007). Infant of 22 months of age with no anomalies born from a HCV- and HIV-infected mother under treatment with pegylated interferon, ribavirin and antiretroviral therapy during the first 16 weeks of pregnancy. Reprod Toxicol24:414–416.

Larouche A, et al (2012). Seronegative hepatitis C virus infection in a child infected via mother-to-child transmission. J Clin Microbiol50:2515–2519.

Law M, et al (2008). Broadly neutralizing antibodies protect against hepatitis C virus quasispecies challenge. Nat Med14:25–27.

Li C, et al (2020). Impact of maternal HIV-HBV coinfection on pregnancy outcomes in an underdeveloped rural area of southwest China. Sex Transm Infect. 2020 Nov;96(7):509-515.

Liu, J.F. et al (2021). Vertical transmission of hepatitis B virus: Propositions and future directions. Chin. Med. J. 2021, 134, 2825–2831.

Liu, Z et al (2022). Management Algorithm for Prevention of Mother-to-child Transmission of Hepatitis B Virus J. Clin. Transl. Hepatol. 2022, 10, 1004–1010.

Papaevangelou Vet al (1998). Increased transmission of vertical hepatitis C virus (HCV) infection to human immunodeficiency virus (HIV)-infected infants of

HIV- and HCV-coinfected women. J Infect Dis178:1047–1052.

Platt L et al (2020). Prevalence and burden of HBV co-infection among people living with HIV: A global systematic review and meta-analysis. Journal of Viral Hepatitis. 2020.

Schillie S, et al (2020). CDC Recommendations for hepatitis C screening among adults—United States, 2020.

Sede M, et al (2014). Inter and intra-host variability of hepatitis C virus genotype 1a hypervariable envelope coding domains followed for a 4–11 year of human immunodeficiency virus coinfection and highly active antiretroviral therapy. Virology471-473: 19–28.

Wang F, et al (2017). Post-vaccination serologic testing of infants born to hepatitis B surface antigen positive mothers in 4 provinces of China. Vaccine. 2017;35(33):4229–35.

Wang W, et al (2020). Dual-targeting nanoparticle vaccine elicits a therapeutic antibody response against chronic hepatitis B. Nat Nanotechnology. 2020; 15:406–16

Wong, et al (2023). Real-world treatment outcome with protease inhibitor direct-acting antiviral in advanced hepatitis C cirrhosis: a REAL-C study Hepatol Int. 2023/

World Health Organization (2016). global health sector strategy on viral hepatitis 2016–2021: towards ending viral hepatitis. Geneva: World Health Organization; 2016 (http://apps.who.int/iris/bitstream/10665/246177/1/WHO-HIV-2016.06-eng.pdf?ua=1, accessed 2 April 2020).

World Health Organization (2017). Elimination of mother to child transmission of HIV and syphilis. Geneva.

World Health Organization, (accessed on 11 May 2025). Hepatitis C Available online: https://www.who.int/news-room/fact-sheets/detail/hepatitis-c.

World Health Organization; (2015). Guidelines for the prevention, care and treatment of persons with chronic hepatitis B infection. Geneva: (http://apps.who.int/iris/bitstream/10665/154590/1/9789241549059_eng.pdf? ua=1&ua=1, accessed 20 March 2020).

Zou H et al (2012) Virologic factors associated with failure to passive-active immunoprophylaxis in infants born to HBsAg-positive mothers. J Viral Hepat. 2012 Feb;19(2): e18-25

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Tatiana Jiengoué, Olivier Lieuga and Augustine Nji Asakizi (2026). Identifying Risk factors of viral Hepatitis B and C among pregnant women at the Bertoua Regional Hospital in the East Region of Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004006. www.iqresearchjournal.com

ANNEXES

Annex I — Table 1: Socio-demographic information amongst pregnant women attending ANC

Variables					
Age	(19-28)	(29-38)	(39-48)	(49-58)	-
n	248	82	62	8	-
%	62%	20.5%	15.5%	2%	-
Marital status	Single	Married	Divorced	Widow	-
n	238	112	33	17	-
%	59.5%	28%	8.25%	4.25%	-
Education level	No formal education	Primary school	Secondary school	College	Postgraduate
n	130	122	84	49	15
%	32.5%	30.5%	21%	12.25%	3.75%
Occupation	Housewife	Business	Framing	Government employed	-
n	155	135	68	42	0
%	38.75%	33.75%	17%	19.5%	0%
Religion	Christian	Muslim	_	_	-
n	358	42	-	-	-
%	89.5%	10.5%	-	-	-
Gravidity and parity	First pregnancy	More than one pregnancy	-	-	-
n	95	305	-	-	-
%	23.75%	76.25%	-	-	-

Annex II — Table 2: Risks factors of HBV and HCV among pregnant women

Variables	Yes		No	
	n	%	n	%
Blood transfusion History	233	58.25%	99	24.75%
Hospitalization history	303	75.75%	97	24.25%
Surgical history	166	41.5%	234	58.5%
Dental history	203	50.75%	197	49.25%
Alcohol history	289	72.5%	111	27.75%
Liver family history	79	19.75%	321	80.25%
Piercing/tattoo history	45	11.25%	355	88.75%
Multiple sexual partners	199	49.75%	201	50.25%
Sexually transmitted diseases	118	29.5%	282	70.5%
Household contact	61	15.25%	339	84.75%

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Prevalence rate of Hepatitis B and Hepatitis C on the 271 patients tested positive with Hepatocellular Carcinoma among 396 patients living with Decompensated Cirrhosis recorded at the Bertoua Regional Hospital in Est Region Cameroon

Olivier Lieuga[a] ,Tatiana Jiengoué[a], and Augustine Nji Asakizi[a]

Affiliations

m. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Olivier Lieuga

Email: *olivierlieuga@kesmondsuniversity.org*

Paper ID: IQRJ-V05I02-26005013

ABSTRACT

Introduction: Hepatocellular Carcinoma (HCC) factors are dominated by chronic liver inflammation, with 80 – 90% of cases arising from cirrhosis. The primary drivers are chronic infections (Hepatitis B/C), alcohol abuse and metabolic dysfunction – associated steatosis liver disease (MASLD formerly NAFLD). Other key factors include smoking, obesity, type 2 diabetes and exposure to aflatoxins.

Methods: It is descriptive cross – sectional study done on 271 patients (187 men and 84 women) tested positive with HCC among 396 patients living with decompensated cirrhosis recorded at the Intensive Care Unit of Bertoua Regional Hospital from June to October 2025 after receiving ethical clearance and Research Authorization from Est Public Health Delegation – Cameroon. Convenience sampling was done. The analysis included major risk factors of HCC such HBV, HCV, Alcohol Toxic, Traditional Drugs Toxic, Aflatoxins and NAFLD.

Results: Out to 396 Cirrhosis patients, the prevalence rate of HCC was 68,43% and among 271 HCC patients, the prevalence rate of HBV for men is estimated at 41,69% and for women was 37,63%, followed by HCV (23,24% for men and 31,99% for women).

Conclusions: This study showed that the prevalence of Hepatitis B and C among patients living with hepatocellular carcinoma was high when compared to similar studies conducted in similar settings.

Keywords: *Hepatocellular Carcinoma,Liver Cirrhosis ,Hepatitis B,Hepatitis C*

1. INTRODUCTION

Global Distribution and Trends indicated in 2021 the high – risk regions like East Asia and Sub – Saharan African (SSA), often linked to endemic Hepatitis B (HBV), intermediate Regions as Southern Europe (eg: Italy, Spain.etc), and low – risks regions like North/South America, Northerm Europe, Oceania, though incidence is rising in the US, Madihi S et al (2020). In Sub-Saharan Africa (SSA), HCC is the second leading cause of cancer-related deaths for men and the fourth for women in 2020, with average age-standardized mortality rates of 8.2 and 4.2 per 100,000 persons/year, respectively, Daniel Mak and Anna Kramvis (2021).

In 2020, almost 906000 people were diagnosed with liver cancer globally, the most common form of which was Hepatocellular carcinoma. Hepatocellular Carcinoma is the third leading cause of cancer deaths worldwide, with a relative 5-year survival rate of approximately 18%, Sung H, Ferlay J and Siegel RL (2021). The similarity between incidence and mortality (830000 deaths per year) underlines the dismal prognosis associated with this disease. The diagnosis of hepatocellular carcinoma peaks in people aged between 60 and 70 years, and predominantly affects men, Siegel RL, Miller KD, Fuchs HE and Jemel A (2022). The incidence of hepatocellular carcinoma varies by geographical region and ethnicity, which is largely attributed to the prevalence of (and the age of exposure to) major risk factors, Jama Netw Open (2021).

[See Annex — Figure 1. Hepatocellular Carcinoma Global Distribution and Trends in the world]

2. RELATED WORKS

According to the Authors, Mohamed EI – Kassas and Mohamed El Badry (2022), the African population accounts for 12% of the Global population, most of them live in the Sub-Saharan Africa (SSA), where most inhabitants are blacks. On the other hand, North Africa’s Inhabitants are Mediterranean rather than African in terms of the race, customs and cultural background, that is why many of the distinctive features of cancers in Africa belong to SSA, Kew MC (2013).

Global Cancer Statistics estimated in 2020, the incidence and mortality of liver cancer cases in Africa represented 7,8% and 8,1% of the Global Cases, respectively. The liver cancer incidence and mortality statistics in different African areas for males and females were picked up. African areas were arranged descendingly according to the number of affected cases. In 2020, SSA had the fourth – highest number of diagnosed primary liver cancer (PLC) cases worldwide after South – Eastern Asia, South Central Asia and North America, with more than 38 000 new cases of PLC, 77% of them are HCCs, Mak D and Kramvis A (2021). Hepatocellular Carcinoma (HCC) is considered as a significant Health Burden in Africa, with high incidence rates, especially in West Africa (eg: Gambia, Guinea.), and major hotspots in Countries like Mozambique, Egypt and parts of Central Africa, driven by High Hepatitis B (HBV) and Hepatitis C (HBV prevalence, aflatoxin exposure, and lifestyle factors), Llovet JM et al (2021).

Africa accounts for nearly 8% of Global Liver Cancer cases, with rates varying significantly showing sub – Saharan Africa (SSA) having higher overall burdens particularly in males, compared to North Africa, though North Africa faces major HCV challenging (Egypt), London WT et al (2018). More than 80% of Global Hepatocellular Carcinoma (HCC) patients are estimated to occur in Sub-Saharan Africa and Eastern Asia. The most risk factor of HCC in SAA

is chronic hepatitis B virus (HBV) infection, with the incidence highest in West Africa. HBV is highly endemic ins SSA and is perpetuated by incomplete adherence to birth dose immunization, lack of longitudinal follow – up care, and impaired access to antiviral therapy, Siegel RL et al (2022).

HBV may directly cause HCC through somatic genetic alterations or indirectly through altered liver function and liver cirrhosis. Most patients with hepatocellular carcinoma have a background of chronic liver disease as a consequence of chronic infections with the hepatitis B virus (HBV) or hepatitis C virus (HCV), alcohol abuse or alcoholic steatohepatitis (ASH), and non-alcoholic fatty liver disease (NAFLD) or non-alcoholic steatohepatitis (NASH) Obesity, diabetes, and nicotine use are also associated with increased incidence of hepatocellular carcinoma, as are rare conditions such as haemochromatosis or hereditary tyrosinaemia type 1, The interventional liver cancer association (2022). Additionally, rates of hepatocellular carcinoma in patients with HIV have increased, specifically in those who are co-infected with HBV or HCV. Exposure to aflatoxin B1 is especially relevant in Asia, where it overlaps with HBV infection, Johnson PJ, Ahamaraj S, Berhane S, Bonnett L and Ma YT (2021).

The prevalence of risk factors for hepatocellular carcinoma varies globally, with a predominance of HBV in Asia, HCV in Japan, and NAFLD and NASH and alcohol in Europe and North America, Kim HS, Yu X, Kramer J et al (2022). In many cases, the risks of developing hepatocellular carcinoma are multifactorial and include demographic factors (age, sex, and ethnicity), severity and activity of underlying disease (fibrosis stage, inflammatory activity, and treatment), metabolic factors (diabetes and obesity), and lifestyle factors (alcohol intake and smoking), Azoulay D, Ramos E, Casellas – Robert M et al (2020). The collection and analysis of epidemiology HCC data will play a critical role in guiding future disease prevention strategies and optimizing patient management, Alan P et al (2010). Previous epidemiology studies have highlighted striking global variations in the incidence of HCC, which is particularly high in much of East Asia and Sub-Saharan Africa, and lower, nut on the increase, in North America and most of Europe.

[See Annex — Figure 2: Global Variation in HCC incidence rates. From Parkin DM, Bray F, Ferlay J et al (2002)]

3. MATERIALS & METHODS

It was a descriptive cross – sectional conducted among 271 patients living with Hepatocellular Carcinoma recorded at the Intensive Care Unit of Bertoua Regional Hospital in the East Region of Cameroon from june to October 2025 after receiving ethical clearance and authorization research letter for Est Region Public Health Delegation - Cameroon which occupies the Southeastern portion of the Republic of Cameroon. It's bordered to the East by the Central African Republic, to the South by Congo, to the North by the Adamawa Region and the West by the Centre and South Regions. The East Region has a type A wet equatorial climate, its capital is Bertoua which is located the Bertoua Regional Hospital, it has a population estimated at more than 3 millions of inhabitants. The Bertoua Regional Hospital has been selected as the reference hospital for the management of chronic liver disease by the Cameroon Public Health Ministry.

Convenience sampling was done, all the cases of elective HCC admitted during the study period

were included. Age > 20, men and women, Cameroonians or not but residents in Cameroon. The simple size was 271 patients calculated was calculated using the formula n = (Z2 x p x q) / e2 where n = minimum sample size, z= confidence interval, p= prevalence of decompensated cirrhosis, q= 1 – p and e= margin of error, 3%.

The data for this study were collected using structured questionnaires, entered and analysed in Microsoft Excel 2016, confidence interval was calculated along with frequency and percentages for binary data

To assess the prevalence rates of risk factors of HCC, we collected data on the results of various tests performed by the Hospital on the 271 patients who tested positive for HCC including Hepatitis B Elisa used to diagnose HBV, Hepatitis C Elisa for HCV, Fibrotest – Actitest for NAFLD or Fatty Liver, Fibroscan and Liver Biospy to diagnose the level of Liver Toxins.

4. RESULTS & DISCUSSION

The study included the analysis of major HCC risk factors such as Chronic Hepatitis B, Chronic Hepatitis C, Alcohol Toxic, Traditional Drugs Toxic, Aflatoxins and NAFLD among 271 patients tested positive with HCC and during our period of research (from June to October 2025), (187 males and 84 females). The prevalence of Patient characteristics tested positive with HCC by nationalities are summarized in table 1.

[See Annex — Table 1: Prevalence Rate of HCC Patient Characteristics]

per Nationalities at Bertoua Regional Hospital tested positive with Hepatocellular Carcinoma

Prevalence Rate of HCC Patient Characteristics

[See Annex — Table 2 : Tests Performed on 271 HCC patients to evaluate the prevalence rate]

of risk factors at the Bertoua Regional Hospital

Diagnostic of HCC Risk Factors

[See Annex — Table 3: Results on HCC Major Risk Factors at Bertoua Regional Hospital]

We have identified the following major risk factors to determine the HCC prevalence during our studies. After collecting and analyze data, it was found that among these 271 patients tested positive with HCC, Hepatitis B is the major risk factor, followed by Hepatitis C, Traditional Drugs Toxic, Alcohol Toxic and others. Hepatitis B represented 41,69% for the male against 37,63% for the female. Hepatitis C, 23,24% for the male and 31,99% for the female, Traditional Drugs Toxic, 21.03% for the male and 18,81% for the female. Results confirmed by the study done and published by Junaid Mahmond Alam et al,..(2023) “Prevalence of Hepatitis Infections in HCC Patients” including Patients both male and females, admitted in wards or visiting various clinics viz gastroenterology, oncology and -hepatology for diagnosis, treatment or recovery regiments were selected through their cas ehistory and lab-diagnosis results of hepatitis profile, AFP and histology. patients were grouped in the age range of>20 yr and <70yr; patients falling out side this sge range were excluded from the study. A breif history of Patients, with confirmed existence of HCC, was taken with clinical sympyoms and signs and initial diagnosis. Exclusively patients with CLD s, HCV and HBV or suspected of hepatitis infections with co-existence of HCC or vice versa, were selected and classified according to gender.

Another retrospective cross-sectional study was conducted at Hayatabad Medical Complex, Peshawar, Pakistan, using medical records of HBV-HCV co-infected patients from January 2023 to December 2024 on 348 HCC patients aged ≥18 years confirmed that the high prevalence rate of HBV and HCV as most risk factors of HCC.

5. CONCLUSION

According to our studies, the Cameroonian population was the most affected (61.99%), followed by Nigeriens (11.80%) and Central Africans (9.59%). Viral hepatitis B and C were the main causes of chronic hepatitis C (CHC) in these patients: 41.69% of men had HBV, compared to 37.63% of women, as confirmed by HBV ELISA tests. For HCV, we found 23.24% of men and 31.99% of women, confirmed by HCV ELISA tests. FibroScan was performed on only 1.84% of men and 5.16% of women, as the hospital lacked the technical facilities to perform FibroTest and Actitest tests, as well as liver biopsies. We recommended that the hospital establish partnerships with foreign medical analysis laboratories for toxicological examinations (aflatoxins, alcohol, chemical agents) in the liver, which are often the cause of liver cancer.

REFERENCES

Atkins D, Best D, Briss PA, Eccles M, Falck-Ytter Y, Flottorp S, et al. Grading quality of evidence and strength

of recommen dations. BMJ 2004;328:1490Okuda K. Hepatocellular carcinoma: recent progress.

HEPATOLOGY 1992;15:948-963

Alan P. Venook, M.D., Division of Medical Oncology, University of California, San Francisco, 1600 Divisadero Box 1770, San Francisco, California 94115, USA. Telephone: 415-353-9888; Fax: 415-353-9959; e-mail: venook@cc.ucsf.edu Received January 22, 2010; accepted for publication September 14, 2010. ©AlphaMed Press 1083-7159/2010/$30.00/0 doi: 10.1634/theoncologist.2010-S4-05

Anstee, Q. M., Reeves, H. L., Kotsiliti, E., Govaere, O. & Heikenwalder, M. From NASH to HCC: current concepts and future challenges. Nat. Rev. Gastroenterol. Hepatol. 16, 411–428 (2019).

Ayers, M. et al. IFN-γ-related mRNA profile predicts clinical response to PD-1 blockade. J. Clin. Invest. 127, 1–11 (2017).

Arizumi T, Ueshima K, Minami T, Kono M, Chishina H, Takita M, Kitai S, Inoue T, Yada N, Hagiwara S, et al. Effectiveness of Sorafenib in Patients with Transcatheter Arterial Chemoembolization (TACE) Refractory and Intermediate-Stage Hepatocellular Carcinoma. Liver Cancer. 2015;4:253–262. doi: 10.1159/000367743. [DOI] [PMC free article] [PubMed] [Google Scholar]

Akkoç, Y., & Gözüaçık, D. (2018). Autophagy and liver cancer. Turkish Journal of Gastroenterology, 29(3), 270–282. https://doi.org/10.5152/tjg.2018.150318

Ahmed F, Perz JF, Kwong S et al. National trends and disparities in the incidence of hepatocellular carcinoma, 1998–2003. Prev Chronic Dis 2008;5:A74

Bruix J, Castells A, Bosch J, Feu F, Fuster J, Garcia-Pagan JC, Visa J, et al. Surgical resection of hepatocellular carcinoma in cirrhotic pa tients: prognostic value of preoperative portal pressure. Gastroenterol ogy 1996;111:1018-1023.

Bruix J, Sherman M. Management of hepatocellular carcinoma: an update. HEPATOLOGY 2011;53:1020-1022. 3) Choo SP, Tan WL, Goh BK, Tai WM, Zhu AX. Comparison of hepatocellular carcinoma in Eastern versus Western popula tions. Cancer 2016;122:3430-3436.

Bruix J, Raoul JL, Sherman M, Mazzaferro V, Bolondi L, Craxi A, Galle PR, Santoro A, Beaugrand M, Sangiovanni A, et al. Efficacy and safety of sorafenib in patients with advanced hepatocellular carcinoma: subanalyses of a phase III trial. J Hepatol. 2012;57:821–829. doi: 10.1016/j.jhep.2012.06.014. [DOI] [PMC free article] [PubMed] [Google Scholar]

Block TM,MehtaAS,Fimmel CJet al. Molecular viral oncology of hepa tocellular carcinoma. Oncogene 2003;22:5093–5107.

Bauer S, Groh V, Wu J, Steinle A, Phillips JH, Lanier LL, et al. Activation of nk cells and T cells by Nkg2d, a receptor for stress-inducible mica. Science (1999) 285 (5428):727–9. doi: 10.1126/science.285.5428.727

Bianco A, Nanomaterials Muller S. Autophagy, and Lupus Disease. Chemmedchem. 2016;11:166–174. doi: 10.1002/cmdc.201500233. [DOI] [PubMed] [Google Scholar]

Boya P, Reggiori F, Codogno P. Emerging regulation and functions of autophagy. Nat Cell Biol. 2013;15:713–720. doi: 10.1038/ncb2788. [DOI] [PMC free article] [PubMed] [Google Scholar]

Bach M, Larance M, James DE, Ramm G. The serine/threonine kinase ULK1 is a target of multiple phosphorylation events. Biochem J. 2011;440:283–291. doi: 10.1042/BJ20101894. [DOI] [PubMed] [Google Scholar]

BriukhovetskaD, Dörr J, Endres S, Libby P, DinarelloCA, Kobold S. Interleukins in cancer: from biology to therapy. Nat Rev Cancer (2021) 21(8):481–99. doi: 10.1038/s41568-021-00363-z

Bernier, J. (2016). Immuno-oncology: Allying forces of radio- and immuno-therapy to enhance cancer cell killing. Critical Reviews in Oncology/Hematology, 108, 97–108. https:// doi.org/10.1016/j.critrevonc.2016.11.001

Bruix, J.; Sherman, M. Management of hepatocellular carcinoma: An update. Hepatology 2011, 53, 1020–1022. [Google Scholar] [CrossRef] [PubMed]

Bruix, J. et al. Adjuvant sorafenib for hepatocellular carcinoma after resection or ablation (STORM): a phase 3, randomised, double-blind, placebo-controlled trial. Lancet Oncol. 16, 1344–1354 (2015).

Burtness, B.; Harrington, K.J.; Greil, R.; Soulières, D.; Tahara, M.; de Castro, G.; Psyrri, A.; Basté, N.; Neupane, P.; Bratland, Å.; et al. Pembrolizumab alone or with chemotherapy versus cetuximab with chemotherapy for recurrent or metastatic squamous cell carcinoma of the head and neck (KEYNOTE-048): A randomised, open-label, phase 3 study. Lancet 2019, 394, 1915–1928. [CrossRef]

Colombo M, de Franchis R, Del Ninno E, Sangiovanni-A, De-Fazio-C, Tommasini-M. Donato-M-F, et al. Hepatocellular carcinoma in Italian patients with cirrhosis. N Engl J Med 1991;325:675-680.

Chaiteerakij R, Addissie BD, Roberts LR. Update on biomarkers of hepatocellular carcinoma. Clin Gastroenterol Hepatol 2015;13: 237-245.

Cappuyns, S., Virginia, C., Yarchoan, M., Finn, R. S. & Llovet, J. M. Critical appraisal of guideline recommendations on systemic therapies for advanced hepatocellular carcinoma. JAMA Oncol. https://doi.org/10.1001/jamaoncol.2023.2677 (2023).

CalvetX, Bruix J, Gine´sP,Bru´C, Sole M, VilanaR, Rodes J. Prognostic factors of hepatocellular carcinoma in the West. A multivariate analysis in 206 patients. HEPATOLOGY 1990;12:753-760.

Cole P, Morrison AS. Basic issues in population screening for cancer. J Natl Cancer Inst 1980;64:1263-1272.

Craig JR, Peters RL, Edmondson HA, et al. Fibrolamellar carcinoma of the liver: a tumour of adolescents and young adults with distinctive clinicopathologic features. Cancer 1980;46:372-9.

Cadwell K, Liu JY, Brown SL, Miyoshi H, Loh J, Lennerz JK, Kishi C, Kc W, Carrero JA, Hunt S, Stone CD, Brunt EM, Xavier RJ, et al. A key role for autophagy and the autophagy gene Atg16l1 in mouse and human intestinal Paneth cells. Nature. 2008;456:259–U262. doi: 10.1038/nature07416. [DOI] [PMC free article] [PubMed] [Google Scholar]

Chen, L.-T. et al. Long-term results of a randomized, observation-controlled, phase III trial of adjuvant interferon Alfa-2b in hepatocellular carcinoma after curative resection. Ann. Surg. 255, 8–17 (2012).

Cheng AL, Kang YK, Chen Z, Tsao CJ, Qin S, Kim JS, Luo R, Feng J, Ye S, Yang TS, et al. Efficacy and safety of sorafenib in patients in the Asia-Pacific region with advanced hepatocellular carcinoma: a phase III randomised, double-blind, placebo-controlled trial. Lancet Oncol. 2009;10:25–34. doi: 10.1016/S1470-2045(08)70285-7. [DOI] [PubMed] [Google Scholar]

Chu, J. S. et al. Expression and prognostic value of VEGFR-2, PDGFR-β, and c-Met in advanced hepatocellular carcinoma. J. Exp. Clin. Cancer Res. 32, 16 (2013)

Cheng AL, Kang YK, Chen Z, Tsao CJ, Qin S, Kim JS, Luo R, Feng J, Ye S, Yang TS, et al. Efficacy and safety of sorafenib in patients in the Asia-Pacific region with advanced hepatocellular carcinoma: a phase III randomised, double-blind, placebo-controlled trial. Lancet Oncol. 2009;10:25–34. doi: 10.1016/S1470-2045(08)70285-7. [DOI] [PubMed] [Google Scholar]

Chang YS, Adnane J, Trail PA, Levy J, Henderson A, Xue D, Bortolon E, Ichetovkin M, Chen C, McNabola A, et al. Sorafenib (BAY 43-9006) inhibits tumor growth and vascularization and induces tumor apoptosis and hypoxia in RCC xenograft models. Cancer Chemother Pharmacol. 2007;59:561–574. doi: 10.1007/s00280-006-0393-4. [DOI] [PubMed] [Google Scholar]

Chen R, Hou B, Zhou Y, et al. Recurrence after percutaneous radiofrequency ablation of hepatocellular carcinoma: analysis of the pattern and risk factors. Front Oncol. 2023;13:1018715. doi:10.3389/fonc.2023.1018715

De Mitri MS, Cassini R, Morsica G et al. Virological analysis, genotypes and mutational patterns of the HBV precore/core gene in HBV/HCV related hepatocellular carcinoma. J Viral Hepat 2006;13:574–581

Dakurah OB, Tamandjou CRT, Zunza M, Preiser W, Maponga TG. Viral hepatitis associated hepatocellular carcinoma on the African continent, the past, present, and future: a systematic review. BMC Cancer. (2021) 21:1 13. doi: 10.1186/s12885-021-08426-y

Deng H, Kan A, Lyu N, Mu L, Han Y, Liu L, et al. Dual vascular endothelial growth factor receptor and fibroblast growth factor receptor inhibition elicits antitumor immunity and enhances programmed cell death-1 checkpoint blockade in hepatocellular carcinoma. Liver Cancer (2020) 9(3):338–57. doi: 10.1159/000505695

Ding, W.X.; Ni, H.M.; Gao, W.; Yoshimori, T.; Stolz, D.B.; Ron, D.; Yin, X.M. Linking of autophagy to ubiquitin-proteasome system is important for the regulation of endoplasmic reticulum stress and cell viability. Am. J. Pathol. 2007, 171, 513–524. [Google Scholar] [CrossRef] [PubMed]

Decaens, T.; Luciani, A.; Itti, E.; Hulin, A.; Roudot-Thoraval, F.; Laurent, A.; Zafrani, E.S.; Mallat, A.; Duvoux, C. Phase II study of sirolimus in treatment-naive patients with advanced hepatocellular carcinoma. Dig. Liver Dis. 2012, 44, 610–616. [Google Scholar] [CrossRef] [PubMed]

El-Serag HB. Hepatocellular carcinoma. N Engl J Med 2011; 365:1118-1127.

El-Serag, H. B., & Rudolph, K. L. (2007). Hepatocellular carcinoma: epidemiology and molecular carcinogenesis. Gastroenterology, 132(7), 2557–2576. https://doi.org/10.1053/j.gastro.2007.04.061

Eskes R, Antonsson B, Osen-Sand A, Montessuit S, Richter C, Sadoul R, et al. Bax-induced cytochrome C release from mitochondria is independent of the permeability transition pore but highly dependent on Mg2+ ions. J Cell Biol. (1998) 143:217–24. doi: 10.1083/jcb.143.1.217

Ebrahimi, N. et al. Receptor tyrosine kinase inhibitors in cancer. Cell. Mol. Life Sci. 80, 104 (2023).

Ezaki, J.; Matsumoto, N.; Takeda-Ezaki, M.; Komatsu, M.; Takahashi, K.; Hiraoka, Y.; Taka, H.; Fujimura, T.; Takehana, K.; Yoshida, M.; et al. Liver autophagy contributes to the maintenance of blood glucose and amino acid levels. Autophagy 2011, 7, 727–736. [Google Scholar] [CrossRef] [PubMed]

Egan, D.F.; Chun, M.G.; Vamos, M.; Zou, H.; Rong, J.; Miller, C.J.; Lou, H.J.; Raveendra-Panickar, D.; Yang, C.C.; Sheffler, D.J.; et al. Small Molecule Inhibition of the Autophagy Kinase ULK1 and Identification of ULK1 Substrates. Mol. Cell 2015, 59, 285–297. [Google Scholar] [PubMed]

Ho, W. J. et al. Neoadjuvant cabozantinib and nivolumab converts locally advanced HCC into resectable disease with enhanced antitumor immunity. Nat. Cancer 2, 891–903 (2021).

Gauthier, A.; Ho, M. Role of sorafenib in the treatment of advanced hepatocellular carcinoma: An update. Hepatol. Res. 2013, 43, 147–154. [Google Scholar] [CrossRef] [PubMed]

Galluzzi L, Chan TA, Kroemer G, Wolchok JD, López-Soto A. The hallmarks of successful anticancer immunotherapy. Sci Trans Med (2018) 10(459):eaat7807. doi: 10.1126/scitranslmed.aat7807

Li, N., Spetz, M. R., & Ho, M. (2020). The role of glypicans in cancer progression and therapy. Journal of Histochemistry & Cytochemistry, 68(12), 841-862.

Llovet, J. M., Kelley, R. K., Villanueva, A., Singal, A. G., Pikarsky, E., Roayaie, S., Lencioni, R., Koike, K., Zucman-Rossi, J., & Finn, R. S. (2021). Hepatocellular carcinoma. Nature Reviews. Disease Primers, 7(1), 6.

Llovet, J. M. et al. Hepatocellular carcinoma. Nat. Rev. Dis. Prim. 2, 16018 (2016).

Llovet, J. M. et al. Nonalcoholic steatohepatitis-related hepatocellular carcinoma: pathogenesis and treatment. Nat. Rev. Gastroenterol. Hepatol. 20, 487–503 (2023).

Llovet JM, Ricci S, Mazzaferro V, Hilgard P, Gane E, Blanc JF, de Oliveira AC, Santoro A, Raoul JL, Forner A, et al. Sorafenib in advanced hepatocellular carcinoma. N Engl J Med. 2008;359:378–390. doi: 10.1056/NEJMoa0708857. [DOI] [PubMed] [Google Scholar]

Lehmann U, Wingen LU, Brakensiek K, Wedemeyer H, Becker T, Heim A, et al. Epigenetic defects of hepatocellular carcinoma are already found in non-neoplastic liver cells from patients with hereditary haemochromatosis. Hum Mol Genet. 2007;16:1335 42.

Lencioni R, Llovet JM, Han G, Tak WY, Yang J, Guglielmi A, Paik SW, Reig M, Kim DY, Chau GY, et al. Sorafenib or placebo plus TACE with doxorubicin-eluting beads for intermediate stage HCC: The SPACE trial. J Hepatol. 2016;64:1090–1098. doi: 10.1016/j.jhep.2016.01.012. [DOI] [PubMed] [Google Scholar]

Liu, Z. L., Chen, H. H., Zheng, L. L., Sun, L. P. & Shi, L. Angiogenic signaling pathways and anti-angiogenic therapy for cancer. Signal Transduct. Target. Ther. 8, 198 (2023).

Ikeda K, Saitoh S, Koida I, Arase Y, Tsubota A, Chayama K, Kumada H, et al. A multivariate analysis of risk factors for hepatocellular carci nogenesis: a prospective observation of 795 patients with viral and alco holic cirrhosis. HEPATOLOGY 1993;18:47-53.

Itakura E, Mizushima N. Characterization of autophagosome formation site by a hierarchical analysis of mammalian Atg proteins. Autophagy. 2010;6:764–776. doi: 10.4161/auto.6.6.12709. [DOI] [PMC free article] [PubMed] [Google Scholar]

Ishizawa, T. et al. Neither multiple tumors nor portal hypertension are surgical contraindications for hepatocellular carcinoma. Gastroenterology 134, 1908–1916 (2008).

Tsukuma H, Hiyama T, Tanaka S, Nakao M, Yabuuchi T, Kitamura T, Nakanishi K, et al. Risk factors for hepatocellular carcinoma among patients with chronic liver disease. N Engl J Med 1993;328:797-801.

Toso, C.; Merani, S.; Bigam, D.L.; Shapiro, A.M.; Kneteman, N.M. Sirolimus-based immunosuppression is associated with increased survival after liver transplantation for hepatocellular carcinoma. Hepatology 2010, 51, 1237–1243. [Google Scholar] [CrossRef] [PubMed

Petrick JL, Braunlin M, Laversanne M, Valery PC, Bray F, McGlynn KA. International trends in liver cancer incidence, overall and by histologic subtype, 1978-2007. Int J Cancer 2016; 139:1534-1545.

Petrick JL, Kelly SP, Altekruse SF, McGlynn KA, Rosenberg PS. Future of hepatocellular carcinoma incidence in the United States forecast through 2030. J Clin Oncol 2016;34:1787-1794.

ParkinDM,Pisani P, Ferlay J. Estimates of the worldwide incidence of 25 major cancers in 1990. Int J Cancer 1999;80:827–841.

Pinyol, R., Sia, D. & Llovet, J. M. Immune exclusion-Wnt/CTNNB1 class predicts resistance to immunotherapies in HCC. Clin. Cancer Res. 25, 2021–2023 (2019).

Pan YX, Fu YZ, Hu DD, et al. Stereotactic body radiotherapy vs. radiofrequency ablation in the treatment of hepatocellular carcinoma: a meta-analysis. Front Oncol. 2020;10:1639. doi:10.3389/fonc.2020.01639

Klionsky DJ. Autophagy: from phenomenology to molecular understanding in less than a decade. Nat

Rev Mol Cell Bio. 2007;8:931–937. doi: 10.1038/nrm2245. [DOI] [PubMed] [Google Scholar]

Kim N, Park PJ, Jung MK, Song G-W, Jung D-H, Lee S-G, et al. Differential function of natural killer cells in the liver graft perfusate of Korean population. In: Transplantation proceedings. Elsevier.

Kaseb, A. O. et al. Perioperative nivolumab monotherapy versus nivolumab plus ipilimumab in resectable hepatocellular carcinoma: a randomised, open-label, phase 2 trial. Lancet Gastroenterol. Hepatol. 7, 208–218 (2022).

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Olivier Lieuga ,Tatiana Jiengoué, and Augustine Nji Asakizi. (2026). Prevalence rate of Hepatitis B and Hepatitis C on the 271 patients tested positive with Hepatocellular Carcinoma among 396 patients living with Decompensated Cirrhosis recorded at the Bertoua Regional Hospital in Est Region Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004013. www.iqresearchjournal.com

ANNEXES

Annex I — Table 1: Prevalence Rate of HCC Patient Characteristics

Prevalence Rate of HCC Patient Characteristics	Cameroonians		Chadians		Nigerians		Centrafric Republic		Others Nationalities	
	n	%	n	%	n	%	n	%	n	%
HCC Patients Tested Positive per Nationalities	168	61,99	22	8,11	32	11,80	26	9,59	23	8,48
Gender										
Male	115	61,49	15	8,02	23	9,62	18	9,62	16	8,55
Female	53	63,09	7	8,33	9	8,52	8	8,52	7	8,33
Age (Years Old)										
20 – 35	98	58,33	7	31,81	15	46,87	9	34,61	12	52,17
35 - 55	56	33,33	11	50	13	40,62	12	46,15	6	26,08
55 – Over	14	8,33	4	18,18	4	12,5	5	19,23	5	21,73
Marital Status										
Single	78	46,42	6	27,27	14	43,75	11	42,30	16	69,56
Married	27	16,07	9	40,90	9	28,12	6	23,07	3	13,04
Divorced	63	37,5	7	31,81	9	28,12	9	34,61	4	17,39
Education Levels										
Primary	69	41,07	13	59,09	15	46,87	9	34,61	7	30,43
College	48	28,57	6	27,27	9	28,12	12	46,15	12	52,17
University	51	30,35	3	13,63	8	25	5	19,23	4	17,39
Employment										
Unemployed	72	42,85	6	27,27	8	25	10	38,46	7	30,43

Student	15	8,92	5	22,72	7	21,87	6	23,07	5	21,73
Employed	13	7,73	2	9,09	4	12,5	5	19,23	3	13,04
Self – Employed	47	27,97	3	13,63	3	9,37	3	11,53	7	30,43
Retired	21	12,5	4	18,18	10	31,25	2	7,69	1	4,34

Annex II — Table 2 : Tests Performed on 271 HCC patients to evaluate the prevalence rate

Diagnostic of HCC Risk Factors	Elisa Hepatitis B Test		Elisa Hepatitis C Test		Fibrotest – Actitest Score		Fibroscan		Liver Biopsy	
	n	%	n	%	n	%	n	%	n	%
Gender										
Male	113	41,69	63	23,24	0	0	5	1,84	0	0
Female	102	37,63	84	31,99	0	0	14	5,16	0	0

Annex III — Table 3: Results on HCC Major Risk Factors at Bertoua Regional Hospital

Prevalence Rate of HCC Major Risk Factors at BRH	Hepatitis B		Hepatitis C		Alcohol Toxic		Traditional Drugs Toxic		Aflatoxins		NAFLD	
	n	%	n	%	n	%	n	%	n	%	n	%
Gender												
Men	113	41,69	63	23,24	0	0	0	0	0	0	5	1,84
Women	102	37,63	84	31,99	0	0	0	0	0	0	14	5,16

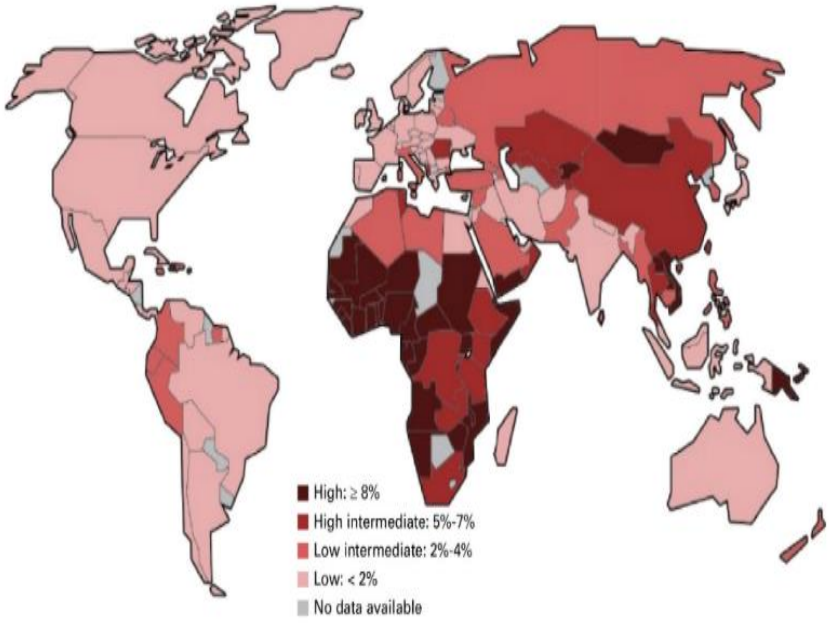


Figure 1. *Hepatocellular Carcinoma Global Distribution and Trends in the world*

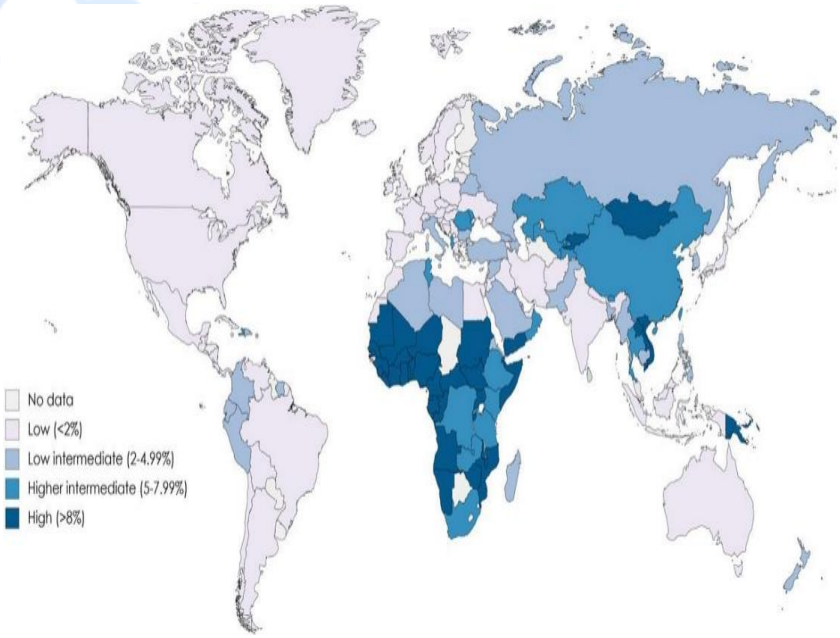


Figure 2. *Global Variation in HCC incidence rates. From Parkin DM, Bray F, Ferlay J et al (2002)]*

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Prevalence rate of Hepatocellular Carcinoma on 68 Cameroonians among 100 patients with Liver Cirrhosis during treatment at The NGaoundere Protestant Hospital in Adamawa Region Cameroon

Olivier Lieuga[a] ,Tatiana Jiengoué[a,*], and Augustine Nji Asakizi[a]

Affiliations

n. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Olivier Lieuga

Email: olivierlieuga@kesmondsuniversity.org

Paper ID: IQRJ-V05I02-26005014

ABSTRACT

Introduction: Liver Cirrhosis, the primary cause of liver failure and hepatocellular carcinoma, affects millions of adults and children worldwide. Cirrhosis affects approximately 2.2 million adults in the World and is associated with mortality rates of 21.9 per 100 000 people. Cirrhosis is defined as the fibrotic replacement of liver tissue that can result from any chronic liver disease.

Methods: It is descriptive cross – sectional study done on 68 Cameroonians patients (53 men and 15 women) among 100 patients living with Liver Cirrhosis at the NGaoundere Protestant Hospital in Adamawa Region Cameroon from June 2023 to June 2024 after receiving Ethical Clearance and Research Authorization from The Health Work of the Evangelical Lutheran Church of Cameroon (HWELCC) Chairman. Convenience sampling was done. The analysis included management of risk factors, clinical manifestations, diagnostic methods, treatment and complications of liver cirrhosis.

Results: Out to 100 Cirrhotic patients, the prevalence of Cameroonians were 68% (77,94% of men and 22,05% of women) including 88,67% with HCC developed for men and 80,00% with HCC developed for women.

Conclusions: This study showed that the prevalence of Hepatocellular Carcinoma to the Cameroonians cirrhotic patients were too high due to lack of the appropriate methods for the management of Cirrhotic Patients

Keywords: *Hepatocellular Carcinoma, Liver Cirrhosis ,Hepatitis B, Hepatitis C*

1. INTRODUCTION

Most prevalent cases of cirrhosis are caused by alcohol use disorder (approximately 45% of all cirrhosis cases), hepatitis C (41%), and non-alcoholic fatty liver disease (26%), with many patients having overlapping causes, Durand F et al (2003). However, hepatitis C is now curable with direct acting antivirals and most newly diagnosed cirrhosis is due to non-alcoholic fatty liver disease (NAFLD) (accounting for 61.8% of incident cases) and alcohol use disorder, Franchis de, et al (2010).

Liver cirrhosis simply means scarring of the liver. Scarring of the liver is a concerns as it causes healthy liver cells to die and be replaced by stiff scar tissue. This process happens slowly and is often irreversible and may lead to the whole hardening and becoming scarred and shrunken, Friedman SL (2008). When something attacks and damages the liver, liver cells are killed and scar tissue is formed. This scarring is called fibrosis, and it happens little by little over many years. When the whole liver is scarred, it shrinks and gets hard. This is called cirrhosis, and usually this damage cannot be undone, Garcia – Tsao G, et al (2009).

In the other hand, any illness that affects the liver over a long period of time may lead to fibrosis and, eventually, cirrhosis. Heavy drinking and viruses (like hepatitis C or hepatitis B) are common causes of cirrhosis, Jiao J, et al (2009). Cirrhosis may also be caused by a build-up of fat in the liver of people who are overweight or have diabetes, called non-alcoholic fatty liver disease. There are others, less common, causes of cirrhosis, Kanwal F, et al (2010). The scarring process is a slow process and usually takes between 20 and 40 years for cirrhosis to develop. The damage can begin slowly, often with no outward symptoms or signs to indicate the gradual progression of scarring to the cells and resulting loss of liver functions, Franchis de, et al (2010). Liver cirrhosis itself also has several stages. In the early stages there are still enough healthy liver cells to perform the functions of the liver. This is called Compensated Cirrhosis. However, if the scarring is allowed to continue, more of the cells are lost and the liver is no longer able to fulfil its important functions, this is called Decompensated Cirrhosis, Durand F et al (2003). Eventually the liver can become so scarred and shrunken that without a liver transplant the result is death. Cirrhosis of the liver also increases the risk of a cancer developing in the liver.

2. RELATED WORKS

A liver with cirrhosis becomes very hardened and lumpy with scar tissue. This makes it very hard for blood to flow through the liver. This causes a build-up or pressure on one side of the liver causing the pressure to increase inside the veins that are attached to the liver. Imagine a hose full of water that has been kinked at one end. This causes the water to build up and flow back toward the tap. One of the veins affected is called the portal vein, which is responsible for bringing blood to the liver, *Carbonell N, Pauwels A, Sergaty L, Fourdan O, Levy VG and Poupon R (2004).*

The cirrhotic nodules may be small (micronodular), large (macronodular), or there may be a mixture of the two in micronodular cirrhosis, the septa are thick and there are regular uniformly small regenerating nodules in every hepatic lobule. In macronodular cirrhosis the nodules are of varying size. This pathological distinction is descriptive, and does not imply any diagnosis or prognosis, *Arvaniti. V et al (2010)*

When the pressure in this vein is increased it is called Portal Hypertension. This then causes a backflow of blood (like the kinked up hose) up into the spleen. The size of the spleen then increases causing it to destroy platelets (a type of blood cell) which affect how well your blood clots, *Berzigott A et al (2011).* Besides causing problems with blood flow, the scar tissue also limits how well the liver can do its job. The liver's role is to filter and remove toxins, produce bile to break down nutrients, control blood clotting and produce important proteins. When the liver cannot carry out these vital roles it causes many of the toxins to escape into the body causing confusion and trouble concentrating and this is called Hepatic Encephalopathy (HE), *Tarao K, Nozaki A, Ikeda T et al (2019).*

There are two different stages of cirrhosis; compensated and decompensated. Compensated cirrhosis often has little or no symptoms. This is because there are still enough healthy cells in the liver to do its job, Lau GK, Piratvi Suth T, Lu KX, Marcelin P, Thoug Sawat S et al (2005). At this point the liver can compensate or make up for the previous damage. Decompensated cirrhosis is very serious. People with decompensated cirrhosis notice a rapid decline in their health and will experience signs and symptoms of liver failure, Conjeevaram HS and Lokas (2003).

Although survival varies with age at diagnosis and extrahepatic comorbidities, 81 patients with compensated cirrhosis have a median survival of 12 years according to a pooled analysis of 806 prospectively followed up patients, *Karayiannis P (2003).* Survival is reduced after any decompensation. Patients with compensated cirrhosis and small varices have a 6% 1-year risk of bleeding, while patients with large varices and decompensated cirrhosis have a 42% to 76% 1-year risk of bleeding. 83 In-hospital mortality after variceal haemorrhage is approximately 14.5% overall and as low as 0% for patients with previously compensated cirrhosis, *Willington K and Jarvis B (2001)*. Ascites in the setting of cirrhosis was associated with a median survival of 1.1 years in a cohort of 13 265 patients enrolled in Medicare. Median survival time following incident overt hepatic encephalopathy was 0.92 years in a study of 49 164 patients with cirrhosis enrolled in Medicare, *Strader BB, Bacon BR, Lintsay KL, La Brecque DR, Morgan T et al (2002)*. 37, 44 Compared with patients with cirrhosis without any hepatic encephalopathy, covert hepatic encephalopathy was also associated with worse outcomes. Such outcomes included a higher 1-year risk of car crashes (17% of 97 patients with covert hepatic encephalopathy vs 3% of 70 without) and, in a cohort of 170 patients with cirrhosis (56% with covert hepatic encephalopathy), higher rates of hospitalization (47% vs 15%) and death (18% vs 3%), *Eisenberg PM, Davis RB, Ettner SL, Appel S, Wilkey S et al (1998).*

It is important to evaluate for the presence of cirrhosis in people with risk factors or any diagnosed chronic liver disease. While physical examination findings may be suggestive, it is recommended to stratify risk for all using the FIB-4 followed by electrography for at-risk patients, *Flora K, Hahn M, Rosen H and Bener K (1998)*. After identifying patients with cirrhosis, optimal care may involve referral to a hepatologist, liver cancer screening, and consideration of endoscopy for varices screening and/or initiation of nonselective β-blockers. BMI indicates body mass index, *Strader BB, Bacon BR, Lintsay KL, La Brecque DR, Morgan T et al (2002)*.

Patients with cirrhosis and a greater than 1.0-cm mass on screening ultrasound or with a

rising or elevated α-fetoprotein level (cutoff >20 ng/mL) should undergo further diagnostic workup to evaluate for HCC. Though biopsy is diagnostic, multiphasic contrast enhanced cross-sectional imaging can be used to make the diagnosis. A solid lesion exhibiting specific features (eg, arterial phase hyper enhancement and portal venous phase washout) in a patient with cirrhosis can be diagnosed as HCC, *Varga Z, Czompa A, Kakuk G, and Antus S (2001)*

Some signs and symptoms of decompensation include variceal bleeding, encephalopathy (Mental Confusion), ascites and jaundice. Large blood vessels (varices) can develop in the oesophagus (the food tube) and get bigger and bigger over time and may even burst open, *Dehmlow C, Erhard J and de Groot H (1996).* When this happens, the patient will vomit blood or notice the stool is black and tarry. If either of these things happens, patient should go to the emergency room immediately to get help and stop the bleeding. The risk of bleeding from varices can be reduced by finding them before any bleeding takes place, *Eisenberg PM, Davis RB, Ettner SL, Appel S, Wilkey S et al (1998).*

Then patient can start taking special blood pressure medicines (called beta-blockers) or have the varices treated by a special procedure in which tiny rubber bands are tied around the varices. Another problem caused by high pressure in the veins of the liver is ascites. Fluid leaks out of the veins and into the belly. As the belly fills with fluid, the abdomen can enlarge, like a balloon filled with water, *Wilasrusmee C, Kitturs S, Shad G, Siddiqui J, Bruch D et al (2000)*. The legs can get swollen too. This can be very uncomfortable. Another problem caused by high pressure in the veins of the liver is ascites. Ascites may go away with a low-salt diet, and with diuretics (water pills) ordered by the provider, *Dehmlow C, Erhard J and de Groot H (1996).* But sometimes a provider must drain the fluid from the belly using a special needle. When patient have ascites and suddenly get a fever or new belly pain, he should go to the emergency room immediately. These could be signs of a serious infection that can be life-threatening.

Other signs are jaundice, Bilirubin is a normal substance in the bloodstream, but when bilirubin levels are too high, there will be a yellowing of the eyes and skin called jaundice. People with jaundice also frequently have itching. A healthy liver helps regulate the bilirubin level in the body, but people with hepatitis and cirrhosis can develop high levels of bilirubin. Too much alcohol and some medicines can also lead to jaundice. If you suddenly develop jaundice, you should go to the emergency room immediately, *Neumann Up, Biermer M, Eurich D, Neuhaus P and Berg T (2010).*

A liver with decompensated cirrhosis also may not be able to get rid of toxic substances like ammonia (which comes from the intestines), and it may allow these substances to go into the brain and cause confusion. Besides confusion, toxins in the brain cause changes in the sleep, mood, concentration, and memory. In severe cases, these toxins can even cause a coma. These changes are all symptoms of hepatic encephalopathy. Patient with encephalopathy, may have problems driving, writing, and performing other activities of daily living. Signs of encephalopathy are trembling and hand "flapping." The provider might prescribe lactulose, a laxative syrup that makes your bowels move more often. The goal is to move the bowels two to three times a day, which helps get rid of ammonia, *Beinhardt S, Rasoul – Rockenschaub S, Maieron A, PH S-M and Hofer HPF (2012).*

As cirrhosis progresses and healthier liver cells are replaced with scarring the liver becomes very hardened and lumpy, making it very hard for blood to flow freely. This causes a build-up of pressure in the veins attached to the liver. Pressure increased in the portal vein is called portal hypertension, *Dehmlow C, Erhard J and de Groot H (1996)*. The increased pressure causes back-flow to the spleen causing an increase in size to the spleen (splenomegaly – big spleen). A larger spleen will hold onto most of the platelets which normally circulate in the blood and help with clotting, and you might bleed or bruise more easily. Signs that you may have developed portal hypertension will be picked up on your ultrasound and through your blood test results, *Varga Z, Czompa A, Kakuk G, and Antus S (2001)*.

Portal hypertension can also lead to oesophageal varices (like varicose veins in the gut), where some of the blood vessels in your oesophagus and gut become swollen and at risk of bleeding. Oesophageal varices can be detected by a gastroscopy, which involves placing a camera on a special tube down your throat. (Sedation can be given to help with the discomfort of the procedure.), *Pares A, Planas R, Tores M, Caballeria J, Viver JM et al (1998).* If the oesophageal varices are small all you might need is a special medication called a beta-blocker to bring down your pulse rate and blood pressure, *Durand JL., et al (2003).* If the oesophageal varices are larger they might be treated by banding, which can be done during the gastroscopy procedure. A gastroscopy might need to be repeated every 1- 3 years. If left untreated there is a risk of the blood vessels bursting, *Franchis De et al (2009).*

Persons with liver disease may present the non-specific generalized symptoms such as weakness or fatigue, specific symptoms suggesting the underlying cause (eg. Alcoholism) of the presence of cirrhosis and symptoms arising from associated Portal Hypertension (eg. Bleeding, jaundice, confusion, abdominal distention), *Targo K, Nozaki A, Ikeda T et al (2019).* The patient may have signs of cirrhosis, its complications (decompensate disease) and causes. Once the suspicion of liver disease has been raised, laboratory tests and diagnostic imaging will prove to be useful to confirm the clinical hypothesis that there is liver disease, but the extent of the abdominal liver enzymes such as transglutaminase and alkaline phosphatise, do not reflect the severity of the liver damage, *Jiao J, et al (2009).* In contrast, the extent of the abdominal blood tests reflects deranged hepatic synthetic or excretory function (albumin, bilirubin, INR) is useful to clarify the severity of the liver disease. For example, the Child-Pugh classification or the MELD score are useful to predict prognosis and help to establish when a liver transplantation may be necessary, *CDC Wonder (2018).*

When the patient presents with non-specific symptoms such as fatigue and malaise, or symptoms from the course of the liver cirrhosis or its complications, when the physical examination shows signs of cirrhosis, and the liver enzymes (ALT, AST, AP, GGT) and liver function tests (albumin, bilirubin, INR) are abnormal, then further blood tests are performed to establish the cause of the liver disease (e.g. HBV, HCU, ferritin, caeruloplasmin, AMA, quantitative immunoglobulins), as well as diagnostic imaging (e.g. abdominal ultrasound with/without Doppler ultrasound), and when tolerated, liver biopsy, *Arsani SK, Hall L, Hagan M et al (2019)*. The clinical examination must be detailed to look for manifestations of disease

beyond the liver itself. Depending upon the initial clinical findings, the search for the cause of the liver disease may need to be considered. Indirect indications of the presence of cirrhosis may be found on diagnostic imagery like MRI, TDM, Endoscopies and Ultrasound. The stage of cirrhosis must be established, *Foster C, Baki J, Nikirk S, Williams S, Parikh ND and Tapper EB (2020).*

The treatment for cirrhosis depends on what has caused it. Cirrhosis cannot usually and any complications, and stop the condition getting worse. There are many different medications that may be prescribed by the doctors at the liver cirrhosis. It is very important that they are taken exactly as prescribed. Some of the common medications that patient with liver cirrhosis may need are listed below, *Kanwal F et al (2010)*. Medications to prevent encephalopathy (Confusion) include Lactulose: lactulose is a liquid laxative medication. It works by absorbing the toxins in the body and passes them out with a bowel motion. It can increase the amount of times the open of bowels each day. Although this may be inconvenient it is extremely important that the patient with this condition continue with this medication. Other medication to prevent confusion is Rifaximin: rifaximin is a type of antibiotic that can reduce the amount of chemicals in the bowel and bloodstream that contribute to encephalopathy, *Sepantou SG, Safiri, Bisignamo C et al (2017).*

Medications to reduce fluid accumulation like ascites and/or ankle swelling, include diuretics such as spironolactone (Aldactone), frusemide (Lasix) and Bunetamide (Burinex) are three medications that can help to get off some excess fluid which is passed out in the urine. In this case, Doctor will need to monitor the kidney function (by blood tests) if patient take these medications and adjust the dose of the medications if they are putting too much strain on your kidneys, *Tapper EB and Parikh ND (2016)*. Propranolol (Inderal) is a medication to prevent variceal bleeding. Propranolol is a blood pressure lowering medication which can reduce the chance of having varices bleeding. When patient starts this medication, its blood pressure and heart rate will be monitored. If patient feel light headed when starting this medication, let doctor know as dose may need to be adjusted, *Arvanitri V et al (2010)*. The better medication to treat effectively liver cirrhosis according to the Clinician – Scientist and Medical Researcher Nicholas Culpepper is Silymarin (Silybon). Silymarin, an extract of the milk thistle herb (silybum marianum), has been in use for the treatment of Chronic Liver Diseases (Advanced Liver Fibrosis, Liver Cirrhosis and primary liver tumors) since ancient times. In the 15th century, Nicholas Culpepper, an English herbalist described the milk thistle as being good against the jaundice. Silymarin is composed of flavonolignans namely silybin, silydianin, silycristin as well as a diastereomer of silybin; isosilybin. Most of its hepatoprotective effect is attributed to silybon which constitutes 60-70% of the drug. It is poorly absorbed in the gastro-intestinal tract and primarily excreted in bile.

Silymarin is the most commonly consumed complementary and alternative medicine reported in patients with chronic Liver Cirrhosis. A study in the USA found the prevalence of use of herbal medications for the treatment of allied medical conditions to have increased from 2.5% to 12.1% between 1990 and 1997, *Johnson PJ, Ahamaraj S, Berhane S, Bonnett L and Ma YT (2021)* . Another study in the same country, reported that as much as 31% of patients attending a hepatology clinic in Oregon, are

using alternative remedies such as milk thistle. The presence of cirrhosis is associated with an increased risk of complications such as liver cancer and decompensation including ascites, hepatic encephalopathy, and variceal haemorrhage, *Kim HS, Yu X, Kramer J et al (2022).*

[See Annex — Figure 1: Liver Cirrhosis and Complications by Ultrasounds]

[See Annex — Figure 2: Liver Cirrhosis and Complications by Contrast – Enhanced CT Scan]

3. MATERIALS & METHODS

It is descriptive cross – sectional study done on 68 Cameroonians patients (53 men and 15 women) among 100 patients living with Liver Cirrhosis at the NGaoundere Protestant Hospital in Adamawa Region Cameroon from June 2023 to June 2024 after receiving Ethical Clearance and Research Authorization from The Health Work of the Evangelical Lutheran Church of Cameroon (HWELCC) Chairman.

Convenience sampling was done, all the cases of elective HCC admitted during the study period were included. Age >gt; 20, men and women, Cameroonians or not but residents in Cameroon. The simple size was 100 cirrhotic patients calculated was calculated using the formula n = (Z2 x p x q) / e2 where n = minimum sample size, z= confidence interval, p= q= 1 – p and e= margin of error, 5%.

The Adamawa region is a constituent region of the republic of Cameroon, it borders the Centre and East regions to the south, the Northwest and West regions to the Southwest, Nigeria to the West, the Central African Republic to the East, and the North region to the North. This mountainous area forms the barrier between Cameroon’s forested South and Savana North. At almost 64,000 km2 in land area, the Adamawa is the third largest of Cameroon’s ten regions.

The Health Work of the Evangelical Lutheran Church of Cameroon (HWELCC) possesses three large hospitals namely: The Ngaoundere Protestant Hospital (NPH), the NGaoubela Protestant Hospital (NGBELAPH) and the Garoua Boulai Protestant Hospital (GBLAIPH) and several Health Centres and Clinics, we have marked our passage particularly at the NGaoundere Protestant Hospital

The data for this study were collected using structured questionnaires, entered and analysed in Microsoft Excel 2016, confidence interval was calculated along with frequency and percentages for binary data. The inclusion criteria included only patients (adults) tested positive with Liver Cirrhosis, aged from 18 years old.

To assess the prevalence of HCC to these Cirrhotic Patients, we collected and analyzed data on patient's characteristics, clinical manifestations, diagnostic methods used, types of treatment given and some complications occurred before, during and after treatment.

4. RESULTS & DISCUSSION

The management of Liver Cirrhosis involves its risks factors, clinical manifestations, diagnostic, complications and treatment. One hundred Patients were recorded with Liver Cirrhosis at the Internal Medicine Department, among which we analyzed these characteristics on each patient and are summarized them by the tables.

[See Annex — Table 1. Results of Patient Characteristics per Liver risk factors at Ngaoundere Protestant Hospital]

[See Annex — Table 2. Results of Liver Cirrhosis Risk Factors analyzed on Cirrhotic Cameroonians Patients]

[See Annex — Table 3. Results of Clinical Manifestations analyzed on Cirrhotic Cameroonians Patients]

[See Annex — Table 4. Results of Diagnostic Methods used by Hospital on 100 Patients]

[See Annex — Table 5. Results of Treatment given by Hospital to the 100 Patients]

[See Annex — Table 6. Results of Liver Cirrhosis Complications occurred during and after treatment received]

The distribution of treatments received by cirrhotic Cameroonians patients about men included: silymarin most common drugs used for the treatment (92,45%), beta-blockers (3.77%), immunoglobulins (0%), antivirals (12.64%), diuretics (13.20%), and liver transplantation (0%). In the other hand, women, 86.66% received silymarin, 13.33% beta-blockers, 6.66% immunoglobulins, 60.00% antivirals (only for HBV/HCV etiology), 13.33% diuretics, and no liver transplants. Despite these treatments, gastrointestinal bleeding occurred in 16.98% for men and 66,66% for women, hepatocellular carcinoma (HCC) for 81.48% men and 80,00% for women, coagulopathy for 9,43% for men and 33,33% for women, portal hypertension 22.63% men and 33,33% for women, variceal hemorrhage 45,28% for men and 13,33% for women. Compared to the results of study conducted by the Authors Javed Iqbal Farooqi and Ruskhusana Javed Farooqi (200) to find out the prevalence of HCC in 410 Cirrhotic Patients. HCC was found in 45 patients lack of Cirrhosis appropriate treatment and most of HCC were caused by HBV and HCV infections.

5. CONCLUSION

Given the prevalence of hepatocellular carcinoma (HCC) occurring during treatment for liver cirrhosis, we concluded that these treatments were ineffective and lacked other determinants in the management process, such as addressing risk factors related to diagnosis, treatment, and complications, including viral loads of hepatitis B and C, liver toxicity levels, and the use of effective antiviral medications.

REFERENCES

Jonas M, Kelly DA, Mizerski J, et al. Clinical trial of lamivudine in children with chronic hepatitis B. N Engl J Med 2002;346:1706–13.

Lai CL, Lin HJ, Lau HJ, et al. Effect of recombinant a2 IFN with or without prednisolone in Chinese HBsAg carrier children. QJM 1991;78:155–63.

Bortolotti F, Jara P, Barbera C, et al. Long term effect of a interferon in children with chronic hepatitis B. Gut 2000; 46:715–8

Lampertico P, Del Ninno E, Vigano M, et al. Long term suppression of HBeAg negative chronic hepatitis B by 24 months’ interferon therapy. Hepatol 2003;37:756–63.

Kasper D.L., Fauci A.S., Longo D.L, et al: Harrison’s principles of internal medicine. 16th edition. 2005.

Keeffe EB, Dieterich DT, Han SH, Jacobson IM, Martin P, Schiff ER, et al. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States: 2008 update. Clin Gastroenterol Hepatol. Dec 2008;6(12):1315-41; quiz 1286.

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology. May 2009;49(5 Suppl):S72-84

Fattovich G, Stroffolini T, Zagni I, et al. Hepatocellular carcinoma in cirrhosis: incidence and risk factors. Gastroenterology 2004; 127(Suppl 1):S35–S50.

McMahon BJ, Alberts SR, Wainwright RB, et al. Hepatitis B-related sequelae. Prospective study in 1400 hepatitis B surface antigenpositive Alaska native carriers. Arch Intern Med 1990;150:1051– 1054.

Mizokami M, Orito E. Molecular evolution of hepatitis viruses. Intervirology 1999;42:159–165

Tanaka Y, Kurbanov F, Mano S, et al. Molecular tracing of the global hepatitis C virus epidemic predicts regional patterns of hepatocellular carcinoma mortality. Gastroenterology 2006;130: 703–714.

El-Serag HB, Rudolph KL. Hepatocellular carcinoma: epidemiology and molecular carcinogenesis. Gastroenterology 2007;132: 2557–2576.

Chang MH. Hepatitis B virus and cancer prevention. Recent Results Cancer Res 2011;188:75–84

Fattovich G, Bortolotti F, Donato F. Natural history of chronic hepatitis B: special emphasis on disease progression and prognostic factors. J Hepatol 2008;48:335–352

Yang JD, Kim WR, Coelho R, et al. Cirrhosis is present in most patients with hepatitis B and hepatocellular carcinoma. Clin Gastroenterol Hepatol 2011;9:64–70

Yang HI, Lu SN, Liaw YF, et al. Hepatitis B e antigen and the risk of hepatocellular carcinoma. N Engl J Med 2002;347:168–174

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology 2009;49(Suppl): S72–S84.

Chun HM, Roediger MP, Hullsiek KH, et al. Hepatitis B Virus Coinfection Negatively Impacts HIV Outcomes

in HIV Seroconverters. The Journal of Infectious Diseases. 2012;205(2):185-193. doi:10.1093/infdis/jir720

McMahon BJ. The natural history of chronic hepatitis B virus infection. Hepatology. 2009;49(5 Suppl):S45-55 2.

Schweitzer A,Horn J, Mikolajczyk RT, Krause G, Ott JJ Estimations of worldwide prevalence of chronic hepatitis B virus infection: a systematic review of data published between 1965 and 2013.

Lancet 2015; 386: 1546–55 3. Ott J, Stevens G, Groeger J, Wiersma S. Global epidemiology of Hepatitis B virus infection: new estimates of age-specific HBsAg seroprevalence and endemicity. Vaccine 2012;30(12): 2212–9 4.

Lukhwareni A, Burnett RJ, Selabe SG, Mzileni MO, Mphahlele MJ. Increased detection of HBV DNA in HBsAg-positive and HBsAgnegative SouthAfrican HIV/AIDS patients enrolling for Highly active antiretroviral therapy at a Tertiary Hospital. J Med Virol 2009;81(3):406–12 5.

Boyles TH, Cohen K. The prevalence of hepatitis B infection in a rural SouthAfrican HIV clinic. S Afr Med J 2011;101(7):470–1 6.

Firnhaber C, Reyneke A, Schulze D, et al. The prevalence of hepatitis B co-infection in a South African urban government HIV clinic. S Afr Med J 2008;98(7):541-4 7.

Burnett RJ, Ngobeni JM, Francois G, et al. Increased exposure to hepatitis B virus infection in HIV-positive South African antenatal women. Int J STD AIDS 2007;18(3):152–6 8.

Andersson MI, Maponga TG, Ijaz S, et al. The epidemiology of hepatitis B virus infection in HIV-infected and HIV-uninfected pregnant women in the Western Cape, South Africa. Vaccine 2013;31(47):5579-84 9.

Puoti M1, Torti C, Bruno R, Filice G, Carosi G. Natural history of chronic hepatitis B in co-infected patients. J Hepatol 2006; 44(1 Suppl):S65-70 10.

Maynard JE. Hepatitis B: Global importance and need for control. Vaccine 1990;8(SUPPL.):S18-S20

Mohd Hanafiah K, Groeger J, Flaxman AD, Wiersma ST. Global epidemiology of hepatitis virus infection: new estimates of age-specific antibody to HCV seroprevalence. Hepatology 2013;57(4):1333-42

Lavanchy D. The global burden of hepatitis C. Liver Int. 2009;29 Suppl 1:74-Ellis LA, Brown D, Conradie JD, Paterson A, Sher R, Millo J, et al. Prevalence of hepatitis C inSouth Africa: detection of anti-HCV in recent and stored serum. J Med Virol 1990;32(4):249- 51

Davis GL. Hepatitis C virus genotypes and quasispecies. Am J Med 1999;107(6B):21S-6S

Messina JP, Humphreys I, Flaxman A, et al. Global distribution and prevalence of hepatitis C virus genotypes. Hepatology. 2015;61(1):77–87

Benova L, Mohamoud YA, Calvert C, AbuRaddad LJ. Vertical transmission of hepatitis C virus: systematic review and meta-analysis. Clin Infect Dis. 2014;59(6):765–73

Floreani A. Hepatitis C and pregnancy. World J Gastroenterol. 2013;19(40):6714–20 Jafari S, Copes R, Baharlou S, Etminan M, Buxton J. Tattooing and the risk of transmission of hepatitis C: a systematic review and metaanalysis. Int J Infect Dis.2010;14(11):E928– E9

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Zein CO, Levy C, Basu A, Zein NN. Chronic hepatitis C and type II diabetes mellitus: a prospective cross-sectional study. Am J Gastroenterol. 2005;100(1):48-5

EASL Recommendations on Treatment of Hepatitis C 2018. J Hepatol 2018; 69(2): 461- 511

Smith JO, Sterling RK. Systematic review: non-invasive methods of fibrosis analysis in chronic hepatitis C. Alimentary pharmacology & therapeutics. 2009;30(6):557-76

Karmochkine M, Carrat F, Dos Santos O, Cacoub P, Raguin G. A case-control study of risk factors for hepatitis C infection in patients with unexplained routes of infection. J Viral Hepat. 2006;13(11):775–82

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Lau GK, Piratvisuth T, Luo KX, Marcellin P, Thongsawat S, et al. (2005) Peginterferon Alfa-2a, lamivudine, and the combination for HBeAgpositive chronic hepatitis B. N Engl J Med 352: 2682-2695. 2.

Conjeevaram HS, Lok AS (2003) Management of chronic hepatitis B. J Hepatol 38 Suppl 1: S90-103.

Karayiannis P (2003) Hepatitis B virus: old, new and future approaches to antiviral treatment. J Antimicrob Chemother 51: 761-785.

Wellington K, Jarvis B (2001) Silymarin: a review of its clinical properties in the management of hepatic disorders. BioDrugs 15: 465-489.

Strader DB, Bacon BR, Lindsay KL, La Brecque DR, Morgan T, et al. (2002) Use of complementary and alternative medicine in patients with liver disease. Am J Gastroenterol 97: 2391-2397.

Eisenberg DM, Davis RB, Ettner SL, Appel S, Wilkey S, et al. (1998) Trends in alternative medicine use in the United States, 1990-1997: results of a follow-up national survey. JAMA 280: 1569-1575.

Flora K, Hahn M, Rosen H, Benner K (1998) Milk thistle (Silybum marianum) for the therapy of liver disease. Am J Gastroenterol 93: 139-143.

Varga Z, Czompa A, Kakuk G, Antus S (2001) Inhibition of the superoxide anion release and hydrogen peroxide formation in PMNLs by flavonolignans. Phytother Res 15: 608-612.

Dehmlow C, Erhard J, de Groot H (1996) Inhibition of Kupffer cell functions as an explanation for the

hepatoprotective properties of silibinin. Hepatology 23: 749-754

Wilasrusmee C, Kittur S, Shah G, Siddiqui J, Bruch D, et al. (2002) Immunostimulatory effect of Silybum Marianum (milk thistle) extract. Med Sci Monit 8: BR439-443.

Neumann UP, Biermer M, Eurich D, Neuhaus P, Berg T (2010) Successful prevention of hepatitis C virus (HCV) liver graft reinfection by silibinin mono-therapy. J Hepatol 52: 951-952.

Beinhardt S, Rasoul-Rockenschaub S, Maieron A, PH S-M, Hofer H PF (2012) Intravenous Silibinin-therapy in patients with chronic hepatitis C in the transplant setting. J Hepatology 56: S77.

Pares A, Planas R, Torres M, Caballeria J, Viver JM, et al. (1998) Effects of silymarin in alcoholic patients with cirrhosis of the liver: results of a controlled, double-blind, randomized and multicenter trial. J Hepatol : 615-621.

Sepanlou SG, Safiri S, Bisignano C, et al; GBD 2017 Cirrhosis Collaborators. The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol Hepatol. 2020;5(3):245-266. doi:10.1016/S2468-1253(19) 30349-8

Tapper EB, Parikh ND. Mortality due to cirrhosis and liver cancer in the United States, 1999-2016. BMJ. 2018;362:k2817. doi:10.1136/bmj.k2817

Centers for Disease Control and Prevention. CDC WONDER: about provisional mortality statistics, 2018 through last month. Accessed March 13, 2023. https://wonder.cdc.gov/mcd-icd10-provisional. html

Serper M, Kaplan DE, Taddei TH, Tapper EB, Cohen JB, Mahmud N. Nonselective beta blockers, hepatic decompensation, and mortality in cirrhosis: a national cohort study. Hepatology. 2023;77(2): 489-500. doi:10.1002/hep.32737

Flemming JA, Dewit Y, Mah JM, Saperia J, Groome PA, Booth CM. Incidence of cirrhosis in young birth cohorts in Canada from 1997 to 2016: a retrospective population-based study. Lancet Gastroenterol Hepatol. 2019;4(3):217-226. doi:10. 1016/S2468-1253(18)30339-X

Hirode G, Saab S, Wong RJ. Trends in the burden of chronic liver disease among hospitalized US adults.JAMA Netw Open. 2020;3(4):e201997-e201997. doi:10.1001/jamanetworkopen.2020.1997

Foster C, Baki J, Nikirk S, Williams S, Parikh ND, Tapper EB. Comprehensive health-state utilities in contemporary patients with cirrhosis. Hepatol Commun. 2020;4 (6):852-858. doi:10.1002/hep4. 1512

Asrani SK, Hall L, Hagan M, et al. Trends in chronic liver disease-related hospitalizations: a population-based study. Am J Gastroenterol. 2019;114(1):98-106. doi:10.1038/s41395-018- 0365-4

Tarao K, Nozaki A, Ikeda T, et al. Real impact of liver cirrhosis on the development of hepatocellular carcinoma in various liver diseases-meta-analytic assessment. Cancer Med. 2019;8(3):1054-1065. doi:10.1002/cam4.1998

Carbonell N, Pauwels A, Serfaty L, Fourdan O, Lévy VG, Poupon R. Improved survival after variceal bleeding in patients with cirrhosis over the past two decades. Hepatology. 2004;40(3):652-659. doi: 10.1002/hep.20339

Arvaniti V, et al. Infections in patients with cirrhosis increase mortality four fold and should be used in determining prognosis. Gastroenterology 2010;139:1246-1256.

Berzigott A., et al. Obesity is an independent risk factor for clinical decompensation in patients with cirrhosis. Hepatology 2011;54:555-561.

Dienstag J.L., et al. A prospective study of progression in compensated, histologically advanced chronic hepatitis C. Hepatology 2011;54:396-405.

Durand F, et al. Assessment of the prognosis of cirrhosis: Child-Pugh versus MELD. Journal of Hepatology2005;42.

Franchis de, et al. Non-invasive diagnosis of cirrhosis and the natural history of it complications. Best Practice & Research Clinical Gastroenterology 2007;21(1):3-18.

Friedman SL. Mechanisms of hepatic fibrogenesis. Gastroenterology 2008;134:1655-1669.

Garcia-Tsao G, et al. Management and treatment of patients with cirrhosis and portal hypertension: recommendations from the Department of Veterans Affairs Hepatitis C Resource Center Program and the National Hepatitis C program. The American Journal of Gastroenterology 2009;104(7):1802-1829

Greenbaum L.E. and Wells R.G. The role of stem cells in liver repair and fibrosis. The International Journal of Biochemistry and Cell Biology 2011;43:222-229.

Jiao J, et al. Hepatic fibrosis. Current Opinion in Gastroenterology 2009;25(3):223-229.

Kanwal F, et al. An explicit quality indicator set for measurement of quality of care in patients with cirrhosis. Clinical Gastroenterology and Hepatology 2010;8:709

Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2021; 71: 209–49.

Siegel RL, Miller KD, Fuchs HE, Jemal A. Cancer statistics, 2022. CA Cancer J Clin 2022; 72: 7–33. 3 Petrick JL, Florio AA, Znaor A, et al. International trends in hepatocellular carcinoma incidence, 1978–2012. Int J Cancer 2020; 147: 317–30.

Sun J, Althoff KN, Jing Y, et al. Trends in hepatocellular carcinoma incidence and risk among persons with HIV in the US and Canada, 1996–2015.

JAMA Netw Open 2021; 4: e2037512. 5 Chen T, Liu J, Li Y, Wei S. Burden of disease associated with dietary exposure to aflatoxins in China in 2020. Nutrients 2022; 14: 1027.

Romano A, Angeli P, Piovesan S, et al. Newly diagnosed hepatocellular carcinoma in patients with advanced hepatitis C treated with DAAs: a prospective population study. J Hepatol 2018; 69: 345–52.

Dave S, Park S, Murad MH, et al. Comparative effectiveness of entecavir versus tenofovir for preventing hepatocellular carcinoma in patients with chronic hepatitis B: a systematic review and meta-analysis. Hepatology 2021; 73: 68–78.

Papatheodoridis GV, Idilman R, Dalekos GN, et al. The risk of hepatocellular carcinoma decreases after the first 5 years of entecavir or tenofovir in Caucasians with chronic hepatitis B. Hepatology 2017; 66: 1444–53.

Chiang CJ, Yang YW, You SL, Lai MS, Chen CJ. Thirty-year outcomes of the national hepatitis B immunization program in Taiwan. JAMA 2013; 310: 974–76.

Kanwal F, Kramer JR, Asch SM, Cao Y, Li L, El-Serag HB. Long-term risk of hepatocellular carcinoma in HCV patients treated with direct acting antiviral agents. Hepatology 2020; 71: 44–55.

Kim HY, Lampertico P, Nam JY, et al. An artificial intelligence model to predict hepatocellular carcinoma risk in Korean and Caucasian patients with chronic hepatitis B. J Hepatol 2022; 76: 311–18.

Kim HS, Yu X, Kramer J, et al. Comparative performance of risk prediction models for hepatitis B-related hepatocellular carcinoma in the United States. J Hepatol 2022; 76: 294–301.

Zheng J, Seier K, Gonen M, et al. Utility of serum inflammatory markers for predicting microvascular invasion and survival for patients with hepatocellular carcinoma. Ann Surg Oncol 2017; 24: 3706–14.

Johnson PJ, Dhanaraj S, Berhane S, Bonnett L, Ma YT. The prognostic and diagnostic significance of the neutrophil-tolymphocyte ratio in hepatocellular carcinoma: a prospective controlled study. Br J Cancer 2021; 125: 714–16.

Bruix J, Cheng AL, Meinhardt G, Nakajima K, De Sanctis Y, Llovet J. Prognostic factors and predictors of sorafenib benefit in patients with hepatocellular carcinoma: analysis of two phase III studies. J Hepatol 2017; 67: 999–1008.

Scheiner B, Pomej K, Kirstein MM, et al. Prognosis of patients with hepatocellular carcinoma treated with immunotherapy—development and validation of the CRAFITY score. J Hepatol 2022; 76: 353–63. 87 Vogel A, Martinelli E, ESMO Guidelines Committe. Updated treatment recommendations for hepatocellular carcinoma (HCC) from the ESMO Clinical Practice guidelines. Ann Oncol 2021; 32: 801–05.

Bruix J, Chan SL, Galle PR, Rimassa L, Sangro B. Systemic treatment of hepatocellular carcinoma: an EASL position paper. J Hepatol 2021; 75: 960–74.

The International Liver Cancer Association. ILCA systemic therapy guidance. https://ilca-online.org/wp-content/uploads/2020/06/Systemic-therapy-guidelines-V1.2.pdf (accessed July 26, 2022).

Kudo M, Kawamura Y, Hasegawa K, et al. Management of hepatocellular carcinoma in Japan: JSH consensus statements and recommendations 2021 update. Liver Cancer 2021; 10: 181–223.

Chen LT, Martinelli E, Cheng AL, et al. Pan-Asian adapted ESMO clinical practice guidelines for the management of patients with intermediate and advanced/relapsed hepatocellular carcinoma: a TOS-ESMO initiative endorsed by CSCO, ISMPO, JSMO, KSMO, MOS and SSO. Ann Oncol 2020; 31: 334–51.

Reig M, Forner A, Rimola J, et al. BCLC strategy for prognosis prediction and treatment recommendation: the 2022 update. J Hepatol 2021; 76: 681–93.

Lang H, Sotiropoulos GC, Dömland M, et al. Liver resection for hepatocellular carcinoma in non-cirrhotic liver without underlying viral hepatitis. Br J Surg 2005; 92: 198–202.

Viganò L, Conci S, Cescon M, et al. Liver resection for hepatocellular carcinoma in patients with metabolic syndrome: a multicenter matched analysis with HCV-related HCC. J Hepatol 2015; 63: 93–101.

Kabir T, Tan ZZ, Syn NL, et al. Laparoscopic versus open resection of hepatocellular carcinoma in patients with cirrhosis: meta analysis. Br J Surg 2021; 109: 21–29. 96 Ivanics T, Claasen MP, Patel MS, et al. Long-term outcomes of laparoscopic liver resection for hepatocellular carcinoma: a propensity score matched analysis of a high-volume North American center. Surgery 2021; 171: 982–91.

Berzigotti A, Reig M, Abraldes JG, Bosch J, Bruix J. Portal hypertension and the outcome of surgery for hepatocellular carcinoma in compensated cirrhosis: a systematic review and meta - analysis. Hepatology 2015; 61: 526–36.

Berardi G, Morise Z, Sposito C, et al. Development of a nomogram to predict outcome after liver resection for hepatocellular carcinoma in Child-Pugh B cirrhosis. J Hepatol 2020; 72: 75–84.

Azoulay D, Ramos E, Casellas-Robert M, et al. Liver resection for hepatocellular carcinoma in patients with clinically significant portal hypertension. JHEP Rep 2020; 3: 100190.

Citterio D, Facciorusso A, Sposito C, Rota R, Bhoori S, Mazzaferro V. Hierarchic interaction of factors associated with liver decompensation after resection for hepatocellular carcinoma. JAMA Surg 2016; 151: 846–53.

Serenari M, Han KH, Ravaioli F, et al. A nomogram based on liver stiffness predicts postoperative complications in patients with hepatocellular carcinoma. J Hepatol 2020; 73: 855–62.

Allaire M, Goumard C, Lim C, Le Cleach A, Wagner M, Scatton O. New frontiers in liver resection for hepatocellular carcinoma. JHEP Rep 2020; 2: 100134.

Marasco G, Alemanni LV, Colecchia A, et al. Prognostic value of the albumin-bilirubin grade for the prediction of post-hepatectomy liver failure: a systematic review and meta-analysis. J Clin Med 2021; 10: 2011.

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Olivier Lieuga, Tatiana Jiengoué, and Augustine Nji Asakizi. (2026). Prevalence rate of Hepatocellular Carcinoma on 68 Cameroonians among 100 patients with Liver Cirrhosis during treatment at The NGaoundere Protestant Hospital in Adamawa Region Cameroon . IQ Research Journal, 5(2), IQRJ-V05I02-26004014. www.iqresearchjournal.com

ANNEXES

Annex I — Table 1. Results of Patient Characteristics per Liver risk factors at Ngaoundere Protestant Hospital

Prevalence Rate of HCC Patient Characteristics	Cameroonians		Chadians		Nigerians		Others Nationalities	
	n	%	n	%	n	%	n	%
Gender								
Male	53	77,94	12	60	5	55,55	3	100
Female	15	22,05	8	40	4	44,44	0	0,00
Age (Years Old)								
18 – 35	42	61,76	9	45	6	66,66	3	100
36 - 55	17	25	7	35	3	33,33	0	0,00
55 – Over	9	12,23	4	20	0	0,00	0	0,00
Marital Status								
Single	52	76,47	20	10	9	100	0	0,00
Married	12	17,64	0	0,00	0	0,00	3	100
Divorced	4	5,88	0	0,00	0	0,00	0	0,00
Education Levels								
Low Education	47	69,11	20	100	9	100	3	100
High Education	21	30,88	0	0,00	0	0,00	0	0,00
Incomes								
Low Income	68	100	20	100	9	100	3	100
High Income	0	0,00	0	0,00	0	0,00	0	0,00

Annex II — Table 2. Results of Liver Cirrhosis Risk Factors analyzed on Cirrhotic Cameroonians Patients

Results of Liver Cirrhosis Risk Factors	HBV		HCV		HBV + HCV		Toxins		NASH		CL		IMLD		BC	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Gender																
Male (53)	38	52,05	24	32,87	7	9,58	3	4.10	1	1,36	0	0,00	0	0,00	0	0,00
Female (15)	12	44,44	9	33,33	4	14.81	1	3,70	1	3,70	0	0,00	0	0,00	0	0,00

Annex III — Table 3. Results of Clinical Manifestations analyzed on Cirrhotic Cameroonians Patients

Results of Liver Cirrhosis Clinical Manifestations	Anorexia		Abdomen Distention		Liquid Collection		Hepatosplenomegaly		Abdominal Mass		Ascites		Jaundice	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Gender														
Men (53)	12	16,43	21	28,76	3	4,10	13	17,80	10	13,69	7	9,58	7	9,58
Women (15)	10	37,03	7	25,92	0	0,00	6	22,22	2	7,40	2	7,40	0	0,00

Annex IV — Table 4. Results of Diagnostic Methods used by Hospital on 100 Patients

Results of Liver Cirrhosis Diagnostic	Liver Biopsy		AST		ALT		Abdominal Ultrasounds		Fibroscan		Fibrotest - Actitest		CT Scan	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Gender														
Men (53)	0	0,00	21	28,76	21	28,76	53	100	8	10,95	7	9,58	7	9,58
Women (15)	0	0,00	12	44,44	12	44,44	15	100	2	7,40	2	7,40	0	0,00

Annex V — Table 5. Results of Treatment given by Hospital to the 100 Patients

Results of Liver Cirrhosis Treatment	Silymarin		Beta – Blockers		Immunoglobulin		Antiviral Drugs		Diuretics		Liver Transplantation	
	n	%	n	%	n	%	n	%	n	%	n	%
Gender												
Men (53)	49	92,45	2	3,77	0	0,00	12	22,64	7	13,20	0	0,00
Women (15)	13	86,66	2	13,33	1	6,66	9	60,00	2	13,33	0	0,00

Annex VI — Table 6. Results of Liver Cirrhosis Complications occurred during and after treatment received

Results of Liver Cirrhosis Complications	Digestive Hemorrhage		Hepatocellular Carcinoma		Coagulopathy		Portal Hypertension		Variceal Hemorrhage		Hepatic Encephalopathy	
	n	%	n	%	n	%	n	%	n	%	n	%
Gender												
Men (53)	9	16,98	47	88,67	5	9,43	12	22,63	24	45,28	2	3,77
Women (15)	10	66,66	12	80,00	5	33,33	5	33,33	2	13,33	2	13,33

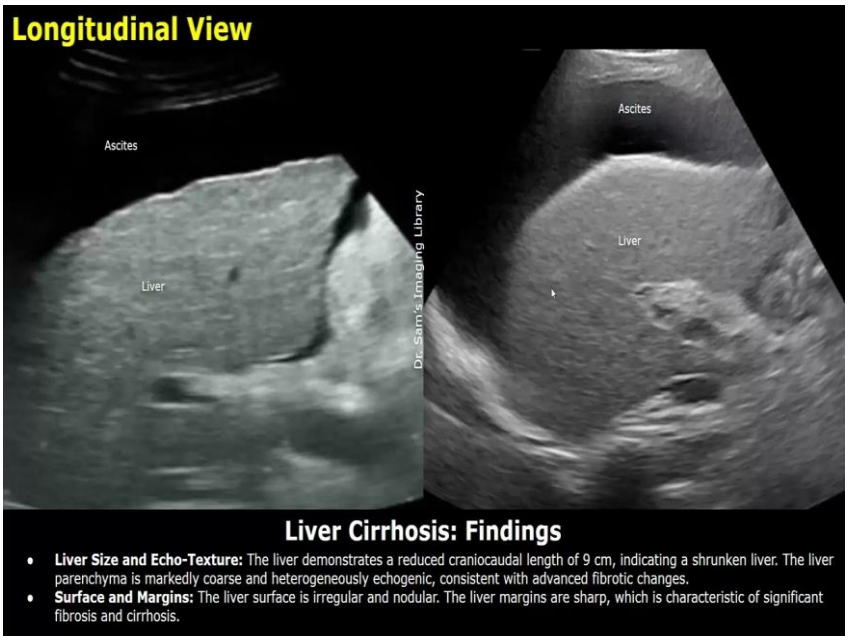


Figure 1. *Liver Cirrhosis and Complications by Ultrasounds*

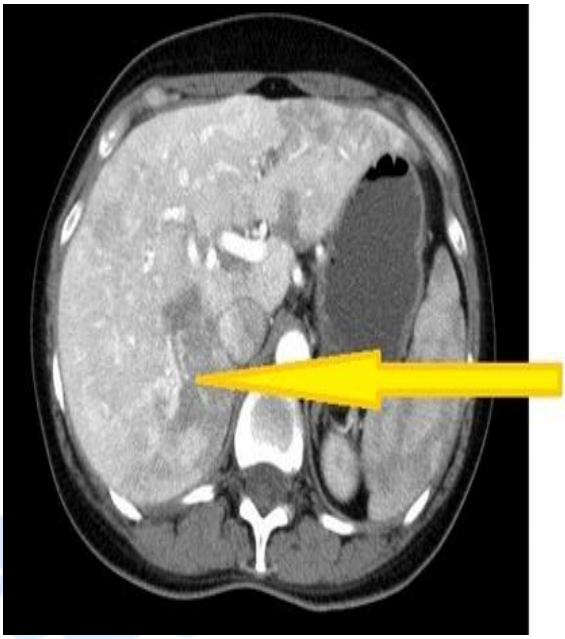


Figure 2. *Liver Cirrhosis and Complications by Contrast – Enhanced CT Scan*

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Prevalence of Liver Cirrhosis on patients with Chronic Hepatitis B during treatment by Tenofovir Disoproxil 300 mg only at The NGaoundere Protestant Hospital in Adamawa Region Cameroon

Olivier Lieuga[a] ,Tatiana Jiengoué[a], and Augustine Nji Asakizi[a]

Affiliations

o. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Olivier Lieuga

Email: olivierlieuga@kesmondsuniversity.org

Paper ID: IQRJ-V05I02-26005015

ABSTRACT

Introduction: Hepatitis virus B infection are the major public Health concern with the implementation of hepatitis immunization program in 1984 and the chronic viral hepatitis therapy program in 2003, its prevalence rate has dropped from 8% to 2% - 7% in the past few years. However, the Global Hepatitis Report (2017) estimated that nearly 257 million people worldwide were still living with chronic HBV infection, which would increase the risk of liver fibrosis, liver cirrhosis and hepatocellular carcinoma (HCC) in those infected.

Methods: It is descriptive cross – sectional study prevalence of liver cirrhosis done on 20 patients tested positives with HBV confirmed by the following exams such as ELISA HBV, Viral Load HBV, Hepatitis D Serology Test and Five HBV Biomarkers among 75 patients presented some clinical manifestations of Chronic Hepatitis at the NGaoundere Protestant Hospital in Adamawa Region Cameroon from June 2023 to August 2023 after receiving Ethical Clearance and Research Authorization from The Health Work of the Evangelical Lutheran Church of Cameroon (HWELCC) Chairman. Convenience sampling was done. The analysis included management of risk factors, clinical manifestations, diagnostic methods, treatment and complications of Chronic Hepatitis B.

Results: The prevalence of chronic hepatitis B on 75 patients was 26,66% and Out to 20 patients tested positive of chronic hepatitis, the prevalence of Liver Cirrhosis were 85%.

Conclusions: This study showed that the prevalence of Liver Cirrhosis estimated high to the patients tested positive with chronic hepatitis B despite tenofovir disoproxil 300 mg received caused by the non-management of Hepatitis B genotype and complications

Keywords: *Hepatocellular Carcinoma, Liver Cirrhosis ,Hepatitis B, Hepatitis C*

1. INTRODUCTION

Hepatitis is a general term referring to an inflammation of the liver. It occurs as a result of infection with various pathogens exposure to alcohol, medications, chemicals and toxins, and autoimmune disorders, Colin JF et al.. (1999). There are five types of hepatotrophic viruses; Hepatitis A,B,C,D and E (HAV, HBV, HCV, HDV and HEV), named according to the order in which there were discovered, Thio CL, et al,.. (2002). In the 1960s, only two types were known (A and B), but by the late 1970s, and beyond, new viruses (C and E) were discovered, hepatitis viruses are either Ribonucleic acid (RNA) like hepatitis A,C,D and E, or Deoxyribonucleic (DNA) viruses like hepatitis B, Central Bureau of Health Intelligence (2016). The five hepatotrophic viruses are broadly classified into group namely, enteric and parenteral.

[See Annex — Table : Classification of Hepatotrophic Group Viral Type]

According to the Dr S. Venkatesh and Dr R.K. Dhiman (2018), viral hepatitis is recognized as a public health problem globally various etiological agents (Hepatitis A, B, C, D and E viruses) have been implicated that can lead to acute, chronic or sequel of chronic infection. While hepatitis A and E are often the cause for sporadic or outbreaks of hepatitis, hepatitis B and C can either clear spontaneously or can lead to chronic infection, and there after sequelal like cirrhosis and hepatocellular carcinoma (HCC), Chun HM, Roediger MP, Hullsiek KH, et al,… (2012). Chronic HBV infection accounts for 40 – 50% of HCC and 20 – 30% cases of cirrhosis in the World and chronic HCV infection accounts for 12 – 32% of HCC and 12 – 20% of cirrhosis, Colin JF, Cazals-Hatem D, Loriot MA, et al (1999).

Transmission of HBV and HCV can occur via sexual, blood – blood contact or vertically (Mother – to – child), there are several known high – risk groups for acquiring a HBV or HCV infection, these include people interned in prisons, people who inject drugs, people with multiple sexual partners, migrants originating from endemic regions and new – borns from HBV or HCV chronically infected mothers called vertical transmission, Lozano R, Naghavi M, Foreman K, Lim S, Shiburya K, Aboyans V, et al (2010). The risk of developing chronic HBV infection depends on the age at infection, chronic infection results in 90% of infants infected at birth, in 30 to 50% of children infected between the age of one to four year, and in 1 to 10% of those infected at older age or as results, WHO, Global Hepatitis Report (2017). According to Schweitzer et al (2014), an estimated 248 million people were chronically infected with Chronic Hepatitis B and C Worldwide in 2010. WHO Executive Board (2019), Peter Byass (2014)

2. RELATED WORKS

HBV, a double – stranded DNA virus, belongs to the family of helped viruses, HBV infection is a global public health problem, McMahon BJ (2009). Perinatal transmission and occasionally horizontal transmission early in life most common in high prevalence areas, sexual contact and percutaneous transmission also contribute to the transmission of HBV, Boyles TH and Cohen K (2011). HBV is an entirely vaccine – preventable disease. Patients with chronic HBV infection have a 15 to 40 per cent risk of developing cirrhosis, liver failure and / or HCC, and 15 to 25 per cent risk of dying from HBV related liver diseases, Mc Maham BJ (2009).The HBV genotypes influence the spectrum of disease, the risk of HCC and the response to antiviral treatment genotypes A,D and E are the predominant HBV genotypes in Africa, genotype A predisposes to chronicity with an elevated risk

of HCC, but has an increased response rate to interferon therapy, the relative risk of HCC is four times higher in the people affected, Burnett RJ, Ngobeni JM, Francois G, et al.(2007). Genotype D has a reduced response rate to interferon therapy, and acute infection is associated with increased risk of acute liver failure. HBV is transmissible via perinatal, percutaneous or sexual exposure to HBV – infected body fluids including serum, saliva, semen and vaginal fluids, Mc Maham BJ (2009). All HBs-Ag positive individuals more infections as they have higher rates of HBV replication, Schweitzer A,Horn J, Mikolajczyk RT, Krause G, Ott JJ (1965 and 2013).

According to Ott J, Stevens G, Groeger J, Wiersma S (2012), the clinical presentation of chronic HBV infections is variable, the risk of chronicity is dependent on age of acute infection; 70 to 95 per cent for infants exposed perinatally (HbeAg – positive Mother), 25 to 50 per cent for children aged between one and five years, six to 10 per cent for five to 20 years and one to three per cent for adults older than 20 years. It is important to establish the phase of chronic infection as this determines the risk of cirrhosis and HCC, the frequency of follow up and the need for treatment, Boyles TH, and Cohen K (2011). Many patients with chronic HBV are asymptomatic (unless they have decompensated cirrhosis or have non – specific symptoms such as fatigue, some patients experience exacerbation of the infection which may be asymptomatic, mimic acute hepatitis or manifest as hepatic failure, Andersson MI, Maponga TG, Ijaz S, et al (2013). Physical examination may be normal, or there may be stigmata of chronic liver disease, jaundice, splenomegaly, ascites, peripheral edema, upper gastrointestinal bleed and encephalopathy may be present in patients with decompensated cirrhosis, McMahon BJ (2009).

There are five different phases of chronic infection such as HbeAg positive chronic HBV infection (immune Tobrant), HbeAg positive chronic HBV (immune clearance), HbeAg negative chronic infection (immune control), HbeAg negative chronic HBV (immune escape) and occult HBV, Maynard JE. (1990). The syndrome of fulminant HBV is characterized by jaundice, hepatic encephalopathy, coagulopathy (INR is more than 1,5) occuring within eight weeks of the onset of the acute illness. Its complications include development of acute portal hypertension, hepatorenal syndrome, cardiorespiratory dysfunction, metabolic disturbances, raised intercranial pressure, life threatening cerebral edema, susceptibility to bacterial and fungal infections, Hoffmann CJ, Thio CL. (2007). In this case, survival rates are estimated for 12 to 36 per cent and liver transplantation is the first therapy choice.

HBV surface antigene (HbsAg) is the key marker in the diagnosis of HBV infection. Laboratory testing during chronic phase reveals elevation in the concentration of alanine (ALT) and asparate (AST) aminotransferase levels; values up to 1000 to 2000 international units /L are typically seen during the chronic phase with ALT being higher than AST, Burnett RJ, Ngobeni JM, Francois G, et al. (2007). The serum bilirubin concentration may be normal in patients with anicteric hepatitis but the prothrombin time is the best indicator of prognosis, Delemos and Mark w. Russo (2019). Routine assessment of HbsAg – positive persons is needed to guide management and indicate the need for treatment, this generally includes assessment serological markers of HBV infection, measurement of HBV DNA levels, assessing

severity of liver disease by liver enzymes, non – invasive tests (NITs) such as asparate aminotransferase (AST) to platelet ration index (APRI), FIB – 4, transient elastography (Fibroscan) and liver biopsy, Schaefer S (2005)

Current treatment of chronic viral hepatitis B with interferon and nucleoside analogues have remained unsatisfactory with seroconversion of hepatitis B virus from a replicative to a non-replicative state occurring in only 15-32% of patients, Lau GK, Piratvisuth T, Luo KX, Marcellin P, Thongsawat S, et al. (2005). Along with this high rate of treatment failure, is the prohibitive cost of these medications especially interferon-based medications and associated numerous side effects. This is especially true in Nigeria where out of pocket payment for healthcare services is still very much the rule, Conjeevaram HS, Lok AS (2003). Many patients are therefore too eager to explore the use of complementary and alternative treatment with their optimistic and somewhat sketchy evidence of benefit, Wellington K, Jarvis B (2001)

It is important to establish the phase of chronic HBV and the need for anti – viral therapy depending on disease activity, HBV DNA level, the presence of advanced fibrosis, cirrhosis or the use of immunosuppressive therapy, Kramvis A and Kew MC (2005). The objectives of treatment are virological suppression, biochemical remission, histological improvement and prevention of complications such as Cirrhosis, Hepatocarcinoma and Extrahepatic manifestations, Zhou Y and Holmes EC (2007). Its management includes assessment of liver disease prior to therapy, there are age and disease duration, complications, of chronic HBV, assessment of compliance with follow up visits and medication, family history of HBV infection, complications of cirrhosis and HCC, full blood count and differential count, liver profile, like total bilirubin, conjugated bilirubin, ALT, AST, ALP, Gamma GT, serum albumin and INR to assess synthetic function and serum creatinine, Schaefer S (2005). It is also important to control serological assessment which includes HbsAg, HbeAg and anti Hbe +/- IgM anti HBc (low positive with a flare), IgG anti HBc (if assessing for occult HBV or previous cleared infection. The decision to start treatment is based on the likelihood of sustained response to treatment and the risk of hepatic morbidity and mortality. In general, the indications are based on three criteria such as serum HBV DNA levels, serum alanine aminotransferase (ALT) levels and severity of liver disease, Araujo NM (2015).

Antiviral therapy is the most important treatment for patients with chronic HBV infection. In addition, there are anti – inflammatory, anti – oxidation, liver protection drugs, anti – fibrosis drugs and Immune regulatory treatment option, WHO (2017). Treatment is long term and there is a risk of relapse after suspension. Currently, there are five antiviral drugs such as lamivudine, telbivudine, entecavir, adefovir and tenofovir. Chronic HBV infection is a risk factor for developing HCC, even in the absence of cirrhosis. In Hong Kong, CHB accounts for 80% of incident HCC. Antiviral therapy does not completely eliminate the risk for HCC, Mc Mahon BJ (2009). Therefore, surveillance for HCC is crucial in reducing the morbidity and mortality of HBV infection. HCC may still develop even after spontaneous HBsAg loss, but the risk is lower if HBsAg loss is achieved at a younger age and in the absence of significant fibrosis, Chun HM, Roedigero MP, Hull Sick KH et al (2012).

Cirrhosis and HCC are the two major long-term complications of CHB. Patients with cirrhosis

and HCC require close follow-up and sometimes inpatient care with multi-disciplinary management, Yang HI, Lu SN, Liaw YF et al (2002). For instance, patients with cirrhosis may require endoscopic screening for varices, admissions for abdominal paracentesis or treatment of hepatic encephalopathy. Patients with HCC often require multi-disciplinary care involving physicians, surgeons, oncologists, and radiologists to formulate the best treatment strategy, Araujo NM (2015).

3. MATERIALS & METHODS

It is descriptive cross – sectional study prevalence of liver cirrhosis done on 20 patients tested positives with HBV confirmed by the following exams such as ELISA HBV, Viral Load HBV, Hepatitis D Serology Test and Five HBV Biomarkers among 75 patients presented some clinical manifestations of Chronic Hepatitis at the NGaoundere Protestant Hospital in Adamawa Region Cameroon from June 2023 to August 2023 after receiving Ethical Clearance and Research Authorization from The Health Work of the Evangelical Lutheran Church of Cameroon (HWELCC) Chairman. Convenience sampling was done. The analysis included management of risk factors, clinical manifestations, diagnostic methods, treatment and complications of Chronic Hepatitis B.

Convenience sampling was done, all the cases of elective Chronic Hepatitis B admitted during the study period were included. Age > 20 residents in Cameroon. The simple size was 25 Hepatitis calculated was calculated using the formula n = (Z2 x p x q) / e2 where n = minimum sample size, z= confidence interval, p= q= 1 – p and e= margin of error, 5%.

The Adamawa region is a constituent region of the republic of Cameroon, it borders the Centre and East regions to the south, the Northwest and West regions to the Southwest, Nigeria to the West, the Central African Republic to the East, and the North region to the North. This mountainous area forms the barrier between Cameroon's forested South and Savana North. At almost 64,000 km2 in land area, the Adamawa is the third largest of Cameroon's ten regions.

The Health Work of the Evangelical Lutheran Church of Cameroon (HWELCC) possesses three large hospitals namely: The Ngaoundere Protestant Hospital (NPH), the NGaoubela Protestant Hospital (NGBELAPH) and the Garoua Boulai Protestant Hospital (GBLAIPH) and several Health Centres and Clinics, we have marked our passage particularly at the NGaoundere Protestant Hospital

The data for this study were collected using structured questionnaires, entered and analysed in Microsoft Excel 2016, confidence interval was calculated along with frequency and percentages for binary data. The inclusion criteria included only patients (adults) tested positive with Liver Cirrhosis, aged from 18 years old.

4. RESULTS & DISCUSSION

To assess the prevalence of Liver Cirrhosis to these HBV Patients, we collected and analyzed data on patient's characteristics, clinical manifestations, diagnostic methods used, types of treatment given and some complications occurred before, during and after treatment.

[See Annex — Table 1. Results of Patient Characteristics]

[See Annex — Table 2. Results of Chronic Hepatitis B Clinical Manifestations]

[See Annex — Table 3. Results of Exams and Tests done to confirm Chronic Hepatitis B Patients]

[See Annex — Table 5. Results of Chronic Hepatitis B Treatment]

[See Annex — Table 6. Results of Hepatitis B Complications analyzed on 100 Patients]

The results concerned exams, tests, treatment and complications occurred during the period of study used by the Hospital to manage chronic hepatitis B to the patient tested positive with HBV. 100% patients have done Elisa Hepatitis B, 45% have done viral load HBV and Hepatitis B Serology, 100% for five biomarkers HBV, 70% for abdominal ultrasounds and 0% for genotype HBV and Liver Function Tests. Tenofovir disoproxil 300 mg has been the only drugs given to these patients. Consequently, we obtained 85% of these 20 patients with liver cirrhosis during the only treatment by tenofovir disoproxil 300 mg. This issue should be solved if the patient had complete their exams by Genotype HBV, Liver function Test and others related tests in order to manage the complications of Chronic Hepatitis B. Certain parameters include the problem of low income observed to some patients. The cost of genotype HBV, viral load HBV and Liver Function Tests are very expensive. Long-term treatment (5+ years) with 300 mg Tenofovir Disoproxil Fumarate (TDF) for Chronic Hepatitis B (CHB) effectively suppresses HBV DNA, leading to significant regression of cirrhosis in 71–74% of patients and low, newly diagnosed hepatocellular carcinoma (HCC) rates (2.1–4.0%). Despite this, cirrhosis remains a significant baseline condition, with studies showing 48.3% of patients may have liver cirrhosis before starting TDF, Lancet (2013).

5. CONCLUSION

The shortcomings observed in the diagnosis and treatment of Chronica Hepatitis B in patients at the Protestant Hospital in N'Gaoundere have caused multiple complications such as Liver Cirrhosis, Fulminant Hepatitis and Pulmonary Embolism, death and non-cure of patients.

The management of Hepatitis B at the NGaoundere is not effective and is subject to several challenges, namely: the absence of internists and gastroenterologists, poorly qualified staff, lack of in-depth diagnostic equipment, lack of appropriate drugs for the treatment of viral Hepatitis B infection, unsuitable and outdated technical platform... From the recommendations, we recommend to the management of the Hospital to install a good practice of treatment of chronic Hepatitis B.

REFERENCES

Jonas M, Kelly DA, Mizerski J, et al. Clinical trial of lamivudine in children with chronic hepatitis B. N Engl J Med 2002;346:1706–13.

Lai CL, Lin HJ, Lau HJ, et al. Effect of recombinant a2 IFN with or without prednisolone in Chinese HBsAg carrier children. QJM 1991;78:155–63.

Bortolotti F, Jara P, Barbera C, et al. Long term effect of a interferon in children with chronic hepatitis B. Gut 2000; 46:715–8

Lampertico P, Del Ninno E, Vigano M, et al. Long term suppression of HBeAg negative chronic hepatitis B by 24 months’ interferon therapy. Hepatol 2003;37:756–63.

Kasper D.L., Fauci A.S., Longo D.L, et al: Harrison’s principles of internal medicine. 16th edition. 2005.

Keeffe EB, Dieterich DT, Han SH, Jacobson IM, Martin P, Schiff ER, et al. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States: 2008 update. Clin Gastroenterol Hepatol. Dec 2008;6(12):1315-41; quiz 1286.

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology. May 2009;49(5 Suppl):S72-84

Fattovich G, Stroffolini T, Zagni I, et al. Hepatocellular carcinoma in cirrhosis: incidence and risk factors. Gastroenterology 2004; 127(Suppl 1):S35–S50.

McMahon BJ, Alberts SR, Wainwright RB, et al. Hepatitis B-related sequelae. Prospective study in 1400 hepatitis B surface antigenpositive Alaska native carriers. Arch Intern Med 1990;150:1051– 1054.

Mizokami M, Orito E. Molecular evolution of hepatitis viruses. Intervirology 1999;42:159–165

Tanaka Y, Kurbanov F, Mano S, et al. Molecular tracing of the global hepatitis C virus epidemic predicts regional patterns of hepatocellular carcinoma mortality. Gastroenterology 2006;130: 703–714.

El-Serag HB, Rudolph KL. Hepatocellular carcinoma: epidemiology and molecular carcinogenesis. Gastroenterology 2007;132: 2557–2576.

Chang MH. Hepatitis B virus and cancer prevention. Recent Results Cancer Res 2011;188:75–84

Fattovich G, Bortolotti F, Donato F. Natural history of chronic hepatitis B: special emphasis on disease progression and prognostic factors. J Hepatol 2008;48:335–352

Yang JD, Kim WR, Coelho R, et al. Cirrhosis is present in most patients with hepatitis B and hepatocellular carcinoma. Clin Gastroenterol Hepatol 2011;9:64–70

Yang HI, Lu SN, Liaw YF, et al. Hepatitis B e antigen and the risk of hepatocellular carcinoma. N Engl J Med 2002;347:168–174

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology 2009;49(Suppl): S72–S84.

Chun HM, Roediger MP, Hullsiek KH, et al. Hepatitis B Virus Coinfection Negatively Impacts HIV Outcomes in HIV Seroconverters. The Journal of Infectious Diseases. 2012;205(2):185-193. doi:10.1093/infdis/jir720

McMahon BJ. The natural history of chronic hepatitis B virus infection. Hepatology. 2009;49(5 Suppl):S45-55 2.

Schweitzer A,Horn J, Mikolajczyk RT, Krause G, Ott JJ Estimations of worldwide prevalence of chronic hepatitis B virus infection: a systematic review of data published between 1965 and 2013.

Lancet 2015; 386: 1546–55 3. Ott J, Stevens G, Groeger J, Wiersma S. Global epidemiology of Hepatitis B virus infection: new estimates of age-specific HBsAg seroprevalence and endemicity. Vaccine 2012;30(12): 2212–9 4.

Lukhwareni A, Burnett RJ, Selabe SG, Mzileni MO, Mphahlele MJ. Increased detection of HBV DNA in HBsAg-positive and HBsAgnegative SouthAfrican HIV/AIDS patients enrolling for Highly active antiretroviral therapy at a Tertiary Hospital. J Med Virol 2009;81(3):406–12 5.

Boyles TH, Cohen K. The prevalence of hepatitis B infection in a rural SouthAfrican HIV clinic. S Afr Med J 2011;101(7):470–1 6.

Firnhaber C, Reyneke A, Schulze D, et al. The prevalence of hepatitis B co-infection in a South African urban government HIV clinic. S Afr Med J 2008;98(7):541-4 7.

Burnett RJ, Ngobeni JM, Francois G, et al. Increased exposure to hepatitis B virus infection in HIV-positive South African antenatal women. Int J STD AIDS 2007;18(3):152–6 8.

Andersson MI, Maponga TG, Ijaz S, et al. The epidemiology of hepatitis B virus infection in HIV-infected and HIV-uninfected pregnant women in the Western Cape, South Africa. Vaccine 2013;31(47):5579-84 9.

Puoti M1, Torti C, Bruno R, Filice G, Carosi G. Natural history of chronic hepatitis B in co-infected patients. J Hepatol 2006; 44(1 Suppl):S65-70 10.

Maynard JE. Hepatitis B: Global importance and need for control. Vaccine 1990;8(SUPPL.):S18-S20

Mohd Hanafiah K, Groeger J, Flaxman AD, Wiersma ST. Global epidemiology of hepatitis virus infection: new estimates of age-specific antibody to HCV seroprevalence. Hepatology 2013;57(4):1333-42

Lavanchy D. The global burden of hepatitis C. Liver Int. 2009;29 Suppl 1:74-Ellis LA, Brown D, Conradie JD, Paterson A, Sher R, Millo J, et al. Prevalence of hepatitis C inSouth Africa: detection of anti-HCV in recent and stored serum. J Med Virol 1990;32(4):249- 51

Davis GL. Hepatitis C virus genotypes and quasispecies. Am J Med 1999;107(6B):21S-6S

Messina JP, Humphreys I, Flaxman A, et al. Global distribution and prevalence of hepatitis C virus genotypes. Hepatology. 2015;61(1):77–87

Benova L, Mohamoud YA, Calvert C, AbuRaddad LJ. Vertical transmission of hepatitis C virus: systematic review and meta-analysis. Clin Infect Dis. 2014;59(6):765–73

Floreani A. Hepatitis C and pregnancy. World J Gastroenterol. 2013;19(40):6714–20 Jafari S, Copes R, Baharlou S, Etminan M, Buxton J. Tattooing and the risk of transmission of hepatitis C: a systematic review and metaanalysis. Int J Infect Dis.2010;14(11):E928– E9

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Zein CO, Levy C, Basu A, Zein NN. Chronic hepatitis C and type II diabetes mellitus: a prospective cross-sectional study. Am J Gastroenterol. 2005;100(1):48-5

EASL Recommendations on Treatment of Hepatitis C 2018. J Hepatol 2018; 69(2): 461- 511

Smith JO, Sterling RK. Systematic review: non-invasive methods of fibrosis analysis in chronic hepatitis C. Alimentary pharmacology & therapeutics. 2009;30(6):557-76

Karmochkine M, Carrat F, Dos Santos O, Cacoub P, Raguin G. A case-control study of risk factors for hepatitis C infection in patients with unexplained routes of infection. J Viral Hepat. 2006;13(11):775–82

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Lau GK, Piratvisuth T, Luo KX, Marcellin P, Thongsawat S, et al. (2005) Peginterferon Alfa-2a, lamivudine, and the combination for HBeAgpositive chronic hepatitis B. N Engl J Med 352: 2682-2695. 2.

Conjeevaram HS, Lok AS (2003) Management of chronic hepatitis B. J Hepatol 38 Suppl 1: S90-103.

Karayiannis P (2003) Hepatitis B virus: old, new and future approaches to antiviral treatment. J Antimicrob Chemother 51: 761-785.

Wellington K, Jarvis B (2001) Silymarin: a review of its clinical properties in the management of hepatic disorders. BioDrugs 15: 465-489.

Strader DB, Bacon BR, Lindsay KL, La Brecque DR, Morgan T, et al. (2002) Use of complementary and alternative medicine in patients with liver disease. Am J Gastroenterol 97: 2391-2397.

Eisenberg DM, Davis RB, Ettner SL, Appel S, Wilkey S, et al. (1998) Trends in alternative medicine use in the United States, 1990-1997: results of a follow-up national survey. JAMA 280: 1569-1575.

Flora K, Hahn M, Rosen H, Benner K (1998) Milk thistle (Silybum marianum) for the therapy of liver disease. Am J Gastroenterol 93: 139-143.

Varga Z, Czompa A, Kakuk G, Antus S (2001) Inhibition of the superoxide anion release and hydrogen peroxide formation in PMNLs by flavonolignans. Phytother Res 15: 608-612.

Dehmlow C, Erhard J, de Groot H (1996) Inhibition of Kupffer cell functions as an explanation for the hepatoprotective properties of silibinin. Hepatology 23: 749-754

Wilasrusmee C, Kittur S, Shah G, Siddiqui J, Bruch D, et al. (2002) Immunostimulatory effect of Silybum Marianum (milk thistle) extract. Med Sci Monit 8: BR439-443.

Neumann UP, Biermer M, Eurich D, Neuhaus P, Berg T (2010) Successful prevention of hepatitis C virus (HCV) liver graft reinfection by silibinin mono-therapy. J Hepatol 52: 951-952.

Beinhardt S, Rasoul-Rockenschaub S, Maieron A, PH S-M, Hofer H PF (2012) Intravenous Silibinin-therapy in patients with chronic hepatitis C in the transplant setting. J Hepatology 56: S77.

Pares A, Planas R, Torres M, Caballeria J, Viver JM, et al. (1998) Effects of silymarin in alcoholic patients with cirrhosis of the liver: results of a controlled, double-blind, randomized and multicenter trial. J Hepatol : 615-621.

Sepanlou SG, Safiri S, Bisignano C, et al; GBD 2017 Cirrhosis Collaborators. The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol Hepatol. 2020;5(3):245-266. doi:10.1016/S2468-1253(19) 30349-8

Tapper EB, Parikh ND. Mortality due to cirrhosis and liver cancer in the United States, 1999-2016. BMJ. 2018;362:k2817. doi:10.1136/bmj.k2817

Centers for Disease Control and Prevention. CDC WONDER: about provisional mortality statistics, 2018 through last month. Accessed March 13, 2023. https://wonder.cdc.gov/mcd-icd10-provisional. html

Serper M, Kaplan DE, Taddei TH, Tapper EB, Cohen JB, Mahmud N. Nonselective beta blockers, hepatic decompensation, and mortality in cirrhosis: a national cohort study. Hepatology. 2023;77(2): 489-500. doi:10.1002/hep.32737

Flemming JA, Dewit Y, Mah JM, Saperia J, Groome PA, Booth CM. Incidence of cirrhosis in young birth cohorts in Canada from 1997 to 2016: a retrospective population-based study. Lancet Gastroenterol Hepatol. 2019;4(3):217-226. doi:10. 1016/S2468-1253(18)30339-X

Hirode G, Saab S, Wong RJ. Trends in the burden of chronic liver disease among hospitalized US adults.JAMA Netw Open. 2020;3(4):e201997-e201997. doi:10.1001/jamanetworkopen.2020.1997

Foster C, Baki J, Nikirk S, Williams S, Parikh ND, Tapper EB. Comprehensive health-state utilities in contemporary patients with cirrhosis. Hepatol Commun. 2020;4 (6):852-858. doi:10.1002/hep4. 1512

Asrani SK, Hall L, Hagan M, et al. Trends in chronic liver disease-related hospitalizations: a population-based study. Am J Gastroenterol. 2019;114(1):98-106. doi:10.1038/s41395-018- 0365-4

Tarao K, Nozaki A, Ikeda T, et al. Real impact of liver cirrhosis on the development of hepatocellular carcinoma in various liver diseases-meta-analytic assessment. Cancer Med. 2019;8(3):1054-1065. doi:10.1002/cam4.1998

Carbonell N, Pauwels A, Serfaty L, Fourdan O, Lévy VG, Poupon R. Improved survival after variceal bleeding in patients with cirrhosis over the past two decades. Hepatology. 2004;40(3):652-659. doi: 10.1002/hep.20339

Arvaniti V, et al. Infections in patients with cirrhosis increase mortality four fold and should be used in determining prognosis. Gastroenterology 2010;139:1246-1256.

Berzigott A., et al. Obesity is an independent risk factor for clinical decompensation in patients with cirrhosis. Hepatology 2011;54:555-561.

Dienstag J.L., et al. A prospective study of progression in compensated, histologically advanced chronic hepatitis C. Hepatology 2011;54:396-405.

Durand F, et al. Assessment of the prognosis of cirrhosis: Child-Pugh versus MELD. Journal of Hepatology2005;42.

Franchis de, et al. Non-invasive diagnosis of cirrhosis and the natural history of it complications. Best Practice & Research Clinical Gastroenterology 2007;21(1):3-18.

Friedman SL. Mechanisms of hepatic fibrogenesis. Gastroenterology 2008;134:1655-1669.

Garcia-Tsao G, et al. Management and treatment of patients with cirrhosis and portal hypertension: recommendations from the Department of Veterans Affairs Hepatitis C Resource Center Program and the National Hepatitis C program. The American Journal of Gastroenterology 2009;104(7):1802-1829

Greenbaum L.E. and Wells R.G. The role of stem cells in liver repair and fibrosis. The International Journal of Biochemistry and Cell Biology 2011;43:222-229.

Jiao J, et al. Hepatic fibrosis. Current Opinion in Gastroenterology 2009;25(3):223-229.

Kanwal F, et al. An explicit quality indicator set for measurement of quality of care in patients with cirrhosis. Clinical Gastroenterology and Hepatology 2010;8:709

Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2021; 71: 209–49.

Siegel RL, Miller KD, Fuchs HE, Jemal A. Cancer statistics, 2022. CA Cancer J Clin 2022; 72: 7–33. 3 Petrick JL, Florio AA, Znaor A, et al. International trends in hepatocellular carcinoma incidence, 1978–2012. Int J Cancer 2020; 147: 317–30.

Sun J, Althoff KN, Jing Y, et al. Trends in hepatocellular carcinoma incidence and risk among persons with HIV in the US and Canada, 1996–2015.

JAMA Netw Open 2021; 4: e2037512. 5 Chen T, Liu J, Li Y, Wei S. Burden of disease associated with dietary exposure to aflatoxins in China in 2020. Nutrients 2022; 14: 1027.

Romano A, Angeli P, Piovesan S, et al. Newly diagnosed hepatocellular carcinoma in patients with advanced hepatitis C treated with DAAs: a prospective population study. J Hepatol 2018; 69: 345–52.

Dave S, Park S, Murad MH, et al. Comparative effectiveness of entecavir versus tenofovir for preventing hepatocellular carcinoma in patients with chronic hepatitis B: a systematic review and meta-analysis. Hepatology 2021; 73: 68–78.

Papatheodoridis GV, Idilman R, Dalekos GN, et al. The risk of hepatocellular carcinoma decreases after the first 5 years of entecavir or tenofovir in Caucasians with chronic hepatitis B. Hepatology 2017; 66: 1444–53.

Chiang CJ, Yang YW, You SL, Lai MS, Chen CJ. Thirty-year outcomes of the national hepatitis B immunization program in Taiwan. JAMA 2013; 310: 974–76.

Kanwal F, Kramer JR, Asch SM, Cao Y, Li L, El-Serag HB. Long-term risk of hepatocellular carcinoma in HCV patients treated with direct acting antiviral agents. Hepatology 2020; 71: 44–55.

Kim HY, Lampertico P, Nam JY, et al. An artificial intelligence model to predict hepatocellular carcinoma risk in Korean and Caucasian patients with chronic hepatitis B. J Hepatol 2022; 76: 311–18.

Kim HS, Yu X, Kramer J, et al. Comparative performance of risk prediction models for hepatitis B-related hepatocellular carcinoma in the United States. J Hepatol 2022; 76: 294–301.

Zheng J, Seier K, Gonen M, et al. Utility of serum inflammatory markers for predicting microvascular invasion and survival for patients with hepatocellular carcinoma. Ann Surg Oncol 2017; 24: 3706–14.

Johnson PJ, Dhanaraj S, Berhane S, Bonnett L, Ma YT. The prognostic and diagnostic significance of the neutrophil-tolymphocyte ratio in hepatocellular carcinoma: a prospective controlled study. Br J Cancer 2021; 125: 714–16.

Bruix J, Cheng AL, Meinhardt G, Nakajima K, De Sanctis Y, Llovet J. Prognostic factors and predictors of sorafenib benefit in patients with hepatocellular carcinoma: analysis of two phase III studies. J Hepatol 2017; 67: 999–1008.

Scheiner B, Pomej K, Kirstein MM, et al. Prognosis of patients with hepatocellular carcinoma treated with immunotherapy—development and validation of the CRAFITY score. J Hepatol 2022; 76: 353–63. 87 Vogel A, Martinelli E, ESMO Guidelines Committe. Updated treatment recommendations for hepatocellular carcinoma (HCC) from the ESMO Clinical Practice guidelines. Ann Oncol 2021; 32: 801–05.

Bruix J, Chan SL, Galle PR, Rimassa L, Sangro B. Systemic treatment of hepatocellular carcinoma: an EASL position paper. J Hepatol 2021; 75: 960–74.

The International Liver Cancer Association. ILCA systemic therapy guidance. https://ilca-online.org/wp-content/uploads/2020/06/Systemic-therapy-guidelines-V1.2.pdf (accessed July 26, 2022).

Kudo M, Kawamura Y, Hasegawa K, et al. Management of hepatocellular carcinoma in Japan: JSH consensus statements and recommendations 2021 update. Liver Cancer 2021; 10: 181–223.

Chen LT, Martinelli E, Cheng AL, et al. Pan-Asian adapted ESMO clinical practice guidelines for the management of patients with intermediate and advanced/relapsed hepatocellular carcinoma: a TOS-ESMO initiative endorsed by CSCO, ISMPO, JSMO, KSMO, MOS and SSO. Ann Oncol 2020; 31: 334–51.

Reig M, Forner A, Rimola J, et al. BCLC strategy for prognosis prediction and treatment recommendation: the 2022 update. J Hepatol 2021; 76: 681–93.

Lang H, Sotiropoulos GC, Dömland M, et al. Liver resection for hepatocellular carcinoma in non-cirrhotic liver without underlying viral hepatitis. Br J Surg 2005; 92: 198–202.

Viganò L, Conci S, Cescon M, et al. Liver resection for hepatocellular carcinoma in patients with metabolic syndrome: a multicenter matched analysis with HCV-related HCC. J Hepatol 2015; 63: 93–101.

Kabir T, Tan ZZ, Syn NL, et al. Laparoscopic versus open resection of hepatocellular carcinoma in patients with cirrhosis: meta analysis. Br J Surg 2021; 109: 21–29. 96 Ivanics T, Claasen MP, Patel MS, et al. Long-term outcomes of laparoscopic liver resection for hepatocellular carcinoma: a propensity score matched analysis of a high-volume North American center. Surgery 2021; 171: 982–91.

Berzigotti A, Reig M, Abraldes JG, Bosch J, Bruix J. Portal hypertension and the outcome of surgery for

hepatocellular carcinoma in compensated cirrhosis: a systematic review and meta - analysis. Hepatology 2015; 61: 526–36.

Berardi G, Morise Z, Sposito C, et al. Development of a nomogram to predict outcome after liver resection for hepatocellular carcinoma in Child-Pugh B cirrhosis. J Hepatol 2020; 72: 75–84.

Azoulay D, Ramos E, Casellas-Robert M, et al. Liver resection for hepatocellular carcinoma in patients with clinically significant portal hypertension. JHEP Rep 2020; 3: 100190.

Citterio D, Facciorusso A, Sposito C, Rota R, Bhoori S, Mazzaferro V. Hierarchic interaction of factors associated with liver decompensation after resection for hepatocellular carcinoma. JAMA Surg 2016; 151: 846–53.

Serenari M, Han KH, Ravaioli F, et al. A nomogram based on liver stiffness predicts postoperative complications in patients with hepatocellular carcinoma. J Hepatol 2020; 73: 855–62.

Allaire M, Goumard C, Lim C, Le Cleach A, Wagner M, Scatton O. New frontiers in liver resection for hepatocellular carcinoma. JHEP Rep 2020; 2: 100134.

Marasco G, Alemanni LV, Colecchia A, et al. Prognostic value of the albumin-bilirubin grade for the prediction of post-hepatectomy liver failure: a systematic review and meta-analysis. J Clin Med 2021; 10: 2011.

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Olivier Lieuga ,Tatiana Jiengoué, and Augustine Nji Asakizi. (2026). Prevalence of Liver Cirrhosis on patients with Chronic Hepatitis B during treatment by Tenofovir Disoproxil 300 mg only at The NGaoundere Protestant Hospital in Adamawa Region Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004015. www.iqresearchjournal.com

ANNEXES

Annex I

Hepatotrophic Viral Type	Genetic	Classification Group		
	RNA	DNA	Enteric	Parenteral
HAV	Yes	No	Yes	No
HBV	No	Yes	No	Yes
HCV	Yes	No	No	Yes
HDV	Yes	No	No	Yes
HEV	Yes	No	Yes	No

Annex II — Table 1. Results of Patient Characteristics

Chronic Hepatitis B Patient Characteristics	Patients	
	n	%
Nationalities		
Cameroonians	20	100
Age (Years Old)		
18 – 35	11	55,00
36 - 55	7	35,00
55 – Over	2	10,00
Marital Status		
Single	9	45,00
Married	8	40,00
Divorced	3	15,00

Annex III — Table 2. Results of Chronic Hepatitis B Clinical Manifestations

Clinical Manifestations	Abdomen Distention		Ascites		Jaundice		Legs Swelling (Godet)		Hepatosplenomegaly	
	n	%	n	%	n	%	n	%	n	%
Patient	20	100	18	90,00	16	80,00	11	55,00	17	85,00

Annex IV — Table 3. Results of Exams and Tests done to confirm Chronic Hepatitis B Patients

Exams and Tests Done	Elisa HBV		Viral Load HBV		5 HBV Biomarkers		Hepatitis Delta Serology		Ultrasounds		HBV Genotype		LFTs	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%
Patient	20	100	9	45,00	20	100	9	45,00	14	70,0	0	0	0	0

Annex V — Table 5. Results of Chronic Hepatitis B Treatment

Treatment Received by HBV Patients	Tenofovir Disoproxil 300 mg		Tenofovir Alafenamide 25 mg		Peginterferon alfa 2 a		Interferon alfa 2 b		Entecavir and/or Adefovir		Lamivudine	
	n	%	n	%	n	%	n	%	n	%	n	%
Patient	20	100	0	0	0	0	0	0	0	0	0	0

Annex VI — Table 6. Results of Hepatitis B Complications analyzed on 100 Patients

Complications of Chronic Hepatitis B	Liver Cirrhosis		Hepatocellular Carcinoma		Fulminant Hepatitis		Pulmonary Embolism		Cardiac Liver		Digestive Bleeding	
	n	%	n	%	n	%	n	%	n	%	n	%
Patient	17	85	0	0	4	20	7	35	0	0	0	0

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Prevalence of Liver Cirrhosis on patients with Chronic Hepatitis C during treatment by Velso 400/100 mg only at The NGaoundere Protestant Hospital in Adamawa Region Cameroon

Olivier Lieuga[a] ,Tatiana Jiengoué[a], and Augustine Nji Asakizi[a]

Affiliations

p. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Olivier Lieuga

Email: *olivierlieuga@kesmondsuniversity.org*

Paper ID: IQRJ-V05I02-26005016

ABSTRACT

Introduction: Globally, 71 million people are viremia for HCV infection, about 15 to 25 per cent of infected individuals spontaneously clear the virus, within six months of infection. However, the remaining 75 to 55 per cent will develop chronic HCV. Of those chronically infected, the risk of cirrhosis is 15 to 30 per cent within 20 years with a one to four per cent per annum risk of hepatocellular carcinoma.

Methods: It is descriptive cross – sectional study prevalence of liver cirrhosis done on 35 patients tested positives with HCV confirmed by the following exams such as ELISA HCV, Viral Load HCV among 100 patients presented some clinical manifestations of Chronic Hepatitis C such as jaundice, abdomen pain hepatosplenomegaly, ascites, hepatic encephalopathy, legs swelling (godet) and abdomen distention at the NGaoundere Protestant Hospital in Adamawa Region Cameroon from June 2023 to September 2023 after receiving Ethical Clearance and Research Authorization from The Health Work of the Evangelical Lutheran Church of Cameroon (HWELCC) Chairman.

Results: The prevalence of chronic hepatitis C on 100 patients was 35% and Out to 35 patients tested positive of chronic hepatitis C, the prevalence of Liver Cirrhosis were 82,85%.

Conclusions: This study showed the high level of Liver Cirrhosis prevalence to the patients tested positive with chronic hepatitis C despite taking Velso 400/100 mg(Sofosbuvir 400 mg and velpastavir100mg). To prevent cirrhosis related to hepatitis C, direct oral antiviral therapy (DOAT) is crucial, curing more than 95% of cases. It is essential to eliminate alcohol, adopt a balanced diet, avoid hepatotoxic medications, and get vaccinated against hepatitis A and B

Keywords: *Hepatocellular Carcinoma, Liver Cirrhosis ,Hepatitis B, Hepatitis C*

1. INTRODUCTION

Hepatitis C virus, which before its identification was labelled "non – A, non – B Hepatitis" is a linear, single – stranded enveloped RNA virus belonging to the flavivinus family, *Mohd Hanafiah K and al (2013).* There are six clinically relevant HCV genotypes and more than 80 subtypes. Genotype prevalence varies according to geographic region and route of acquisition. HCV remains viable on environmental surfaces at room temperature for at least 16 hours, but typically no longer than four days, 13, 14 and transmission occurs via parenteral and non-parenteral routes, *Ellis LA, Brown D, Conradie JD and al (1990)*. Then, to 40 per cent of HCV infected individuals have no clear identifiable risk factor.HCV infection is usually acquired through infected syringes and needles, and transfusion of infected blood, *David GI (1999)*. Sexual transmission of HCV occurs infrequently in heterosexual couples. It is reported to be more common in HIV – positive persons, particularly in MSM.

The risk of transmission of HCV from a mother to her child occurs in 4 – 8% of births to women with HCV infection, and in 10,8 – 25% of births to women with HIV and HCV co - infection, *Choo QL, Richman KH, Han JH and al (1991).* HCV causes both acute and chronic hepatitis. Acute hepatitis is often clinically mild and marked by fluctuating elevations of serum aminotransferase levels > 50% likelihood of chronicity, leading to cirrhosis in > 20%, *Scheibe A, Young K, Moses L and al (1991).* Chronic infection with HCV is usually clinically silent, and is only very rarely associated with life – threatening disease. Spontaneous clearance of acute HCV infection occurs within six months of infection in 15 – 45% of infected individuals in the absence of treatment.

2. RELATED WORKS

Many people with HCV do not have symptoms and do not know there are infected, *Amedei B., et al (2011)*. The incubation period ranges from 5 to 10 weeks (mean 7 weeks), acute hepatitis c commonly has a mild clinical course, and most are anicteric. Those with symptoms are more likely to clear the virus, *David GE (1999).* Chronic HCV infection develops in 70 to 80% of patients, who then have a significant risk of developing cirrhosis and chronic liver failure. Fatigue is the most frequent complaint followed by some vague manifestations like nausea, anorexia, myalgia, arthralgia and cognitive impairment, *Balagopal A, et al (2010)*

According to *Scheibe A, Young K, Moses L and al (2013),* chronic HCV has been associated with several extrahepatic manifestations such as autoimmune disorders like (Sjogren syndrome, Cryoglobulinaemia and polyarthritis nodosa), porphyria cutanea tarda, lymphoproliferative diseases like (B-cell non-Hodgkin's lymphoma), insulin resistance disturbances like (progressive insulin resistance impaired fasting glucose (IFG), and/or type 2 diabetes mellitus (DM), are higher in chronic patients (50%), than in the general population and neurocognitive infection.

HCV infection is usually acquired through infected syringes and needles, and transfusion of infected blood, *David GI (1999)*. Sexual transmission of HCV occurs infrequently in heterosexual couples. It is reported to be more common in HIV – positive persons, particularly in MSM. The risk of transmission of HCV from a mother to her child occurs in 4 – 8% of births to women with HCV infection, and in 10,8 – 25% of births to women with HIV and HCV co - infection, *Choo QL, Richman KH, Han JH and al (1991).* HCV causes both acute and chronic hepatitis. Acute hepatitis is often clinically mild and marked by fluctuating elevations of serum aminotransferase levels > 50% likelihood of chronicity, leading to cirrhosis in > 20%, *Scheibe A, Young K, Moses L and al (1991).* Chronic

infection with HCV is usually clinically silent, and is only very rarely associated with life – threatening disease. Spontaneous clearance of acute HCV infection occurs within six months of infection in 15 – 45% of infected individuals in the absence of treatment.

Many people with HCV do not have symptoms and do not know there are infected, *Amedei B., et al (2011)*. The incubation period ranges from 5 to 10 weeks (mean 7 weeks), acute hepatitis c commonly has a mild clinical course, and most are anicteric. Those with symptoms are more likely to clear the virus, *David GE (1999).* Chronic HCV infection develops in 70 to 80% of patients, who then have a significant risk of developing cirrhosis and chronic liver failure. Fatigue is the most frequent complaint followed by some vague manifestations like nausea, anorexia, myalgia, arthralgia and cognitive impairment, *Balagopal A, et al (2010)*

According to *Scheibe A, Young K, Moses L and al (2013),* chronic HCV has been associated with several extrahepatic manifestations such as autoimmune disorders like (Sjogren syndrome, Cryoglobulinaemia and polyarthritis nodosa), porphyria cutanea tarda, lymphoproliferative diseases like (B-cell non-Hodgkin's lymphoma), insulin resistance disturbances like (progressive insulin resistance impaired fasting glucose (IFG), and/or type 2 diabetes mellitus (DM), are higher in chronic patients (50%), than in the general population and neurocognitive infection.

Serological tests for HCV either identify viral antibody (ELISA) or constitute molecular tests to detect the virus (HCV-RNA using polymerase chain reaction, termed PCR) and classify its genotype, *Arora S., et al (2010).* The presence of anti-HCV antibody suggests viral exposure and is not a marker of immunity. The majority of patients exposed to HCV then become carriers of the virus. The presence of anti-HCV antibody however does not necessarily indicate chronic infection, *Balagropal A, et al (2016).* The diagnosis of chronic hepatitis C infection requires detecting the virus with the PCR test. The antibody test (ELISA, enzyme-linked immunosorbent assay) is the primary screening test. It identifies antibodies to the non-structural as well as the structural epitopes of the virus, *Dessouki O., et al (2010)*.

Being both sensitive and specific, this ELIZA test is very useful in identifying patients who had exposure to the hepatitis C virus. False-positive ELISA testing occurs with hypergammaglobulinemia. False negatives occur soon after acute hepatitis C onsets, and in association with immunosuppression or renal failure, *Ahlenstiel G. et al (2011).* HCV RNA PCR test is thus essential to clinch the diagnosis and assess the viral load. This information guides the management of chronic hepatitis C infection including the response to treatment. Genotypes assist in understanding the epidemiology, the natural course of HCV and importantly, the response to therapy, *Messina JP, Humphreys et al (2015)*

Chronic HCV becomes difficult to treat. The goal of treatment in chronic HCV hepatitis seeks to eradicate the virus, best predicted by a sustained virologic response: absence of HCV-RNA 6 months after stopping treatment, *David GE (1999)*. A combination of antiviral agents is necessary: PEGylated interferon plus ribavirin. Genotype 1, being the most difficult HCV to treat, requires the addition of a direct antiviral agent: a protease inhibitor like telaprevir or boceprevir, boosting the response rate to 70-80%, *Scheibe A, Young K, Moses L and al (2013)* These antiviral therapies are undergoing further evaluation. Chronic hepatitis C requires surveillance for

progression to cirrhosis, liver failure and the development of hepatocellular hepatoma. About Prevention There is no vaccine or specific immunoglobulin available for HCV. The risk of sexual transmission is extremely low. In stable, monogamous relationships, the use of condoms is unnecessary, *Mohd Hanafiah K et al (2013)*. Chronic hepatitis C infection remains a significant health issue and may lead to the development of liver cirrhosis, liver failure and hepatocellular carcinoma. The incidence of these complications is expected to continue to rise and peak by 2020, *Tapper EB, Parikh ND et al (2016)*. However, H is curable, and viral eradication has been shown to reduce these complications and improve quality of life. Hepatitis C virus remain underdiagnosed; hence, all patients with risk factors or a high clinical index of suspicion should be screened and referred for treatment, *Flemming JA, Dewit Y, Mah JM, Saperia J, Groome PA, Booth et al (2019)*.

The addition of telaprevir and boceprevir for genotype-1 infection, which have recently been approved in Australia, has significantly increased cure rates and will hopefully improve treatment uptake, *Hirode G, Saab S, and Wong RJ (2020).* The HCV treatment landscape is rapidly evolving, with highly efficacious interferon-free all-oral therapies on the horizon. HCV is the leading cause of chronic liver disease and cirrhosis and is the main cause of liver transplantation in the Western world, *Arsani SK, Hall L, Hagan M et al (2019).* Although the total number of HCV-infected individuals is estimated to be stable or decline in the future, an increase in liver cirrhosis, liver cancer, hepatic decompensation and liver-related deaths is expected in the coming years, *Tarao K, Nozaki A, Ikeda T et al (2019).* This assessment is underlined by the World Health Organization (WHO) statement that the burden of HCV disease has been largely ignored as a health priority, and the organization has developed the first-ever global health sector strategy for addressing the viral hepatitis pandemic.Untreated chronic hepatitis C can lead to severe, long-term complications, primarily cirrhosis (liver scarring), which affects up to 1 in 3 people within 20–30 years. Other major, life-threatening complications include liver failure and liver cancer (hepatocellular carcinoma), *Choo QL, Richman KH, Han JH and al (1991).* Early diagnosis and treatment are crucial to prevent these outcomes. Chronic inflammation causes scarred tissue to replace healthy liver tissue, which can take decades to develop.

As cirrhosis progresses, the liver loses its ability to function, *Mohd Hanafiah K and al (2013),* leading to potential complications such as fluid buildup in the abdomen (ascites) or legs (oedema). Hepatitis C is a leading cause of hepatocellular carcinoma, particularly in those who have already developed cirrhosis. Increased pressure in the vein that carries blood from the digestive organs to the liver, which can lead to bleeding. Hepatitis C can also cause serious health issues in other parts of the body, often due to the immune system reacting to the virus, *Colin JF, Cazals-Hatem D, Loriot MA, et al (1999).* Chronic hepatitis C is a serious infection that can lead to long-term health issues, including cirrhosis (liver scarring), liver failure and hepatocellular carcinoma (liver cancer). It is a systemic disease causing extrahepatic complications like cryoglobulinemia, diabetes, cardiovascular disease, and chronic kidney disease, *Thio CL, et al,.. (2002).*

Chronic hepatitis C infection remains a significant health issue and may lead to the development of liver cirrhosis, liver failure and

hepatocellular carcinoma. The incidence of these complications is expected to continue to rise and peak by 2020, *Tapper EB, Parikh ND et al (2016)*. However, H is curable, and viral eradication has been shown to reduce these complications and improve quality of life. Hepatitis C virus remain underdiagnosed; hence, all patients with risk factors or a high clinical index of suspicion should be screened and referred for treatment, *Flemming JA, Dewit Y, Mah JM, Saperia J, Groome PA, Booth et al (2019)*. The addition of telaprevir and boceprevir for genotype-1 infection, which have recently been approved in Australia, has significantly increased cure rates and will hopefully improve treatment uptake, *Hirode G, Saab S, and Wong RJ (2020).* The HCV treatment landscape is rapidly evolving, with highly efficacious interferon-free all-oral therapies on the horizon. HCV is the leading cause of chronic liver disease and cirrhosis and is the main cause of liver transplantation in the Western world, *Arsani SK, Hall L, Hagan M et al (2019).* Although the total number of HCV-infected individuals is estimated to be stable or decline in the future, an increase in liver cirrhosis, liver cancer, hepatic decompensation and liver-related deaths is expected in the coming years, *Tarao K, Nozaki A, Ikeda T et al (2019).* This assessment is underlined by the World Health Organization (WHO) statement that the burden of HCV disease has been largely ignored as a health priority, and the organization has developed the first-ever global health sector strategy for addressing the viral hepatitis pandemic.

3. MATERIALS & METHODS

It is descriptive cross – sectional study prevalence of liver cirrhosis done on 35 patients tested positives with HCV confirmed by the following exams such as ELISA HCV, Viral Load HCV among 100 patients presented some clinical manifestations of Chronic Hepatitis C such as jaundice, abdomen pain, hepatosplenomegaly, ascites, hepatic encephalopathy, legs swelling (godet) and abdomen distention at the NGaoundere Protestant Hospital in Adamawa Region Cameroon from June 2023 to September 2023 after receiving Ethical Clearance and Research Authorization from The Health Work of the Evangelical Lutheran Church of Cameroon (HWELCC) Chairman. Convenience sampling was done. The analysis included management of risk factors, clinical manifestations, diagnostic methods, treatment and complications of Chronic Hepatitis C.

Convenience sampling was done, all the cases of elective Chronic Hepatitis B admitted during the study period were included. Age > 20 residents in Cameroon. The simple size was 25 Hepatitis calculated was calculated using the formula $n = (Z^2 \times p \times q) / e^2$ where n = minimum sample size, z= confidence interval, p= q= 1 – p and e= margin of error, 5%.

The Adamawa region is a constituent region of the republic of Cameroon, it borders the Centre and East regions to the south, the Northwest and West regions to the Southwest, Nigeria to the West, the Central African Republic to the East, and the North region to the North. This mountainous area forms the barrier between Cameroon's forested South and Savana North. At almost 64,000 km2 in land area, the Adamawa is the third largest of Cameroon's ten regions.

The East region on its part occupies the South portion of the Republic of Cameroon, it is bordered to the East by the Central African Republic, to the South by Congo, to the North by the Adamawa region and to the West by the Centre and South regions. With 109,002 km2 of

territory, it is the largest region in the nation as well as the most sparsely populated.

The Health Work of the Evangelical Lutheran Church of Cameroon (HWELCC) possesses three large hospitals namely: The Ngaoundere Protestant Hospital (NPH), the Ngaoubela Protestant Hospital (NGBELAPH) and the Garoua Boulai Protestant Hospital (GBLAIPH) and several health centres and clinics, we have marked our passage particularly at the Ngaoundere Protestant Hospital.

The data for this study were collected using structured questionnaires, entered and analyzed in Microsoft Excel 2016, confidence interval was calculated along with frequency and percentages for binary data. The inclusion criteria included only patients (adults) tested positive with Liver Cirrhosis, aged from 18 years old..

4. RESULTS & DISCUSSION

To assess the prevalence of Liver Cirrhosis to these HCV Patients, we collected and analyzed data on patient's characteristics, clinical manifestations, diagnostic methods used, types of treatment given and some complications occurred before, during and after treatment

[See Annex — Table 1. Results of Patient Characteristics]

[See Annex — Table 2. Results of Chronic Hepatitis C Clinical Manifestations]

[See Annex — Table 3. Results of Exams and Tests done to confirm Chronic Hepatitis C]

[See Annex — Table 4. Results of Chronic Hepatitis C Treatment]

[See Annex — Table 5. Results of Hepatitis B Complications analyzed on 100 Patients]

82,85% of patients have developed liver cirrhosis despite taking Velso (sofosbuvir 400mg/velpastavir100mg), 11,42% have developed Hepatocellular Carcinoma and 20% developed fulminant hepatitis. 65,72% of patients didn't take viral load HCV test, 0% for genotype HCV and 68,58% for liver function tests. These shortcomings observed in the diagnosis and treatment of hepatitis C in patients at the Protestant Hospital of N'Gaoundere led to numerous complications, including the development of cirrhosis and liver cancer. This study aimed to evaluate the prevalence of liver cirrhosis in patients who tested positive for hepatitis C and were being treated with Velso 400/100 mg. Based on clinical data, the prevalence of liver cirrhosis (both compensated and decompensated) in patients with chronic hepatitis C (CHC) treated with sofosbuvir/velpatasvir 400/100 mg (such as Velso) varies across studies, generally ranging from approximately 28% to over 50% in real-world cohort studies focusing on patients with advanced liver disease, Chen CJ et al,..(2019). Despite the high sustained virologic response (SVR) rates of DAAs, a small number of patients does not eradicate the virus, and these patients represent a challenge, Da BL et al,..(2021). The Sofosbuvir (SOF)/Velpatasvir (VEL) and a second-generation HCV-protease inhibitor, Voxilaprevir (VOX), is a pan-genotypic DAAs combination and the only combination approved for treatment-experienced patients, including those who received non-structural-5A inhibitors. Real-world experiences are still needed for this combination, El Kassas ME et al,..(2000). Unfortunately, current salvage therapies are not accessible in many regions and there are limited data available about the efficacy of different retreatment regimens.

5. CONCLUSION

Treatment failure for hepatitis C, although rare (over 95% cure rate with direct-acting antivirals - DAAs), is usually manifested by a virological

drop within 6 months of stopping medication, when viral RNA reappears. Causes include viral resistance, poor treatment adherence, or host-related factors.

To prevent cirrhosis related to hepatitis C, direct oral antiviral therapy (DOAT) is crucial, curing more than 95% of cases. It is essential to eliminate alcohol, adopt a balanced diet, avoid hepatotoxic medications, and get vaccinated against hepatitis A and B.

REFERENCES

Jonas M, Kelly DA, Mizerski J, et al. Clinical trial of lamivudine in children with chronic hepatitis B. N Engl J Med 2002;346:1706–13.

Lai CL, Lin HJ, Lau HJ, et al. Effect of recombinant a2 IFN with or without prednisolone in Chinese HBsAg carrier children. QJM 1991;78:155–63.

Bortolotti F, Jara P, Barbera C, et al. Long term effect of a interferon in children with chronic hepatitis B. Gut 2000; 46:715–8

Lampertico P, Del Ninno E, Vigano M, et al. Long term suppression of HBeAg negative chronic hepatitis B by 24 months’ interferon therapy. Hepatol 2003;37:756–63.

Kasper D.L., Fauci A.S., Longo D.L, et al: Harrison’s principles of internal medicine. 16th edition. 2005.

Keeffe EB, Dieterich DT, Han SH, Jacobson IM, Martin P, Schiff ER, et al. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States: 2008 update. Clin Gastroenterol Hepatol. Dec 2008;6(12):1315-41; quiz 1286.

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology. May 2009;49(5 Suppl):S72-84

Fattovich G, Stroffolini T, Zagni I, et al. Hepatocellular carcinoma in cirrhosis: incidence and risk factors. Gastroenterology 2004; 127(Suppl 1):S35–S50.

McMahon BJ, Alberts SR, Wainwright RB, et al. Hepatitis B-related sequelae. Prospective study in 1400 hepatitis B surface antigenpositive Alaska native carriers. Arch Intern Med 1990;150:1051– 1054.

Mizokami M, Orito E. Molecular evolution of hepatitis viruses. Intervirology 1999;42:159–165

Tanaka Y, Kurbanov F, Mano S, et al. Molecular tracing of the global hepatitis C virus epidemic predicts regional patterns of hepatocellular carcinoma mortality. Gastroenterology 2006;130: 703–714.

El-Serag HB, Rudolph KL. Hepatocellular carcinoma: epidemiology and molecular carcinogenesis. Gastroenterology 2007;132: 2557–2576.

Chang MH. Hepatitis B virus and cancer prevention. Recent Results Cancer Res 2011;188:75–84

Fattovich G, Bortolotti F, Donato F. Natural history of chronic hepatitis B: special emphasis on disease progression and prognostic factors. J Hepatol 2008;48:335–352

Yang JD, Kim WR, Coelho R, et al. Cirrhosis is present in most patients with hepatitis B and hepatocellular carcinoma. Clin Gastroenterol Hepatol 2011;9:64–70

Yang HI, Lu SN, Liaw YF, et al. Hepatitis B e antigen and the risk of hepatocellular carcinoma. N Engl J Med 2002;347:168–174

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology 2009;49(Suppl): S72–S84.

Chun HM, Roediger MP, Hullsiek KH, et al. Hepatitis B Virus Coinfection Negatively Impacts HIV Outcomes in HIV Seroconverters. The Journal of Infectious Diseases. 2012;205(2):185-193. doi:10.1093/infdis/jir720

McMahon BJ. The natural history of chronic hepatitis B virus infection. Hepatology. 2009;49(5 Suppl):S45-55 2.

Schweitzer A,Horn J, Mikolajczyk RT, Krause G, Ott JJ Estimations of worldwide prevalence of chronic hepatitis B virus infection: a systematic review of data published between 1965 and 2013.

Lancet 2015; 386: 1546–55 3. Ott J, Stevens G, Groeger J, Wiersma S. Global epidemiology of Hepatitis B virus infection: new estimates of age-specific HBsAg seroprevalence and endemicity. Vaccine 2012;30(12): 2212–9 4.

Lukhwareni A, Burnett RJ, Selabe SG, Mzileni MO, Mphahlele MJ. Increased detection of HBV DNA in HBsAg-positive and HBsAgnegative SouthAfrican HIV/AIDS patients enrolling for Highly active antiretroviral therapy at a Tertiary Hospital. J Med Virol 2009;81(3):406–12 5.

Boyles TH, Cohen K. The prevalence of hepatitis B infection in a rural SouthAfrican HIV clinic. S Afr Med J 2011;101(7):470–1 6.

Firnhaber C, Reyneke A, Schulze D, et al. The prevalence of hepatitis B co-infection in a South African urban government HIV clinic. S Afr Med J 2008;98(7):541-4 7.

Burnett RJ, Ngobeni JM, Francois G, et al. Increased exposure to hepatitis B virus infection in HIV-positive South African antenatal women. Int J STD AIDS 2007;18(3):152–6 8.

Andersson MI, Maponga TG, Ijaz S, et al. The epidemiology of hepatitis B virus infection in HIV-infected and HIV-uninfected pregnant women in the Western Cape, South Africa. Vaccine 2013;31(47):5579-84 9.

Puoti M1, Torti C, Bruno R, Filice G, Carosi G. Natural history of chronic hepatitis B in co-infected patients. J Hepatol 2006; 44(1 Suppl):S65-70 10.

Maynard JE. Hepatitis B: Global importance and need for control. Vaccine 1990;8(SUPPL.):S18-S20

Mohd Hanafiah K, Groeger J, Flaxman AD, Wiersma ST. Global epidemiology of hepatitis virus infection:

new estimates of age-specific antibody to HCV seroprevalence. Hepatology 2013;57(4):1333-42

Lavanchy D. The global burden of hepatitis C. Liver Int. 2009;29 Suppl 1:74-Ellis LA, Brown D, Conradie JD, Paterson A, Sher R, Millo J, et al. Prevalence of hepatitis C inSouth Africa: detection of anti-HCV in recent and stored serum. J Med Virol 1990;32(4):249- 51

Davis GL. Hepatitis C virus genotypes and quasispecies. Am J Med 1999;107(6B):21S-6S

Messina JP, Humphreys I, Flaxman A, et al. Global distribution and prevalence of hepatitis C virus genotypes. Hepatology. 2015;61(1):77–87

Benova L, Mohamoud YA, Calvert C, AbuRaddad LJ. Vertical transmission of hepatitis C virus: systematic review and meta-analysis. Clin Infect Dis. 2014;59(6):765–73

Floreani A. Hepatitis C and pregnancy. World J Gastroenterol. 2013;19(40):6714–20 Jafari S, Copes R, Baharlou S, Etminan M, Buxton J. Tattooing and the risk of transmission of hepatitis C: a systematic review and metaanalysis. Int J Infect Dis.2010;14(11):E928– E9

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Zein CO, Levy C, Basu A, Zein NN. Chronic hepatitis C and type II diabetes mellitus: a prospective cross-sectional study. Am J Gastroenterol. 2005;100(1):48-5

EASL Recommendations on Treatment of Hepatitis C 2018. J Hepatol 2018; 69(2): 461- 511

Smith JO, Sterling RK. Systematic review: non-invasive methods of fibrosis analysis in chronic hepatitis C. Alimentary pharmacology & therapeutics. 2009;30(6):557-76

Karmochkine M, Carrat F, Dos Santos O, Cacoub P, Raguin G. A case-control study of risk factors for hepatitis C infection in patients with unexplained routes of infection. J Viral Hepat. 2006;13(11):775–82

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Lau GK, Piratvisuth T, Luo KX, Marcellin P, Thongsawat S, et al. (2005) Peginterferon Alfa-2a, lamivudine, and the combination for HBeAgpositive chronic hepatitis B. N Engl J Med 352: 2682-2695. 2.

Conjeevaram HS, Lok AS (2003) Management of chronic hepatitis B. J Hepatol 38 Suppl 1: S90-103.

Karayiannis P (2003) Hepatitis B virus: old, new and future approaches to antiviral treatment. J Antimicrob Chemother 51: 761-785.

Wellington K, Jarvis B (2001) Silymarin: a review of its clinical properties in the management of hepatic disorders. BioDrugs 15: 465-489.

Strader DB, Bacon BR, Lindsay KL, La Brecque DR, Morgan T, et al. (2002) Use of complementary and alternative medicine in patients with liver disease. Am J Gastroenterol 97: 2391-2397.

Eisenberg DM, Davis RB, Ettner SL, Appel S, Wilkey S, et al. (1998) Trends in alternative medicine use in the United States, 1990-1997: results of a follow-up national survey. JAMA 280: 1569-1575.

Flora K, Hahn M, Rosen H, Benner K (1998) Milk thistle (Silybum marianum) for the therapy of liver disease. Am J Gastroenterol 93: 139-143.

Varga Z, Czompa A, Kakuk G, Antus S (2001) Inhibition of the superoxide anion release and hydrogen peroxide formation in PMNLs by flavonolignans. Phytother Res 15: 608-612.

Dehmlow C, Erhard J, de Groot H (1996) Inhibition of Kupffer cell functions as an explanation for the hepatoprotective properties of silibinin. Hepatology 23: 749-754

Wilasrusmee C, Kittur S, Shah G, Siddiqui J, Bruch D, et al. (2002) Immunostimulatory effect of Silybum Marianum (milk thistle) extract. Med Sci Monit 8: BR439-443.

Neumann UP, Biermer M, Eurich D, Neuhaus P, Berg T (2010) Successful prevention of hepatitis C virus (HCV) liver graft reinfection by silibinin mono-therapy. J Hepatol 52: 951-952.

Beinhardt S, Rasoul-Rockenschaub S, Maieron A, PH S-M, Hofer H PF (2012) Intravenous Silibinin-therapy in patients with chronic hepatitis C in the transplant setting. J Hepatology 56: S77.

Pares A, Planas R, Torres M, Caballeria J, Viver JM, et al. (1998) Effects of silymarin in alcoholic patients with cirrhosis of the liver: results of a controlled, double-blind, randomized and multicenter trial. J Hepatol : 615-621.

Sepanlou SG, Safiri S, Bisignano C, et al; GBD 2017 Cirrhosis Collaborators. The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol Hepatol. 2020;5(3):245-266. doi:10.1016/S2468-1253(19) 30349-8

Tapper EB, Parikh ND. Mortality due to cirrhosis and liver cancer in the United States, 1999-2016. BMJ. 2018;362:k2817. doi:10.1136/bmj.k2817

Centers for Disease Control and Prevention. CDC WONDER: about provisional mortality statistics, 2018 through last month. Accessed March 13, 2023. https://wonder.cdc.gov/mcd-icd10-provisional. html

Serper M, Kaplan DE, Taddei TH, Tapper EB, Cohen JB, Mahmud N. Nonselective beta blockers, hepatic decompensation, and mortality in cirrhosis: a national cohort study. Hepatology. 2023;77(2): 489-500. doi:10.1002/hep.32737

Flemming JA, Dewit Y, Mah JM, Saperia J, Groome PA, Booth CM. Incidence of cirrhosis in young birth cohorts in Canada from 1997 to 2016: a retrospective population-based study. Lancet

Gastroenterol Hepatol. 2019;4(3):217-226. doi:10. 1016/S2468-1253(18)30339-X

Hirode G, Saab S, Wong RJ. Trends in the burden of chronic liver disease among hospitalized US adults.JAMA Netw Open. 2020;3(4):e201997-e201997. doi:10.1001/jamanetworkopen.2020.1997

Foster C, Baki J, Nikirk S, Williams S, Parikh ND, Tapper EB. Comprehensive health-state utilities in contemporary patients with cirrhosis. Hepatol Commun. 2020;4 (6):852-858. doi:10.1002/hep4. 1512

Asrani SK, Hall L, Hagan M, et al. Trends in chronic liver disease-related hospitalizations: a population-based study. Am J Gastroenterol. 2019;114(1):98-106. doi:10.1038/s41395-018- 0365-4

Tarao K, Nozaki A, Ikeda T, et al. Real impact of liver cirrhosis on the development of hepatocellular carcinoma in various liver diseases-meta-analytic assessment. Cancer Med. 2019;8(3):1054-1065. doi:10.1002/cam4.1998

Carbonell N, Pauwels A, Serfaty L, Fourdan O, Lévy VG, Poupon R. Improved survival after variceal bleeding in patients with cirrhosis over the past two decades. Hepatology. 2004;40(3):652-659. doi: 10.1002/hep.20339

Arvaniti V, et al. Infections in patients with cirrhosis increase mortality four fold and should be used in determining prognosis. Gastroenterology 2010;139:1246-1256.

Berzigott A., et al. Obesity is an independent risk factor for clinical decompensation in patients with cirrhosis. Hepatology 2011;54:555-561.

Dienstag J.L., et al. A prospective study of progression in compensated, histologically advanced chronic hepatitis C. Hepatology 2011;54:396-405.

Durand F, et al. Assessment of the prognosis of cirrhosis: Child-Pugh versus MELD. Journal of Hepatology2005;42.

Franchis de, et al. Non-invasive diagnosis of cirrhosis and the natural history of it complications. Best Practice & Research Clinical Gastroenterology 2007;21(1):3-18.

Friedman SL. Mechanisms of hepatic fibrogenesis. Gastroenterology 2008;134:1655-1669.

Garcia-Tsao G, et al. Management and treatment of patients with cirrhosis and portal hypertension: recommendations from the Department of Veterans Affairs Hepatitis C Resource Center Program and the National Hepatitis C program. The American Journal of Gastroenterology 2009;104(7):1802-1829

Greenbaum L.E. and Wells R.G. The role of stem cells in liver repair and fibrosis. The International Journal of Biochemistry and Cell Biology 2011;43:222-229.

Jiao J, et al. Hepatic fibrosis. Current Opinion in Gastroenterology 2009;25(3):223-229.

Kanwal F, et al. An explicit quality indicator set for measurement of quality of care in patients with cirrhosis. Clinical Gastroenterology and Hepatology 2010;8:709

Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2021; 71: 209–49.

Siegel RL, Miller KD, Fuchs HE, Jemal A. Cancer statistics, 2022. CA Cancer J Clin 2022; 72: 7–33. 3 Petrick JL, Florio AA, Znaor A, et al. International trends in hepatocellular carcinoma incidence, 1978–2012. Int J Cancer 2020; 147: 317–30.

Sun J, Althoff KN, Jing Y, et al. Trends in hepatocellular carcinoma incidence and risk among persons with HIV in the US and Canada, 1996–2015.

JAMA Netw Open 2021; 4: e2037512. 5 Chen T, Liu J, Li Y, Wei S. Burden of disease associated with dietary exposure to aflatoxins in China in 2020. Nutrients 2022; 14: 1027.

Romano A, Angeli P, Piovesan S, et al. Newly diagnosed hepatocellular carcinoma in patients with advanced hepatitis C treated with DAAs: a prospective population study. J Hepatol 2018; 69: 345–52.

Dave S, Park S, Murad MH, et al. Comparative effectiveness of entecavir versus tenofovir for preventing hepatocellular carcinoma in patients with chronic hepatitis B: a systematic review and meta-analysis. Hepatology 2021; 73: 68–78.

Papatheodoridis GV, Idilman R, Dalekos GN, et al. The risk of hepatocellular carcinoma decreases after the first 5 years of entecavir or tenofovir in Caucasians with chronic hepatitis B. Hepatology 2017; 66: 1444–53.

Chiang CJ, Yang YW, You SL, Lai MS, Chen CJ. Thirty-year outcomes of the national hepatitis B immunization program in Taiwan. JAMA 2013; 310: 974–76.

Kanwal F, Kramer JR, Asch SM, Cao Y, Li L, El-Serag HB. Long-term risk of hepatocellular carcinoma in HCV patients treated with direct acting antiviral agents. Hepatology 2020; 71: 44–55.

Kim HY, Lampertico P, Nam JY, et al. An artificial intelligence model to predict hepatocellular carcinoma risk in Korean and Caucasian patients with chronic hepatitis B. J Hepatol 2022; 76: 311–18.

Kim HS, Yu X, Kramer J, et al. Comparative performance of risk prediction models for hepatitis B-related hepatocellular carcinoma in the United States. J Hepatol 2022; 76: 294–301.

Zheng J, Seier K, Gonen M, et al. Utility of serum inflammatory markers for predicting microvascular invasion and survival for patients with hepatocellular carcinoma. Ann Surg Oncol 2017; 24: 3706–14.

Johnson PJ, Dhanaraj S, Berhane S, Bonnett L, Ma YT. The prognostic and diagnostic significance of the neutrophil-tolymphocyte ratio in hepatocellular carcinoma: a prospective controlled study. Br J Cancer 2021; 125: 714–16.

Bruix J, Cheng AL, Meinhardt G, Nakajima K, De Sanctis Y, Llovet J. Prognostic factors and predictors of sorafenib benefit in patients with hepatocellular

carcinoma: analysis of two phase III studies. J Hepatol 2017; 67: 999–1008.

Scheiner B, Pomej K, Kirstein MM, et al. Prognosis of patients with hepatocellular carcinoma treated with immunotherapy—development and validation of the CRAFITY score. J Hepatol 2022; 76: 353–63. 87 Vogel A, Martinelli E, ESMO Guidelines Committe. Updated treatment recommendations for hepatocellular carcinoma (HCC) from the ESMO Clinical Practice guidelines. Ann Oncol 2021; 32: 801–05.

Bruix J, Chan SL, Galle PR, Rimassa L, Sangro B. Systemic treatment of hepatocellular carcinoma: an EASL position paper. J Hepatol 2021; 75: 960–74.

The International Liver Cancer Association. ILCA systemic therapy guidance. https://ilca-online.org/wp-content/uploads/2020/06/Systemic-therapy-guidelines-V1.2.pdf (accessed July 26, 2022).

Kudo M, Kawamura Y, Hasegawa K, et al. Management of hepatocellular carcinoma in Japan: JSH consensus statements and recommendations 2021 update. Liver Cancer 2021; 10: 181–223.

Chen LT, Martinelli E, Cheng AL, et al. Pan-Asian adapted ESMO clinical practice guidelines for the management of patients with intermediate and advanced/relapsed hepatocellular carcinoma: a TOS-ESMO initiative endorsed by CSCO, ISMPO, JSMO, KSMO, MOS and SSO. Ann Oncol 2020; 31: 334–51.

Reig M, Forner A, Rimola J, et al. BCLC strategy for prognosis prediction and treatment recommendation: the 2022 update. J Hepatol 2021; 76: 681–93.

Lang H, Sotiropoulos GC, Dömland M, et al. Liver resection for hepatocellular carcinoma in non-cirrhotic liver without underlying viral hepatitis. Br J Surg 2005; 92: 198–202.

Viganò L, Conci S, Cescon M, et al. Liver resection for hepatocellular carcinoma in patients with metabolic syndrome: a multicenter matched analysis with HCV-related HCC. J Hepatol 2015; 63: 93–101.

Kabir T, Tan ZZ, Syn NL, et al. Laparoscopic versus open resection of hepatocellular carcinoma in patients with cirrhosis: meta analysis. Br J Surg 2021; 109: 21–29. 96 Ivanics T, Claasen MP, Patel MS, et al. Long-term outcomes of laparoscopic liver resection for hepatocellular carcinoma: a propensity score matched analysis of a high-volume North American center. Surgery 2021; 171: 982–91.

Berzigotti A, Reig M, Abraldes JG, Bosch J, Bruix J. Portal hypertension and the outcome of surgery for hepatocellular carcinoma in compensated cirrhosis: a systematic review and meta - analysis. Hepatology 2015; 61: 526–36.

Berardi G, Morise Z, Sposito C, et al. Development of a nomogram to predict outcome after liver resection for hepatocellular carcinoma in Child-Pugh B cirrhosis. J Hepatol 2020; 72: 75–84.

Azoulay D, Ramos E, Casellas-Robert M, et al. Liver resection for hepatocellular carcinoma in patients with clinically significant portal hypertension. JHEP Rep 2020; 3: 100190.

Citterio D, Facciorusso A, Sposito C, Rota R, Bhoori S, Mazzaferro V. Hierarchic interaction of factors associated with liver decompensation after resection for hepatocellular carcinoma. JAMA Surg 2016; 151: 846–53.

Serenari M, Han KH, Ravaioli F, et al. A nomogram based on liver stiffness predicts postoperative complications in patients with hepatocellular carcinoma. J Hepatol 2020; 73: 855–62.

Allaire M, Goumard C, Lim C, Le Cleach A, Wagner M, Scatton O. New frontiers in liver resection for hepatocellular carcinoma. JHEP Rep 2020; 2: 100134.

Marasco G, Alemanni LV, Colecchia A, et al. Prognostic value of the albumin-bilirubin grade for the prediction of post-hepatectomy liver failure: a systematic review and meta-analysis. J Clin Med 2021; 10: 2011.

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Olivier Lieuga ,Tatiana Jiengoué and Augustine Nji Asakizi. (2026). Prevalence of Liver Cirrhosis on patients with Chronic Hepatitis C during treatment by Velso 400/100 mg only at The NGaoundere Protestant Hospital in Adamawa Region Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004016. www.iqresearchjournal.com

ANNEXES

Annex I — Table 1. Results of Patient Characteristics

Chronic Hepatitis C Patient Characteristics	Patients	
	n	%
Nationalities		
Cameroonians	35	100
Age (Years Old)		
18 – 35	13	37,14
36 - 55	7	20,00
55 – Over	15	42,85
Marital Status		
Single	15	42,85
Married	9	25,71
Divorced	11	31,42

Annex II — Table 2. Results of Chronic Hepatitis C Clinical Manifestations

Clinical Manifestations	Abdomen Distention		Ascites		Jaundice		Legs Swelling (Godet)		Hepatosplenomegaly	
	n	%	n	%	n	%	n	%	n	%
Patient	35	100	16	45,71	31	88,57	21	60	30	85,71

Annex III — Table 3. Results of Exams and Tests done to confirm Chronic Hepatitis C

Exams and Tests Done	Elisa HCV		Viral Load HCV		Genotype HCV		Ultrasounds		LFTs	
	n	%	n	%	n	%	n	%	n	%
Patient	35	100	12	34,28	0	0	27	77,14	11	31,42

Annex IV — Table 4. Results of Chronic Hepatitis C Treatment

Treatment Received by HCV Patients	Velso 400/100 mg (Sofosbuvir/Velpastavir)		Sofosbuvir 400mg/Daclastavir 100 mg		Sofosbuvir 400mg/Ledipasvir 90mg		Interferon alfa 2 b	
	n	%	n	%	n	%	n	%
Patient	20	100	0	0	0	0	0	0

Annex V — Table 5. Results of Hepatitis B Complications analyzed on 100 Patients

Complications of Chronic Hepatitis C	Liver Cirrhosis		Hepatocellular Carcinoma		Fulminant Hepatitis		Pulmonary Embolism		Cardiac Liver		Digestive Bleeding	
	n	%	n	%	n	%	n	%	n	%	n	%
Patient	29	82,85	4	11,42	7	20	0	0	0	0	0	0

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

The Assessment of Methods Used to diagnose Hepatocellular Carcinoma to the 396 decompensated cirrhotic patients at the Bertoua Regional Hospital

Olivier Lieuga[a] ,Tatiana Jiengoué[a] and Augustine Nji Asakizi[aa]

Affiliations

q. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Olivier Lieuga

Email:
olivierlieuga@kesmondsuniversity.org

Paper ID: IQRJ-V05I02-26005017

ABSTRACT

Introduction: Hepatocellular carcinoma (HCC) is diagnosed primarily through non-invasive, contrast-enhanced imaging techniques based on tumor vascularity. these methods include multiphasic contrast-enhanced MRI (preferred for high sensitivity) and CT scans, along with contrast-enhanced ultrasound (CEUS). Diagnostic algorithms often incorporate blood biomarkers like Alpha-fetoprotein (AFP) and liver biopsy for atypical cases, often categorized using the LI-RADS system.

Methods: It is descriptive cross – sectional study done on 396 patients living with decompensated cirrhosis recorded at the Intensive Care Unit of Bertoua Regional Hospital from June to October 2025 to diagnose Hepatocellular Carcinoma, after receiving ethical clearance and Research Authorization from Est Public Health Delegation – Cameroon. Convenience sampling was done. The analysis included the methods such as GALAD Score, Fibrotest – Actitest Score, CT Scans, LFTs, APHE, LIRADS, Multiphase CT Scan and Dicckof – 1.

Results: Out to 396 Cirrhosis patients, the prevalence rate of HCC obtained was 68,43%.

Conclusions: At the end of our studies, 68.63% of the 396 patients with liver cirrhosis underwent the following examinations: multiphasic computed tomography (CT) scan, multiphasic magnetic resonance imaging (MRI), FibroScan, FibroTest, Actitest, and AFP testing to confirm the diagnosis of hepatocellular carcinoma (HCC). The absence of other examinations, such as AFP-L3, DCP, Dickkoff-1, and BCLC liver biopsy, does not always allow for the determination of the cancer stage (stage 0, stage A, stage B, stage C, and stage D) and raises concerns about the quality of treatment.

Keywords : *Hepatocellular Carcinoma, Liver Cirrhosis , Hepatitis B, Hepatitis C*

1. INTRODUCTION

Hepatocellular Carcinoma (HCC) is a higher aggressive malignancy with global impact, especially in the context of the rising epidemic of metabolic dysfunction associated with chronic hepatitis, Emily Kinsey and Hannah M. Lee (2024). It is noted that liver cancer is the sixth most common cancer, but its aggressive nature and poor prognosis raise it to the third highest cause of cancer related deaths. Hepatocellular Carcinoma (HCC) makes up approximately 75% of primary liver cancer cases, with a smaller proportion due to Cholangiocarcinoma, Baffy G (2016).

Hepatocellular Carcinoma (HCC) is the fourth leading cause of cancer related mortality Worldwide and a leading cause of death in cirrhosis, Previn Ganesan and Laura M.Kulik (2023).The prognosis in HCC is poor, with mortalities approximating incidence rates Worldwide. The second leading cause of cancer death after lung cancer in men is HCC, Jemal A, Ward EM, Johnson CJ et al (2014). Considered as a primary tumor of the liver, HCC occurs in approximately 85% of patients diagnosed with cirrhosis caused either by viral hepatitis infection, or metabolic dysfunction, Semmler G and Mandofer (2022). The Global burden of HCC is highest in Asia and Sub-Saharan Africa due to the high prevalence of chronic hepatitis in those regions. Men are more commonly affected and have a higher mortality than Women, with liver cancer being the leading cause of cancer death among men in over 20 countries, Emily Kinsey and Hannah M. Lee (2024)

HCC account for one third of total cancer incidence and mortality in developing countries, Ahmad Zia Shms and Ulrike Haug (2017). To date, there is no systematic synthesis of evidence regarding strategies to prevent HCC in developing countries. The epidemiology of HCC is rapidly evolving with chronic liver disease becoming an increasing cause of fibrosis, cirrhosis and HCC, Previn Ganesan and Laura M. Kulik (2022). Most patients develop HCC in the setting of Cirrhosis, and the risk factors for HCC include Hepatitis B (HBV), Hepatitis C (HCV), alcohol related liver disease (ALD), and excess body weight, and type 2 diabetes, with is often associated metabolic dysfunction associated with steatotic liver disease (MASLD), as well as aflatoxin exposure, Ahmad Zia Shms (2023). The prevalence of various risk factors for HCC is region specific with viral causes being more common in the East and non - viral etiologies in the West. In Japan and Egypt, HCV is the primary driver of HCC, Venook AP, Papandreouc et al (2010). In Asia and Africa, Chronic HBV is a primary cause of HCC.

2. RELATED WORKS

Multiphase CT or MRI with contrast is used for definitive diagnosis of HCC; arterial phase enhancement (APHE); and washout on the portal venous phase. The Liver Reporting and Data System (LIRADS) allows for the classification of liver lesions in cirrhosis in a standardized manner, Marasco G, Alemanni LV, Coleechia A, et al (2021). Multiphase MRI may have higher sensitivity but comparable specificity to CT. Imaging is often sufficient to definitively diagnose HCC (LIRADS-5), eliminating the need for a biopsy for diagnostic purposes, Allaire M, Goumard C, Lim C, Le cleach A, Wagner M and Sertton O (2020). However, when imaging findings are nondiagnostic but highly suspicious for HCC (LIRADS-4), clinicians may repeat an imaging study in 3 months, attempt the alternative imaging modality, or consider pursuing a biopsy for definitive diagnosis, Cittero D, Faccidrusso A, Sposito C, Rota R,

Ghouri S, and Mazzaferro V (2016). The advantages of CT include shorter duration of examination and lower cost. The disadvantages include exposure to radiation and iodinated contrast. This is especially relevant in individuals with advanced liver disease who can often have concurrent renal dysfunction, Azoulay D, Ramos E, Casellas – Robert M, et al (2020).

Hepatocellular carcinoma can be diagnosed on the basis of validated imaging criteria (in people who have liver cirrhosis) or tissue biopsy. Commonly used imaging modalities include multiphasic CT or MRI, in which hepatocellular carcinoma typically shows enhancement (brightness compared with surrounding parenchyma) in the early arterial phase, and washout (temporal decrease in enhancement relative to surrounding parenchyma) in the delayed phase, Marasco G, Alemanni LV, ColeechiaA, et al (2021). The latter creates a peripheral rim of enhancement around the tumour, resulting in the formation of a capsule; an observation highly specific for hepatocellular carcinoma, Kabir T, Tan ZZ, Syn NL et al (2021). This imaging feature has been prospectively confirmed and universally adopted by guidelines.

Usually, solid hepatic nodules raise suspicion for hepatocellular carcinoma once they are ≥1 cm, especially in patients with liver cirrhosis. Lesions that are identified incidentally or through regular screening by ultrasound, dynamic contrast-enhanced CT or MRI of the abdomen should be obtained for further assessment, Lang H, Sotiropoulos GC, Domland M et al (2005). Pathological diagnosis of hepatocellular carcinoma is typically based on the examination of a resection or explant specimen, or from a biopsy sample.

Historically, biopsy has been reserved for lesions in which non-invasive imaging criteria for diagnosis are not met or are not applicable (for patients without cirrhosis), Reig M, Former A, Rimola J et al (2022). The prognosis for hepatocellular carcinoma depends not only on tumour characteristics, such as tumour burden, extrahepatic spread, vascular infiltration, or tumour differentiation, but is heavily influenced by the underlying liver disease, Chen LT Martinelli E, Cheng AL et al (2021). Additionally, higher levels of serum AFP are significantly associated with increased mortality, independent of demographic and clinical factors or treatment, and have been shown to predict the risk of tumour recurrence after resection and liver transplantation, Kudo M, Kawamura Y, Hasegawa K et al (2021).

3. MATERIALS & METHODS

It is descriptive cross – sectional study done on 396 patients living with decompensated cirrhosis recorded at the Intensive Care Unit of Bertoua Regional Hospital from June to October 2025 to diagnose Hepatocellular Carcinoma, after receiving ethical clearance and Research Authorization from Est Public Health Delegation – Cameroon. Convenience sampling was done. The analysis included the methods such as GALAD Score, Fibrotest – Actitest Score, CT Scans, LFTs, APHE, LIRADS, Multiphase CT Scan and Dicckof – 1.

Convenience sampling was done, all the cases of elective HCC admitted during the study period were included. Age > 20, men and women, Cameroonians or not but residents in Cameroon. The simple size was 271 patients calculated was calculated using the formula n = (Z2 x p x q) / e2 where n = minimum sample size, z= confidence interval, p= prevalence of decompensated cirrhosis, q= 1 – p and e= margin of error, 3%.

The data for this study were collected using structured questionnaires, entered and analysed in Microsoft Excel 2016, confidence interval was calculated along with frequency and percentages for binary data

4. RESULTS & DISCUSSION

The Assessment of Methods Used to diagnose Hepatocellular Carcinoma to the 396 decompensated cirrhotic patients at the Bertoua Regional Hospital among which we analyzed these characteristics on each patient and are summarized them by the tables.

[See Annex — Table 1: Hepatocellular Carcinoma Patient Characteristics]

[See Annex — Table 2 : Results of Exams/Tests done for the diagnostic of HCC]

[See Annex — Table 3 : Results of Exams/Tests done for the diagnostic of HCC]

At the end of our studies, 68.63% of the 396 patients with liver cirrhosis underwent the following examinations: multiphase CT scan, multiphase MRI, FibroScan, FibroTest, Actitest, and AFP to confirm the diagnosis of HCC. The absence of other examinations such as AFP-L3, DCP, Dickkoff-1, and liver biopsy using the BCLC method does not always allow for the determination of the cancer stage (Stage 0, Stage A, Stage B, Stage C, and Stage D). This leaves some ambiguity regarding treatment. We also noted that the GALAD score remained unavailable due to the unavailability of two tests (AFP-L3 and DCP) in local laboratories. Another identified factor is the low income of some patients, who are unable to afford these expensive examinations in both local and foreign laboratories. Diagnosing hepatocellular carcinoma (HCC) in decompensated cirrhotic patients is a complex, high-stakes process that relies heavily on advanced, non-invasive imaging, as liver function is too compromised for routine biopsies, Johnson PJ et al,…(2021). While conventional ultrasound is used for surveillance, it lacks the sensitivity needed for definitive diagnosis in advanced liver disease, often requiring follow-up with multiphasic computed tomography (CT) or magnetic resonance imaging (MRI), Boyles TH et al,..(2011). It also involves using contrast-enhanced MRI or CT to identify arterial phase hyperenhancement and venous washout, with MRI offering superior sensitivity. Guidelines emphasize leveraging LI-RADS for standardized imaging, with liver biopsy reserved for inconclusive cases, Bruix J et al,..(2021).

5. CONCLUSION

We recommended that the hospital improve its diagnostic equipment for HCC, seek funding from NGOs to provide free or subsidized care for underprivileged patients, and recruit specialists such as gastroenterologists, digestive surgeons, and digestive oncologists.

REFERENCES

Jonas M, Kelly DA, Mizerski J, et al. Clinical trial of lamivudine in children with chronic hepatitis B. N Engl J Med 2002;346:1706–13.

Lai CL, Lin HJ, Lau HJ, et al. Effect of recombinant a2 IFN with or without prednisolone in Chinese HBsAg carrier children. QJM 1991;78:155–63.

Bortolotti F, Jara P, Barbera C, et al. Long term effect of a interferon in children with chronic hepatitis B. Gut 2000; 46:715–8

Lampertico P, Del Ninno E, Vigano M, et al. Long term suppression of HBeAg negative chronic hepatitis B by 24 months’ interferon therapy. Hepatol 2003;37:756–63.

Kasper D.L., Fauci A.S., Longo D.L, et al: Harrison’s principles of internal medicine. 16th edition. 2005.

Keeffe EB, Dieterich DT, Han SH, Jacobson IM, Martin P, Schiff ER, et al. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States: 2008 update. Clin Gastroenterol Hepatol. Dec 2008;6(12):1315-41; quiz 1286.

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology. May 2009;49(5 Suppl):S72-84

Fattovich G, Stroffolini T, Zagni I, et al. Hepatocellular carcinoma in cirrhosis: incidence and risk factors. Gastroenterology 2004; 127(Suppl 1):S35–S50.

McMahon BJ, Alberts SR, Wainwright RB, et al. Hepatitis B-related sequelae. Prospective study in 1400 hepatitis B surface antigenpositive Alaska native carriers. Arch Intern Med 1990;150:1051– 1054.

Mizokami M, Orito E. Molecular evolution of hepatitis viruses. Intervirology 1999;42:159–165

Tanaka Y, Kurbanov F, Mano S, et al. Molecular tracing of the global hepatitis C virus epidemic predicts regional patterns of hepatocellular carcinoma mortality. Gastroenterology 2006;130: 703–714.

El-Serag HB, Rudolph KL. Hepatocellular carcinoma: epidemiology and molecular carcinogenesis. Gastroenterology 2007;132: 2557–2576.

Chang MH. Hepatitis B virus and cancer prevention. Recent Results Cancer Res 2011;188:75–84

Fattovich G, Bortolotti F, Donato F. Natural history of chronic hepatitis B: special emphasis on disease progression and prognostic factors. J Hepatol 2008;48:335–352

Yang JD, Kim WR, Coelho R, et al. Cirrhosis is present in most patients with hepatitis B and hepatocellular carcinoma. Clin Gastroenterol Hepatol 2011;9:64–70

Yang HI, Lu SN, Liaw YF, et al. Hepatitis B e antigen and the risk of hepatocellular carcinoma. N Engl J Med 2002;347:168–174

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology 2009;49(Suppl): S72–S84.

Chun HM, Roediger MP, Hullsiek KH, et al. Hepatitis B Virus Coinfection Negatively Impacts HIV Outcomes in HIV Seroconverters. The Journal of Infectious Diseases. 2012;205(2):185-193. doi:10.1093/infdis/jir720

McMahon BJ. The natural history of chronic hepatitis B virus infection. Hepatology. 2009;49(5 Suppl):S45-55 2.

Schweitzer A,Horn J, Mikolajczyk RT, Krause G, Ott JJ Estimations of worldwide prevalence of chronic hepatitis B virus infection: a systematic review of data published between 1965 and 2013.

Lancet 2015; 386: 1546–55 3. Ott J, Stevens G, Groeger J, Wiersma S. Global epidemiology of Hepatitis B virus infection: new estimates of age-specific HBsAg seroprevalence and endemicity. Vaccine 2012;30(12): 2212–9 4.

Lukhwareni A, Burnett RJ, Selabe SG, Mzileni MO, Mphahlele MJ. Increased detection of HBV DNA in HBsAg-positive and HBsAgnegative SouthAfrican HIV/AIDS patients enrolling for Highly active antiretroviral therapy at a Tertiary Hospital. J Med Virol 2009;81(3):406–12 5.

Boyles TH, Cohen K. The prevalence of hepatitis B infection in a rural SouthAfrican HIV clinic. S Afr Med J 2011;101(7):470–1 6.

Firnhaber C, Reyneke A, Schulze D, et al. The prevalence of hepatitis B co-infection in a South African urban government HIV clinic. S Afr Med J 2008;98(7):541-4 7.

Burnett RJ, Ngobeni JM, Francois G, et al. Increased exposure to hepatitis B virus infection in HIV-positive South African antenatal women. Int J STD AIDS 2007;18(3):152–6 8.

Andersson MI, Maponga TG, Ijaz S, et al. The epidemiology of hepatitis B virus infection in HIV-infected and HIV-uninfected pregnant women in the Western Cape, South Africa. Vaccine 2013;31(47):5579-84 9.

Puoti M1, Torti C, Bruno R, Filice G, Carosi G. Natural history of chronic hepatitis B in co-infected patients. J Hepatol 2006; 44(1 Suppl):S65-70 10.

Maynard JE. Hepatitis B: Global importance and need for control. Vaccine 1990;8(SUPPL.):S18-S20

Mohd Hanafiah K, Groeger J, Flaxman AD, Wiersma ST. Global epidemiology of hepatitis virus infection: new estimates of age-specific antibody to HCV seroprevalence. Hepatology 2013;57(4):1333-42

Lavanchy D. The global burden of hepatitis C. Liver Int. 2009;29 Suppl 1:74-Ellis LA, Brown D, Conradie JD, Paterson A, Sher R, Millo J, et al. Prevalence of hepatitis C inSouth Africa: detection of anti-HCV in recent and stored serum. J Med Virol 1990;32(4):249- 51

Davis GL. Hepatitis C virus genotypes and quasispecies. Am J Med 1999;107(6B):21S-6S

Messina JP, Humphreys I, Flaxman A, et al. Global distribution and prevalence of hepatitis C virus genotypes. Hepatology. 2015;61(1):77–87

Benova L, Mohamoud YA, Calvert C, AbuRaddad LJ. Vertical transmission of hepatitis C virus: systematic review and meta-analysis. Clin Infect Dis. 2014;59(6):765–73

Floreani A. Hepatitis C and pregnancy. World J Gastroenterol. 2013;19(40):6714–20 Jafari S, Copes R, Baharlou S, Etminan M, Buxton J. Tattooing and the risk of transmission of hepatitis C: a systematic review and metaanalysis. Int J Infect Dis.2010;14(11):E928– E9

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Zein CO, Levy C, Basu A, Zein NN. Chronic hepatitis C and type II diabetes mellitus: a prospective cross-sectional study. Am J Gastroenterol. 2005;100(1):48-5

EASL Recommendations on Treatment of Hepatitis C 2018. J Hepatol 2018; 69(2): 461- 511

Smith JO, Sterling RK. Systematic review: non-invasive methods of fibrosis analysis in chronic hepatitis C. Alimentary pharmacology & therapeutics. 2009;30(6):557-76

Karmochkine M, Carrat F, Dos Santos O, Cacoub P, Raguin G. A case-control study of risk factors for hepatitis C infection in patients with unexplained

routes of infection. J Viral Hepat. 2006;13(11):775–82

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Lau GK, Piratvisuth T, Luo KX, Marcellin P, Thongsawat S, et al. (2005) Peginterferon Alfa-2a, lamivudine, and the combination for HBeAgpositive chronic hepatitis B. N Engl J Med 352: 2682-2695. 2.

Conjeevaram HS, Lok AS (2003) Management of chronic hepatitis B. J Hepatol 38 Suppl 1: S90-103.

Karayiannis P (2003) Hepatitis B virus: old, new and future approaches to antiviral treatment. J Antimicrob Chemother 51: 761-785.

Wellington K, Jarvis B (2001) Silymarin: a review of its clinical properties in the management of hepatic disorders. BioDrugs 15: 465-489.

Strader DB, Bacon BR, Lindsay KL, La Brecque DR, Morgan T, et al. (2002) Use of complementary and alternative medicine in patients with liver disease. Am J Gastroenterol 97: 2391-2397.

Eisenberg DM, Davis RB, Ettner SL, Appel S, Wilkey S, et al. (1998) Trends in alternative medicine use in the United States, 1990-1997: results of a follow-up national survey. JAMA 280: 1569-1575.

Flora K, Hahn M, Rosen H, Benner K (1998) Milk thistle (Silybum marianum) for the therapy of liver disease. Am J Gastroenterol 93: 139-143.

Varga Z, Czompa A, Kakuk G, Antus S (2001) Inhibition of the superoxide anion release and hydrogen peroxide formation in PMNLs by flavonolignans. Phytother Res 15: 608-612.

Dehmlow C, Erhard J, de Groot H (1996) Inhibition of Kupffer cell functions as an explanation for the hepatoprotective properties of silibinin. Hepatology 23: 749-754

Wilasrusmee C, Kittur S, Shah G, Siddiqui J, Bruch D, et al. (2002) Immunostimulatory effect of Silybum Marianum (milk thistle) extract. Med Sci Monit 8: BR439-443.

Neumann UP, Biermer M, Eurich D, Neuhaus P, Berg T (2010) Successful prevention of hepatitis C virus (HCV) liver graft reinfection by silibinin mono-therapy. J Hepatol 52: 951-952.

Beinhardt S, Rasoul-Rockenschaub S, Maieron A, PH S-M, Hofer H PF (2012) Intravenous Silibinin-therapy in patients with chronic hepatitis C in the transplant setting. J Hepatology 56: S77.

Pares A, Planas R, Torres M, Caballeria J, Viver JM, et al. (1998) Effects of silymarin in alcoholic patients with cirrhosis of the liver: results of a controlled, double-blind, randomized and multicenter trial. J Hepatol : 615-621.

Sepanlou SG, Safiri S, Bisignano C, et al; GBD 2017 Cirrhosis Collaborators. The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol Hepatol. 2020;5(3):245-266. doi:10.1016/S2468-1253(19)30349-8

Tapper EB, Parikh ND. Mortality due to cirrhosis and liver cancer in the United States, 1999-2016. BMJ. 2018;362:k2817. doi:10.1136/bmj.k2817

Centers for Disease Control and Prevention. CDC WONDER: about provisional mortality statistics, 2018 through last month. Accessed March 13, 2023. https://wonder.cdc.gov/mcd-icd10-provisional.html

Serper M, Kaplan DE, Taddei TH, Tapper EB, Cohen JB, Mahmud N. Nonselective beta blockers, hepatic decompensation, and mortality in cirrhosis: a national cohort study. Hepatology. 2023;77(2):489-500. doi:10.1002/hep.32737

Flemming JA, Dewit Y, Mah JM, Saperia J, Groome PA, Booth CM. Incidence of cirrhosis in young birth cohorts in Canada from 1997 to 2016: a retrospective population-based study. Lancet Gastroenterol Hepatol. 2019;4(3):217-226. doi:10.1016/S2468-1253(18)30339-X

Hirode G, Saab S, Wong RJ. Trends in the burden of chronic liver disease among hospitalized US adults.JAMA Netw Open. 2020;3(4):e201997-e201997. doi:10.1001/jamanetworkopen.2020.1997

Foster C, Baki J, Nikirk S, Williams S, Parikh ND, Tapper EB. Comprehensive health-state utilities in contemporary patients with cirrhosis. Hepatol Commun. 2020;4 (6):852-858. doi:10.1002/hep4.1512

Asrani SK, Hall L, Hagan M, et al. Trends in chronic liver disease-related hospitalizations: a population-based study. Am J Gastroenterol. 2019;114(1):98-106. doi:10.1038/s41395-018- 0365-4

Tarao K, Nozaki A, Ikeda T, et al. Real impact of liver cirrhosis on the development of hepatocellular carcinoma in various liver diseases-meta-analytic assessment. Cancer Med. 2019;8(3):1054-1065. doi:10.1002/cam4.1998

Carbonell N, Pauwels A, Serfaty L, Fourdan O, Lévy VG, Poupon R. Improved survival after variceal bleeding in patients with cirrhosis over the past two decades. Hepatology. 2004;40(3):652-659. doi: 10.1002/hep.20339

Arvaniti V, et al. Infections in patients with cirrhosis increase mortality four fold and should be used in determining prognosis. Gastroenterology 2010;139:1246-1256.

Berzigott A., et al. Obesity is an independent risk factor for clinical decompensation in patients with cirrhosis. Hepatology 2011;54:555-561.

Dienstag J.L., et al. A prospective study of progression in compensated, histologically advanced chronic hepatitis C. Hepatology 2011;54:396-405.

Durand F, et al. Assessment of the prognosis of cirrhosis: Child-Pugh versus MELD. Journal of Hepatology2005;42.

Franchis de, et al. Non-invasive diagnosis of cirrhosis and the natural history of it complications. Best

Practice & Research Clinical Gastroenterology 2007;21(1):3-18.

Friedman SL. Mechanisms of hepatic fibrogenesis. Gastroenterology 2008;134:1655-1669.

Garcia-Tsao G, et al. Management and treatment of patients with cirrhosis and portal hypertension: recommendations from the Department of Veterans Affairs Hepatitis C Resource Center Program and the National Hepatitis C program. The American Journal of Gastroenterology 2009;104(7):1802-1829

Greenbaum L.E. and Wells R.G. The role of stem cells in liver repair and fibrosis. The International Journal of Biochemistry and Cell Biology 2011;43:222-229.

Jiao J, et al. Hepatic fibrosis. Current Opinion in Gastroenterology 2009;25(3):223-229.

Kanwal F, et al. An explicit quality indicator set for measurement of quality of care in patients with cirrhosis. Clinical Gastroenterology and Hepatology 2010;8:709

Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2021; 71: 209–49.

Siegel RL, Miller KD, Fuchs HE, Jemal A. Cancer statistics, 2022. CA Cancer J Clin 2022; 72: 7–33. 3 Petrick JL, Florio AA, Znaor A, et al. International trends in hepatocellular carcinoma incidence, 1978–2012. Int J Cancer 2020; 147: 317–30.

Sun J, Althoff KN, Jing Y, et al. Trends in hepatocellular carcinoma incidence and risk among persons with HIV in the US and Canada, 1996–2015.

JAMA Netw Open 2021; 4: e2037512. 5 Chen T, Liu J, Li Y, Wei S. Burden of disease associated with dietary exposure to aflatoxins in China in 2020. Nutrients 2022; 14: 1027.

Romano A, Angeli P, Piovesan S, et al. Newly diagnosed hepatocellular carcinoma in patients with advanced hepatitis C treated with DAAs: a prospective population study. J Hepatol 2018; 69: 345–52.

Dave S, Park S, Murad MH, et al. Comparative effectiveness of entecavir versus tenofovir for preventing hepatocellular carcinoma in patients with chronic hepatitis B: a systematic review and meta-analysis. Hepatology 2021; 73: 68–78.

Papatheodoridis GV, Idilman R, Dalekos GN, et al. The risk of hepatocellular carcinoma decreases after the first 5 years of entecavir or tenofovir in Caucasians with chronic hepatitis B. Hepatology 2017; 66: 1444–53.

Chiang CJ, Yang YW, You SL, Lai MS, Chen CJ. Thirty-year outcomes of the national hepatitis B immunization program in Taiwan. JAMA 2013; 310: 974–76.

Kanwal F, Kramer JR, Asch SM, Cao Y, Li L, El-Serag HB. Long-term risk of hepatocellular carcinoma in HCV patients treated with direct acting antiviral agents. Hepatology 2020; 71: 44–55.

Kim HY, Lampertico P, Nam JY, et al. An artificial intelligence model to predict hepatocellular carcinoma risk in Korean and Caucasian patients with chronic hepatitis B. J Hepatol 2022; 76: 311–18.

Kim HS, Yu X, Kramer J, et al. Comparative performance of risk prediction models for hepatitis B-related hepatocellular carcinoma in the United States. J Hepatol 2022; 76: 294–301.

Zheng J, Seier K, Gonen M, et al. Utility of serum inflammatory markers for predicting microvascular invasion and survival for patients with hepatocellular carcinoma. Ann Surg Oncol 2017; 24: 3706–14.

Johnson PJ, Dhanaraj S, Berhane S, Bonnett L, Ma YT. The prognostic and diagnostic significance of the neutrophil-tolymphocyte ratio in hepatocellular carcinoma: a prospective controlled study. Br J Cancer 2021; 125: 714–16.

Bruix J, Cheng AL, Meinhardt G, Nakajima K, De Sanctis Y, Llovet J. Prognostic factors and predictors of sorafenib benefit in patients with hepatocellular carcinoma: analysis of two phase III studies. J Hepatol 2017; 67: 999–1008.

Scheiner B, Pomej K, Kirstein MM, et al. Prognosis of patients with hepatocellular carcinoma treated with immunotherapy—development and validation of the CRAFITY score. J Hepatol 2022; 76: 353–63. 87 Vogel A, Martinelli E, ESMO Guidelines Committe. Updated treatment recommendations for hepatocellular carcinoma (HCC) from the ESMO Clinical Practice guidelines. Ann Oncol 2021; 32: 801–05.

Bruix J, Chan SL, Galle PR, Rimassa L, Sangro B. Systemic treatment of hepatocellular carcinoma: an EASL position paper. J Hepatol 2021; 75: 960–74.

The International Liver Cancer Association. ILCA systemic therapy guidance. https://ilca-online.org/wp-content/uploads/2020/06/Systemic-therapy-guidelines-V1.2.pdf (accessed July 26, 2022).

Kudo M, Kawamura Y, Hasegawa K, et al. Management of hepatocellular carcinoma in Japan: JSH consensus statements and recommendations 2021 update. Liver Cancer 2021; 10: 181–223.

Chen LT, Martinelli E, Cheng AL, et al. Pan-Asian adapted ESMO clinical practice guidelines for the management of patients with intermediate and advanced/relapsed hepatocellular carcinoma: a TOS-ESMO initiative endorsed by CSCO, ISMPO, JSMO, KSMO, MOS and SSO. Ann Oncol 2020; 31: 334–51.

Reig M, Forner A, Rimola J, et al. BCLC strategy for prognosis prediction and treatment recommendation: the 2022 update. J Hepatol 2021; 76: 681–93.

Lang H, Sotiropoulos GC, Dömland M, et al. Liver resection for hepatocellular carcinoma in non-cirrhotic liver without underlying viral hepatitis. Br J Surg 2005; 92: 198–202.

Viganò L, Conci S, Cescon M, et al. Liver resection for hepatocellular carcinoma in patients with metabolic

syndrome: a multicenter matched analysis with HCV-related HCC. J Hepatol 2015; 63: 93–101.

Kabir T, Tan ZZ, Syn NL, et al. Laparoscopic versus open resection of hepatocellular carcinoma in patients with cirrhosis: meta analysis. Br J Surg 2021; 109: 21–29. 96 Ivanics T, Claasen MP, Patel MS, et al. Long-term outcomes of laparoscopic liver resection for hepatocellular carcinoma: a propensity score matched analysis of a high-volume North American center. Surgery 2021; 171: 982–91.

Berzigotti A, Reig M, Abraldes JG, Bosch J, Bruix J. Portal hypertension and the outcome of surgery for hepatocellular carcinoma in compensated cirrhosis: a systematic review and meta - analysis. Hepatology 2015; 61: 526–36.

Berardi G, Morise Z, Sposito C, et al. Development of a nomogram to predict outcome after liver resection for hepatocellular carcinoma in Child-Pugh B cirrhosis. J Hepatol 2020; 72: 75–84.

Azoulay D, Ramos E, Casellas-Robert M, et al. Liver resection for hepatocellular carcinoma in patients with clinically significant portal hypertension. JHEP Rep 2020; 3: 100190.

Citterio D, Facciorusso A, Sposito C, Rota R, Bhoori S, Mazzaferro V. Hierarchic interaction of factors associated with liver decompensation after resection for hepatocellular carcinoma. JAMA Surg 2016; 151: 846–53.

Serenari M, Han KH, Ravaioli F, et al. A nomogram based on liver stiffness predicts postoperative complications in patients with hepatocellular carcinoma. J Hepatol 2020; 73: 855–62.

Allaire M, Goumard C, Lim C, Le Cleach A, Wagner M, Scatton O. New frontiers in liver resection for hepatocellular carcinoma. JHEP Rep 2020; 2: 100134.

Marasco G, Alemanni LV, Colecchia A, et al. Prognostic value of the albumin-bilirubin grade for the prediction of post-hepatectomy liver failure: a systematic review and meta-analysis. J Clin Med 2021; 10: 2011.

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Olivier Lieuga, Tatiana Jiengoue Tchakonang, and Augustine Nji Asakizi. (2026). The Assessment of Methods Used to diagnose Hepatocellular Carcinoma to the 396 decompensated cirrhotic patients at the Bertoua Regional Hospital. IQ Research Journal, 5(2), IQRJ-V05I02-26004017. www.iqresearchjournal.com

ANNEXES

Annex I — Table 1: Hepatocellular Carcinoma Patient Characteristics

Patient Characteristics	Cameroonians		Chadians		Nigerians		Centrafric Republic		Others Nationalities	
	n	%	n	%	n	%	n	%	n	%
Patient Characteristics	213	58,33	87	21,96	54	13,63	32	8,08	10	2,52
Age (Years Old)										
20 – 35	125	58,68	61	70,11	15	27,77	9	28,12	5	50
35 - 55	56	26,29	14	16,09	13	24,07	12	37,5	3	30
55 – Over	32	15,02	12	13,79	26	48,14	11	34,37	2	20

Annex II — Table 2 : Results of Exams/Tests done for the diagnostic of HCC

Imaging Tests	Multiphase CT Scan		Multiphase MRI		Arterial Phase Enhancement		Fibroscan		Liver Imaging Reporting and Data System	
	n	%	n	%	n	%	n	%	n	%
Patient	271	68,43	0	0	0	0	271	68,43	0	0

Annex III — Table 3 : Results of Exams/Tests done for the diagnostic of HCC

Liver Function Tests	Fibrotest – Actitest		GALAD Score		AFP Test	
	n	%	n	%	n	%
Patient	271	68,43	0	0	271	68,43

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

The Prevalence of Liver Metastasis and Cancer Death to the HCC patient under treatment at the Bertoua Regional Hospital in Est Region Cameroon

Olivier Lieuga[a] ,Tatiana Jiengoué[a] and Augustine Nji Asakizi[a]

Affiliations

r. [a] School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Olivier Lieuga

Email: *olivierlieuga@kesmondsuniversity.org*

Paper ID: IQRJ-V05I02-26005018

ABSTRACT

Introduction: Effective therapies for hepatocellular carcinoma (HCC) are selected based on tumor stage, liver function (Child-Pugh score), and overall patient health, with a multidisciplinary approach providing the best outcomes. Curative treatments are generally reserved for early-stage disease, while systemic therapies are used for advanced stages

Methods: It is descriptive study done on the prevalence of cancer death occurred to the HCC patients (187 men and 84 women) under treatment at the Intensive Care Unit of Bertoua Regional Hospital from June to September 2025 after receiving ethical clearance and Research Authorization from Est Public Health Delegation – Cameroon. Convenience sampling was done. The analysis included chemotherapy, immunotherapy, locoregional therapies, liver transplantation and surgical resection. Data were collected using structured questionnaires, observations and analyzed using descriptive statistics and analyses

Results: Despite Sorafenib 200mg given to the 28,04% of 271 HCC patients, 45,38% has developed liver metastasis and 25,46% are died. Any others therapies used to treat patient such as immunotherapy, surgical resection, liver transplantation, locoregional therapies and local abblations.

Keywords: *Hepatocellular, Liver Cirrhosis ,Therapies ,Carcinoma*

1. INTRODUCTION

HCC differs from other cancers in that the majority of cases arise on the background of a diseased organ, with chronic necro-inflammation, often accompanied by fibrosis or cirrhosis, therefore likely to be dominant influences on tumour immunity, *(Bermier, J. 2016)*. Treatment options for patients with hepatocellular carcinoma are outlined in national and international guidelines, with slight differences in the therapeutic approach between Asia, Europe, and North America, *Kim HY, Lampertico P, Nam JY, et al (2022). The Barcelona Clinic of Liver Cancer (BCLC)* algorithm is the most widely used staging system and subdivides patients with hepatocellular carcinoma into five clinical stages: very early stage (BCLC 0), early stage (BCLC A), intermediate stage (BCLC B), advanced stage (BCLC C), and terminal stage (BCLC D), *Kim HS, Yu X, Kramar J et al (2022)*.

Treatment options are broadly categorized as curative (liver transplantation, resection, or ablation/segmental transarterial radioembolization [TARE]) and noncurative (transarterial chemoembolization [TACE], systemic therapies), *Zheng J, Seier K, Gonen M, et al (2017)*. When feasible, transplantation is the most definitive treatment option, but organ shortage limits this curative therapy. Systemic therapies are being studied in BCLC A and B to see if this addition to traditional therapy can further improve OS, *Johnson PJ, Ahamaraj S, Berhane S, Bonnett L and Ma YT (2021).*

Surgery (liver resection or liver transplantation) represents the main curative treatment option for patients with hepatocellular carcinoma. Ideal candidates for liver surgery are those with single tumours and maintained liver function, *Bruix J, Cheng AL, Meinhardt G, Nakajima K, De sanctis Y and L Lovet (2017)*. Liver transplantation is generally recommended for patients with multifocal disease or decompensated cirrhosis. The surgical management of patients with hepatocellular carcinoma who have cirrhosis is complex, *Scheiner B, Pomeij K, Kirstein MM, et al (2022)*. Patients should therefore be assessed by multidisciplinary teams in experienced centres and both resection and transplantation should be considered. When treating a patient with hepatocellular carcinoma with liver resection it is important to determine the patient's underlying liver function, the international liver cancer association (2022). Hepatocellular carcinoma in non-cirrhotic liver is less common but, in this population, liver resection should be the first treatment option if the tumour is technically resectable. The goal of liver resection in patients with hepatocellular carcinoma is to achieve a complete R0 outcome, with clear resection margins, *Zheng J, Seier K, Gonen M, et al (2017)*

HCC onset and progression result from the dysregulation of mechanisms regulating various cellular aspects, such as proliferation, apoptosis, motility, autophagy, angiogenesis and metabolism. The drugs used currently for HCC treatment interface with these processes at multiple levels, *Shigeta et al (2020).* With the increasing knowledge of the HCC tumor immune environment, immunotherapy either alone or in combination with targeted treatments has been developed. Immune response mechanisms during tumor progression have been investigated for decades, the efficacy and safety of immunotherapeutic methods are also being tested in the clinical treatment of malignancies, *Deng H et al (2020)*.

2. RELATED WORKS

In the 1980s, the first studies in immunotherapy were initiated to use it in the treatment of HCC, is an immunotherapy that stimulates the immune system as an adjuvant treatment for cancer, with the recognition of cellular receptors that stimulate the body's defences against tumor cells, *Mei J et al.. (2021).* Immunotherapy is a hot spot in the field of cancer treatment represented by immune checkpoint inhibitors (ICIs), which aim to improve the immune system's ability to recognize target and eliminate cancer cells, *Johnson D.B et al (2015).* The composition of the HCC immune microenvironment is the result of the interaction of immunosuppressive cells, immune effector cells, cytokine environment, and tumor cell intrinsic signalling pathway, *Owonikolo T.K et al (2021)* and immunotherapy with strong antitumor immunity has received more and more research attention due to the limited responsiveness of HCC to ICIs monotherapy, *Zhong L et al (2021).*

The immune system has safeguards in place to prevent it from attacking healthy cells, the safeguards are called checkpoints, *Wang L et al (2012).* Some cancers have learned how to activate these checkpoints to avoid being found and killed by the immune system. Various modes of immunotherapy – including monoclonal antibodies, immune checkpoint inhibitors, cancers vaccines, adoptive – T – cell therapies and oncolytic viruses have achieved considerable success in clinical trials and patient results, *Zheng X et al (2022).* Immune Checkpoint Inhibitors (ICIs) is the most established method cancer cells often checkpoints "(proteins on immune cells that keep responses in check, like PD-1 and CT LA – 4), PD – 1/PD – L1 inhibitors like nivolumab, pembrolizumab, atezolizumab, durvalumab) block the interaction between the programmed death – 1 protein on T – cells and its ligand on tumor cells, unleashing the T-cells to attack the cancer, *Kim N et al (2018).*

CTL A – L1 inhibitors (eg; ipilimumab, tremelimumab block another inhibitory pathway, further enhancing T – Cell activity, *Meerveld – Eggink A et al (2017).* Combination therapy has proven more effective than monotherapy. A common first line treatment for advanced HCC combines atezolizumag (anti – PD – L1) with becacizumab (an anti VEGF agent that starves the tumor of blood and also has immunomodulatory effects, *(Majidpoor J and Mortezaee, 2021).* The primary approaches leverage the immune system's components to target the tumor within the liver's unique, often immune-suppressive, microenvironnement, *Sperandio RC et al (2022)*.

Chemotherapy a type of cancer treatment that uses drugs to destroy cancer cells, *Zhen, J. et al. (2017),* it works by stopping or showing the growth of cancer cells, which grow and divide quickly, it harms healthy cells that divide quickly, *Scheiner B. et al. (2022).* Depending on the type of cancer and how advanced it is, chemotherapy can cure or control cancer or ease symptoms caused by cancer, *Bruix J. et al. (2017)*. It cures cancer by destroying cancer cells to the point that the Doctor can no longer detect them in the body, *Ahamaraj S. et al. (2017).* It is a type of treatment that uses drugs to attack cancer, *Zheng J. et al. (2017).* including HCC. These drugs work by preventing the growth and division of the cancer cells, *Kim H.S. et al. (2000)*. Chemotherapy has been used as a treatment for HCC for many years, and it remains an important treatment option despite the addition in recent years of news types of treatment, *Decaens T. et al. (2022).* The goal of chemotherapy is to destroy cancer cells, Mercer C.A. et al. (2009). traditional

chemotherapies work by killing cells that divide rapidly, Klion Sky D.J. (2022). Damage to healthy blood cells lead to side effects such as fatigue, or infection, Boya, P. et al. (2013). Chemotherapy can also damage the cells that line mucous membranes, these drugs may be used alone or in combination with other chemotherapy drugs or other types of cancer treatments such as immunotherapy and targeted therapy, *Komatsu, M.(2011).*

Locoregional Therapies (LRTs) for HCC are image – guided, tumor directed procedures designed to destroy tumors, prevent progression and bridge patients to transplant, acting as the standard of care for early (ablation) and intermediate stages, *Weis, S.M. & Cheresh, D.A. (2000)*. They play a crucial role in HCC management and are selectively adopted on technical aspects, *Lui, Z.L. et al. (2023)*. patient clinical status and tumor characteristics, previous studies have consistently highlighted the efficacy of combining LRTs with molecular targeted agents in HCC treatment, *Morse, M.A. et al. (2019).* Recent studies propose that integrating LRTs with immune checkpoint inhibitors and molecular targeted agents could provide substantial therapeutic benefits, *Kong, F.H. et al. (2021)*, a notion underpinned by both basic and clinical evidence, *Melincivci, C.S. et al. (2018)*.

LRTs, pivotal in HCC management, encompass tumour targeted procedures under imaging guidance, generally categorised into the percutaneous approach and the intra-arterial approach, *Chu J.S. et al. (2013)*. As a result, various percutaneous, mage – guided, locoregional therapies have emerged in order to improve outcomes, initially among inoperable patients, *Sharma, A. et al. (2020)*. After decades of thorough investigation and clinical experience in the field of interventional oncology, numerous minimal invasive treatment options have been developed and include curative modalities such as percutaneous radiofrequency ablation (RFA), *Xu, Z.H et al.(2024),* microwave Ablation (MWA), percutaneous ethanol injection (PEI), cryoablation (CA), irreversible electroporation (IRE) and palliative therapies such as bland trans – arterial embolization (TAE), conventional Transarterial Chemoembolization (Ctace) or chemoembolization with drug – electing beads and more recently local endovascular radiotherapy via the trans – arterial delivery of beta emitting microparticles, *Torimuror, T. et al. (2016)*. Radiation therapy such as Stereotactic Body Radiotherapy (SERT), utilized for localized, unresectable tumors, particularly when they are not amenable to other ablative techniques, *Wang, Z et al. (2018).* CT – guided brachytherapy is used like emerging technique for large central tumors, *Pan, Y.X. et al. (2021).*

3. MATERIALS & METHODS

It is descriptive study done on the prevalence of cancer death occurred to the HCC patients (187 men and 84 women) under treatment at the Intensive Care Unit of Bertoua Regional Hospital from June to September 2025 after receiving ethical clearance and Research Authorization from Est Public Health Delegation – Cameroon. The analysis included chemotherapy, immunotherapy, locoregional therapies, liver transplantation and surgical resection. Data were collected using structured questionnaires, observations and analyzed using descriptive statistics and analyses. The analysis included chemotherapy, immunotherapy, locoregional therapies, liver transplantation and surgical resection. Data were collected using structured

questionnaires, observations and analyzed using descriptive statistics and analyses

Convenience sampling was done, all the cases of elective HCC admitted during the study period were included. Age > 20, men and women, Cameroonians or not but residents in Cameroon. The simple size was 271 patients calculated was calculated using the formula $n = (Z^2 \times p \times q) / e^2$ where n = minimum sample size, z= confidence interval, p= prevalence of decompensated cirrhosis, q= 1 – p and e= margin of error, 3%.

The data for this study were collected using structured questionnaires, entered and analysed in Microsoft Excel 2016, confidence interval was calculated along with frequency and percentages for binary data

4. RESULTS & DISCUSSION

This part analyzes results concerning treatment used to treat HCC patients as well as the liver metastasis at the Bertoua Regional Hospital.

[See Annex — Table 1: HCC Patients Characteristics]

[See Annex — Table 2 : Evaluation of Curative Therapies]

[See Annex — Table 3: Evaluation of Systemic Therapies]

[See Annex — Table 4: Evaluation of prevalence of Liver Metastasis and Cancer Death]

Despite Sorafenib 200mg given to the 28,04% of 271 HCC patients, 45,38% has developed liver metastasis and 25,46% are died.8,48% in the Intensive Care Unit. Any others therapies used to treat patient such as immunotherapy, surgical resection, liver transplantation, locoregional therapies and local ablations. The majority of patients were Cameroonians with 61,99%. The Hospital indicated that there are shortages of sorafenib 200 mg in the Pharmacy. Hepatocellular carcinoma (HCC) is highly aggressive, and the development of metastasis or recurrence is a primary cause of death in patients under treatment. Approximately 50-60% of patients are diagnosed at or progress to an advanced stage, with a median overall survival (mOS) of 6-8 months in such cases, Siegel RH et al,..(2022). Liver metastasis in Hepatocellular Carcinoma (HCC) patients—specifically the spread to other areas of the liver or the development of new,Tarao K et al,..(2019) distant intrahepatic lesions during treatment is highly common, as the liver is the primary site of both tumor recurrence and metastatic spread, Sung H et al,..(2020) While the prevalence of extrahepatic metastasis (metastasis outside the liver) is around 10–15%, the rate of intrahepatic recurrence (new metastasis within the liver) after curative treatment is very high, with reports of up to 60–70% within five years, Kanwal F et al,..(2020).

5. CONCLUSION

Hepatocellular carcinoma (HCC) treatment failure often stems from advanced stage at diagnosis, underlying liver dysfunction (cirrhosis), and high tumor recurrence rates after ablation or resection. Common causes include poor response to systemic therapy, drug resistance, and comorbidities preventing further treatment. The prognosis for advanced HCC remains poor, with low 5-year survival rates, necessitating multidisciplinary. Avoiding failure in hepatocellular carcinoma (HCC) treatment requires a multidisciplinary approach focused on early detection, precise staging, effective management of underlying liver disease, and utilizing tailored treatments, particularly in the context of preventing high recurrence rates.

REFERENCES

Jonas M, Kelly DA, Mizerski J, et al. Clinical trial of lamivudine in children with chronic hepatitis B. N Engl J Med 2002;346:1706–13.

Lai CL, Lin HJ, Lau HJ, et al. Effect of recombinant a2 IFN with or without prednisolone in Chinese HBsAg carrier children. QJM 1991;78:155–63.

Bortolotti F, Jara P, Barbera C, et al. Long term effect of a interferon in children with chronic hepatitis B. Gut 2000; 46:715–8

Lampertico P, Del Ninno E, Vigano M, et al. Long term suppression of HBeAg negative chronic hepatitis B by 24 months’ interferon therapy. Hepatol 2003;37:756–63.

Kasper D.L., Fauci A.S., Longo D.L, et al: Harrison’s principles of internal medicine. 16th edition. 2005.

Keeffe EB, Dieterich DT, Han SH, Jacobson IM, Martin P, Schiff ER, et al. A treatment algorithm for the management of chronic hepatitis B virus infection in the United States: 2008 update. Clin Gastroenterol Hepatol. Dec 2008;6(12):1315-41; quiz 1286.

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology. May 2009;49(5 Suppl):S72-84

Fattovich G, Stroffolini T, Zagni I, et al. Hepatocellular carcinoma in cirrhosis: incidence and risk factors. Gastroenterology 2004; 127(Suppl 1):S35–S50.

McMahon BJ, Alberts SR, Wainwright RB, et al. Hepatitis B-related sequelae. Prospective study in 1400 hepatitis B surface antigenpositive Alaska native carriers. Arch Intern Med 1990;150:1051– 1054.

Mizokami M, Orito E. Molecular evolution of hepatitis viruses. Intervirology 1999;42:159–165

Tanaka Y, Kurbanov F, Mano S, et al. Molecular tracing of the global hepatitis C virus epidemic predicts regional patterns of hepatocellular carcinoma mortality. Gastroenterology 2006;130: 703–714.

El-Serag HB, Rudolph KL. Hepatocellular carcinoma: epidemiology and molecular carcinogenesis. Gastroenterology 2007;132: 2557–2576.

Chang MH. Hepatitis B virus and cancer prevention. Recent Results Cancer Res 2011;188:75–84

Fattovich G, Bortolotti F, Donato F. Natural history of chronic hepatitis B: special emphasis on disease progression and prognostic factors. J Hepatol 2008;48:335–352

Yang JD, Kim WR, Coelho R, et al. Cirrhosis is present in most patients with hepatitis B and hepatocellular carcinoma. Clin Gastroenterol Hepatol 2011;9:64–70

Yang HI, Lu SN, Liaw YF, et al. Hepatitis B e antigen and the risk of hepatocellular carcinoma. N Engl J Med 2002;347:168–174

Chen CJ, Yang HI, Iloeje UH. Hepatitis B virus DNA levels and outcomes in chronic hepatitis B. Hepatology 2009;49(Suppl): S72–S84.

Chun HM, Roediger MP, Hullsiek KH, et al. Hepatitis B Virus Coinfection Negatively Impacts HIV Outcomes in HIV Seroconverters. The Journal of Infectious Diseases. 2012;205(2):185-193. doi:10.1093/infdis/jir720

McMahon BJ. The natural history of chronic hepatitis B virus infection. Hepatology. 2009;49(5 Suppl):S45-55 2.

Schweitzer A,Horn J, Mikolajczyk RT, Krause G, Ott JJ Estimations of worldwide prevalence of chronic hepatitis B virus infection: a systematic review of data published between 1965 and 2013.

Lancet 2015; 386: 1546–55 3. Ott J, Stevens G, Groeger J, Wiersma S. Global epidemiology of Hepatitis B virus infection: new estimates of age-specific HBsAg seroprevalence and endemicity. Vaccine 2012;30(12): 2212–9 4.

Lukhwareni A, Burnett RJ, Selabe SG, Mzileni MO, Mphahlele MJ. Increased detection of HBV DNA in HBsAg-positive and HBsAgnegative SouthAfrican HIV/AIDS patients enrolling for Highly active antiretroviral therapy at a Tertiary Hospital. J Med Virol 2009;81(3):406–12 5.

Boyles TH, Cohen K. The prevalence of hepatitis B infection in a rural SouthAfrican HIV clinic. S Afr Med J 2011;101(7):470–1 6.

Firnhaber C, Reyneke A, Schulze D, et al. The prevalence of hepatitis B co-infection in a South African urban government HIV clinic. S Afr Med J 2008;98(7):541-4 7.

Burnett RJ, Ngobeni JM, Francois G, et al. Increased exposure to hepatitis B virus infection in HIV-positive South African antenatal women. Int J STD AIDS 2007;18(3):152–6 8.

Andersson MI, Maponga TG, Ijaz S, et al. The epidemiology of hepatitis B virus infection in HIV-infected and HIV-uninfected pregnant women in the Western Cape, South Africa. Vaccine 2013;31(47):5579-84 9.

Puoti M1, Torti C, Bruno R, Filice G, Carosi G. Natural history of chronic hepatitis B in co-infected patients. J Hepatol 2006; 44(1 Suppl):S65-70 10.

Maynard JE. Hepatitis B: Global importance and need for control. Vaccine 1990;8(SUPPL.):S18-S20

Mohd Hanafiah K, Groeger J, Flaxman AD, Wiersma ST. Global epidemiology of hepatitis virus infection: new estimates of age-specific antibody to HCV seroprevalence. Hepatology 2013;57(4):1333-42

Lavanchy D. The global burden of hepatitis C. Liver Int. 2009;29 Suppl 1:74-Ellis LA, Brown D, Conradie JD, Paterson A, Sher R, Millo J, et al. Prevalence of hepatitis C inSouth Africa: detection of anti-HCV in recent and stored serum. J Med Virol 1990;32(4):249- 51

Davis GL. Hepatitis C virus genotypes and quasispecies. Am J Med 1999;107(6B):21S-6S

Messina JP, Humphreys I, Flaxman A, et al. Global distribution and prevalence of hepatitis C virus genotypes. Hepatology. 2015;61(1):77–87

Benova L, Mohamoud YA, Calvert C, AbuRaddad LJ. Vertical transmission of hepatitis C virus:

systematic review and meta-analysis. Clin Infect Dis. 2014;59(6):765–73

Floreani A. Hepatitis C and pregnancy. World J Gastroenterol. 2013;19(40):6714–20 Jafari S, Copes R, Baharlou S, Etminan M, Buxton J. Tattooing and the risk of transmission of hepatitis C: a systematic review and metaanalysis. Int J Infect Dis.2010;14(11):E928– E9

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Zein CO, Levy C, Basu A, Zein NN. Chronic hepatitis C and type II diabetes mellitus: a prospective cross-sectional study. Am J Gastroenterol. 2005;100(1):48-5

EASL Recommendations on Treatment of Hepatitis C 2018. J Hepatol 2018; 69(2): 461- 511

Smith JO, Sterling RK. Systematic review: non-invasive methods of fibrosis analysis in chronic hepatitis C. Alimentary pharmacology & therapeutics. 2009;30(6):557-76

Karmochkine M, Carrat F, Dos Santos O, Cacoub P, Raguin G. A case-control study of risk factors for hepatitis C infection in patients with unexplained routes of infection. J Viral Hepat. 2006;13(11):775–82

Nelson PK, Mathers BM, Cowie B, et al. Global epidemiology of hepatitis B and hepatitis C in people who inject drugs: results of systematic reviews. Lancet. 2011;378(9791):571–83

Lau GK, Piratvisuth T, Luo KX, Marcellin P, Thongsawat S, et al. (2005) Peginterferon Alfa-2a, lamivudine, and the combination for HBeAgpositive chronic hepatitis B. N Engl J Med 352: 2682-2695. 2.

Conjeevaram HS, Lok AS (2003) Management of chronic hepatitis B. J Hepatol 38 Suppl 1: S90-103.

Karayiannis P (2003) Hepatitis B virus: old, new and future approaches to antiviral treatment. J Antimicrob Chemother 51: 761-785.

Wellington K, Jarvis B (2001) Silymarin: a review of its clinical properties in the management of hepatic disorders. BioDrugs 15: 465-489.

Strader DB, Bacon BR, Lindsay KL, La Brecque DR, Morgan T, et al. (2002) Use of complementary and alternative medicine in patients with liver disease. Am J Gastroenterol 97: 2391-2397.

Eisenberg DM, Davis RB, Ettner SL, Appel S, Wilkey S, et al. (1998) Trends in alternative medicine use in the United States, 1990-1997: results of a follow-up national survey. JAMA 280: 1569-1575.

Flora K, Hahn M, Rosen H, Benner K (1998) Milk thistle (Silybum marianum) for the therapy of liver disease. Am J Gastroenterol 93: 139-143.

Varga Z, Czompa A, Kakuk G, Antus S (2001) Inhibition of the superoxide anion release and hydrogen peroxide formation in PMNLs by flavonolignans. Phytother Res 15: 608-612.

Dehmlow C, Erhard J, de Groot H (1996) Inhibition of Kupffer cell functions as an explanation for the hepatoprotective properties of silibinin. Hepatology 23: 749-754

Wilasrusmee C, Kittur S, Shah G, Siddiqui J, Bruch D, et al. (2002) Immunostimulatory effect of Silybum Marianum (milk thistle) extract. Med Sci Monit 8: BR439-443.

Neumann UP, Biermer M, Eurich D, Neuhaus P, Berg T (2010) Successful prevention of hepatitis C virus (HCV) liver graft reinfection by silibinin mono-therapy. J Hepatol 52: 951-952.

Beinhardt S, Rasoul-Rockenschaub S, Maieron A, PH S-M, Hofer H PF (2012) Intravenous Silibinin-therapy in patients with chronic hepatitis C in the transplant setting. J Hepatology 56: S77.

Pares A, Planas R, Torres M, Caballeria J, Viver JM, et al. (1998) Effects of silymarin in alcoholic patients with cirrhosis of the liver: results of a controlled, double-blind, randomized and multicenter trial. J Hepatol : 615-621.

Sepanlou SG, Safiri S, Bisignano C, et al; GBD 2017 Cirrhosis Collaborators. The global, regional, and national burden of cirrhosis by cause in 195 countries and territories, 1990-2017: a systematic analysis for the Global Burden of Disease Study 2017. Lancet Gastroenterol Hepatol. 2020;5(3):245-266. doi:10.1016/S2468-1253(19) 30349-8

Tapper EB, Parikh ND. Mortality due to cirrhosis and liver cancer in the United States, 1999-2016. BMJ. 2018;362:k2817. doi:10.1136/bmj.k2817

Centers for Disease Control and Prevention. CDC WONDER: about provisional mortality statistics, 2018 through last month. Accessed March 13, 2023. https://wonder.cdc.gov/mcd-icd10-provisional. html

Serper M, Kaplan DE, Taddei TH, Tapper EB, Cohen JB, Mahmud N. Nonselective beta blockers, hepatic decompensation, and mortality in cirrhosis: a national cohort study. Hepatology. 2023;77(2): 489-500. doi:10.1002/hep.32737

Flemming JA, Dewit Y, Mah JM, Saperia J, Groome PA, Booth CM. Incidence of cirrhosis in young birth cohorts in Canada from 1997 to 2016: a retrospective population-based study. Lancet Gastroenterol Hepatol. 2019;4(3):217-226. doi:10. 1016/S2468-1253(18)30339-X

Hirode G, Saab S, Wong RJ. Trends in the burden of chronic liver disease among hospitalized US adults.JAMA Netw Open. 2020;3(4):e201997-e201997. doi:10.1001/jamanetworkopen.2020.1997

Foster C, Baki J, Nikirk S, Williams S, Parikh ND, Tapper EB. Comprehensive health-state utilities in contemporary patients with cirrhosis. Hepatol Commun. 2020;4 (6):852-858. doi:10.1002/hep4. 1512

Asrani SK, Hall L, Hagan M, et al. Trends in chronic liver disease-related hospitalizations: a population-based study. Am J Gastroenterol. 2019;114(1):98-106. doi:10.1038/s41395-018- 0365-4

Tarao K, Nozaki A, Ikeda T, et al. Real impact of liver cirrhosis on the development of hepatocellular carcinoma in various liver diseases-meta-analytic assessment. Cancer Med. 2019;8(3):1054-1065. doi:10.1002/cam4.1998

Carbonell N, Pauwels A, Serfaty L, Fourdan O, Lévy VG, Poupon R. Improved survival after variceal bleeding in patients with cirrhosis over the past two decades. Hepatology. 2004;40(3):652-659. doi: 10.1002/hep.20339

Arvaniti V, et al. Infections in patients with cirrhosis increase mortality four fold and should be used in determining prognosis. Gastroenterology 2010;139:1246-1256.

Berzigott A., et al. Obesity is an independent risk factor for clinical decompensation in patients with cirrhosis. Hepatology 2011;54:555-561.

Dienstag J.L., et al. A prospective study of progression in compensated, histologically advanced chronic hepatitis C. Hepatology 2011;54:396-405.

Durand F, et al. Assessment of the prognosis of cirrhosis: Child-Pugh versus MELD. Journal of Hepatology2005;42.

Franchis de, et al. Non-invasive diagnosis of cirrhosis and the natural history of it complications. Best Practice & Research Clinical Gastroenterology 2007;21(1):3-18.

Friedman SL. Mechanisms of hepatic fibrogenesis. Gastroenterology 2008;134:1655-1669.

Garcia-Tsao G, et al. Management and treatment of patients with cirrhosis and portal hypertension: recommendations from the Department of Veterans Affairs Hepatitis C Resource Center Program and the National Hepatitis C program. The American Journal of Gastroenterology 2009;104(7):1802-1829

Greenbaum L.E. and Wells R.G. The role of stem cells in liver repair and fibrosis. The International Journal of Biochemistry and Cell Biology 2011;43:222-229.

Jiao J, et al. Hepatic fibrosis. Current Opinion in Gastroenterology 2009;25(3):223-229.

Kanwal F, et al. An explicit quality indicator set for measurement of quality of care in patients with cirrhosis. Clinical Gastroenterology and Hepatology 2010;8:709

Sung H, Ferlay J, Siegel RL, et al. Global Cancer Statistics 2020: GLOBOCAN estimates of incidence and mortality worldwide for 36 cancers in 185 countries. CA Cancer J Clin 2021; 71: 209–49.

Siegel RL, Miller KD, Fuchs HE, Jemal A. Cancer statistics, 2022. CA Cancer J Clin 2022; 72: 7–33. 3 Petrick JL, Florio AA, Znaor A, et al. International trends in hepatocellular carcinoma incidence, 1978–2012. Int J Cancer 2020; 147: 317–30.

Sun J, Althoff KN, Jing Y, et al. Trends in hepatocellular carcinoma incidence and risk among persons with HIV in the US and Canada, 1996–2015.

JAMA Netw Open 2021; 4: e2037512. 5 Chen T, Liu J, Li Y, Wei S. Burden of disease associated with dietary exposure to aflatoxins in China in 2020. Nutrients 2022; 14: 1027.

Romano A, Angeli P, Piovesan S, et al. Newly diagnosed hepatocellular carcinoma in patients with advanced hepatitis C treated with DAAs: a prospective population study. J Hepatol 2018; 69: 345–52.

Dave S, Park S, Murad MH, et al. Comparative effectiveness of entecavir versus tenofovir for preventing hepatocellular carcinoma in patients with chronic hepatitis B: a systematic review and meta-analysis. Hepatology 2021; 73: 68–78.

Papatheodoridis GV, Idilman R, Dalekos GN, et al. The risk of hepatocellular carcinoma decreases after the first 5 years of entecavir or tenofovir in Caucasians with chronic hepatitis B. Hepatology 2017; 66: 1444–53.

Chiang CJ, Yang YW, You SL, Lai MS, Chen CJ. Thirty-year outcomes of the national hepatitis B immunization program in Taiwan. JAMA 2013; 310: 974–76.

Kanwal F, Kramer JR, Asch SM, Cao Y, Li L, El-Serag HB. Long-term risk of hepatocellular carcinoma in HCV patients treated with direct acting antiviral agents. Hepatology 2020; 71: 44–55.

Kim HY, Lampertico P, Nam JY, et al. An artificial intelligence model to predict hepatocellular carcinoma risk in Korean and Caucasian patients with chronic hepatitis B. J Hepatol 2022; 76: 311–18.

Kim HS, Yu X, Kramer J, et al. Comparative performance of risk prediction models for hepatitis B-related hepatocellular carcinoma in the United States. J Hepatol 2022; 76: 294–301.

Zheng J, Seier K, Gonen M, et al. Utility of serum inflammatory markers for predicting microvascular invasion and survival for patients with hepatocellular carcinoma. Ann Surg Oncol 2017; 24: 3706–14.

Johnson PJ, Dhanaraj S, Berhane S, Bonnett L, Ma YT. The prognostic and diagnostic significance of the neutrophil-tolymphocyte ratio in hepatocellular carcinoma: a prospective controlled study. Br J Cancer 2021; 125: 714–16.

Bruix J, Cheng AL, Meinhardt G, Nakajima K, De Sanctis Y, Llovet J. Prognostic factors and predictors of sorafenib benefit in patients with hepatocellular carcinoma: analysis of two phase III studies. J Hepatol 2017; 67: 999–1008.

Scheiner B, Pomej K, Kirstein MM, et al. Prognosis of patients with hepatocellular carcinoma treated with immunotherapy—development and validation of the CRAFITY score. J Hepatol 2022; 76: 353–63. 87 Vogel A, Martinelli E, ESMO Guidelines Committe. Updated treatment recommendations for hepatocellular carcinoma (HCC) from the ESMO Clinical Practice guidelines. Ann Oncol 2021; 32: 801–05.

Bruix J, Chan SL, Galle PR, Rimassa L, Sangro B. Systemic treatment of hepatocellular carcinoma: an EASL position paper. J Hepatol 2021; 75: 960–74.

The International Liver Cancer Association. ILCA systemic therapy guidance. https://ilca-online.org/wp-

content/uploads/2020/06/Systemic-therapy-guidelines-V1.2.pdf (accessed July 26, 2022).

Kudo M, Kawamura Y, Hasegawa K, et al. Management of hepatocellular carcinoma in Japan: JSH consensus statements and recommendations 2021 update. Liver Cancer 2021; 10: 181–223.

Chen LT, Martinelli E, Cheng AL, et al. Pan-Asian adapted ESMO clinical practice guidelines for the management of patients with intermediate and advanced/relapsed hepatocellular carcinoma: a TOS-ESMO initiative endorsed by CSCO, ISMPO, JSMO, KSMO, MOS and SSO. Ann Oncol 2020; 31: 334–51.

Reig M, Forner A, Rimola J, et al. BCLC strategy for prognosis prediction and treatment recommendation: the 2022 update. J Hepatol 2021; 76: 681–93.

Lang H, Sotiropoulos GC, Dömland M, et al. Liver resection for hepatocellular carcinoma in non-cirrhotic liver without underlying viral hepatitis. Br J Surg 2005; 92: 198–202.

Viganò L, Conci S, Cescon M, et al. Liver resection for hepatocellular carcinoma in patients with metabolic syndrome: a multicenter matched analysis with HCV-related HCC. J Hepatol 2015; 63: 93–101.

Kabir T, Tan ZZ, Syn NL, et al. Laparoscopic versus open resection of hepatocellular carcinoma in patients with cirrhosis: meta analysis. Br J Surg 2021; 109: 21–29. 96 Ivanics T, Claasen MP, Patel MS, et al. Long-term outcomes of laparoscopic liver resection for hepatocellular carcinoma: a propensity score matched analysis of a high-volume North American center. Surgery 2021; 171: 982–91.

Berzigotti A, Reig M, Abraldes JG, Bosch J, Bruix J. Portal hypertension and the outcome of surgery for hepatocellular carcinoma in compensated cirrhosis: a systematic review and meta - analysis. Hepatology 2015; 61: 526–36.

Berardi G, Morise Z, Sposito C, et al. Development of a nomogram to predict outcome after liver resection for hepatocellular carcinoma in Child-Pugh B cirrhosis. J Hepatol 2020; 72: 75–84.

Azoulay D, Ramos E, Casellas-Robert M, et al. Liver resection for hepatocellular carcinoma in patients with clinically significant portal hypertension. JHEP Rep 2020; 3: 100190.

Citterio D, Facciorusso A, Sposito C, Rota R, Bhoori S, Mazzaferro V. Hierarchic interaction of factors associated with liver decompensation after resection for hepatocellular carcinoma. JAMA Surg 2016; 151: 846–53.

Serenari M, Han KH, Ravaioli F, et al. A nomogram based on liver stiffness predicts postoperative complications in patients with hepatocellular carcinoma. J Hepatol 2020; 73: 855–62.

Allaire M, Goumard C, Lim C, Le Cleach A, Wagner M, Scatton O. New frontiers in liver resection for hepatocellular carcinoma. JHEP Rep 2020; 2: 100134.

Marasco G, Alemanni LV, Colecchia A, et al. Prognostic value of the albumin-bilirubin grade for the prediction of post-hepatectomy liver failure: a systematic review and meta-analysis. J Clin Med 2021; 10: 2011.

Wang, Z. et al. Adjuvant transarterial chemoembolization for HBV-related hepatocellular carcinoma after resection: a randomized controlled study. Clin. Cancer Res. 24, 2074–2081 (2018).

Wahl DR, Stenmark MH, Tao Y, et al. Outcomes after stereotactic body radiotherapy or radiofrequency ablation for hepatocellular carcinoma. J Clin Oncol. 2016;34(5):452-459. doi:10.1200/JCO.2015.61.4925

Wong H, Tang YF, Yao TJ, Chiu J, Leung R, Chan P, Cheung TT, Chan AC, Pang RW, Poon R, et al. The outcomes and safety of single-agent sorafenib in the treatment of elderly patients with advanced hepatocellular carcinoma (HCC) Oncologist. 2011;16:1721–1728. doi: 10.1634/theoncologist.2011-0192. [DOI] [PMC free article] [PubMed] [Google Scholar]

Wang Z, Ren Z, Chen Y, et al. Adjuvant transarterial chemoembolization for HBV-related hepatocellular carcinoma after resection: a randomized controlled study. Clin Cancer Res. 2018;24(9):2074-2081. doi:10.1158/1078-0432.CCR-17-2899

Weis, S. M. & Cheresh, D. A. Tumor angiogenesis: molecular pathways and therapeutic targets. Nat. Med. 17, 1359–1370 (2011).

WangL,Jia D, Duan F, Sun Z, Liu X, Zhou L, etal. Combined anti-tumor effects of ifn-a and sorafenib on hepatocellular carcinoma in vitro and in vivo. Biochem Biophys Res Commun (2012) 422(4):687–92. doi: 10.1016/j.bbrc.2012.05.056

Yorimitsu T, Klionsky DJ. Autophagy: molecular machinery for self-eating. Cell Death Differ. 2005;12:1542–1552. doi: 10.1038/sj.cdd.4401765. [DOI] [PMC free article] [PubMed] [Google Scholar]

Yang JD, Mohamed EA, Aziz AOA, Shousha HI, Hashem MB, Nabeel MM, et al. Characteristics, management, and outcomes of patients with hepatocellular carcinoma in Africa: a multicountry observational study from the Africa Liver Cancer Consortium. Lancet Gastroenterol Hepatol. (2017) 2:103–11. doi: 10.1016/S2468-1253(16)30161-3

Yoshida, H. et al. Effect of vitamin K2 on the recurrence of hepatocellular carcinoma. Hepatology 54, 532–540 (2011).

Zhang, N. & Li, Y. Receptor tyrosine kinases: biological functions and anticancer targeted therapy. MedComm. 4, e446 (2023).

Zhao Y Li Z. Interplay of estrogen receptors and FOXA factors in the liver cancer. Mol Cell Endocrinol. 2015;418:334-339

Zhang B-H, Yang B-H, Tang Z-Y. Randomized controlled trial of screening for hepatocellular carcinoma. J Cancer Res Clin Oncol 2004;130:417-422.

Zheng K, Zhu X, Fu S, et al. Sorafenib plus hepatic arterial infusion chemotherapy versus sorafenib for

hepatocellular carcinoma with major portal vein tumor thrombosis: a randomized trial. Radiology. 2022;303(2):455-464. doi:10.1148/radiol.211545

Zhu W, Peng Y, Wang L, Hong Y, Jiang X, Li Q, et al. Identification of a Fetoprotein-Specific T-cell receptors for hepatocellular carcinoma immunotherapy. Hepatology (2018) 68(2):574–89. doi: 10.1002/hep.29844

Zander R, Schauder D, Xin G, Nguyen C, Wu X, Zajac A, et al. Cd4(+) T cell help is required for the formation of a cytolytic Cd8(+) T cell subset that protects against chronic infection and cancer. Immunity (2019) 51(6):1028–42.e4. doi: 10.1016/ j.immuni.2019.10.009

Zhang B, Shi X, Cui K, et al. Real-world practice of conversion surgery for unresectable hepatocellular carcinoma—a single center data of 26 consecutive patients. BMC Cancer. 2023;23(1):465. doi:10.1186/s12885-023-10955-7

Zheng K, Zhu X, Fu S, et al. Sorafenib plus hepatic arterial infusion chemotherapy versus sorafenib for hepatocellular carcinoma with major portal vein tumor thrombosis: a randomized trial. Radiology. 2022;303(2):455-464. doi:10.1148/radiol.211545

Zheng X, Wu Y, Bi J, Huang Y, Cheng Y, Li Y, et al. The use of supercytokines, immunocytokines, engager cytokines, and other synthetic cytokines in immunotherapy. Cell Mol Immunol (2022) 19(2):192–209. doi: 10.1038/s41423-021 00786-6

Zou F, Tan J, Liu T, Liu B, Tang Y, Zhang H, et al. The Cd39(+) hbv surface protein-targeted car-T and personalized tumor-reactive Cd8(+) T cells exhibit potent anti-hcc activity. Mol Ther (2021) 29(5):1794–807. doi: 10.1016/j.ymthe.2021.01.021

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Olivier Lieuga , Tatiana Jiengoue Tchakonang , and Augustine Nji Asakizi (2026). The Prevalence of Liver Metastasis and Cancer Death to the HCC patient under treatment at the Bertoua Regional Hospital in Est Region Cameroon. IQ Research Journal, 5(2), IQRJ-V05I02-26004018. www.iqresearchjournal.com

ANNEXES

Annex I — Table 1: HCC Patients Characteristics

Prevalence Rate of HCC Patient Characteristics	Cameroonians		Chadians		Nigerians		Centrafric Republic		Others Nationalities	
	n	%	n	%	n	%	n	%	n	%
HCC Patients Tested Positive per Nationalities	168	61,99	22	8,11	32	11,80	26	9,59	23	8,48
Gender										
Male	115	61,49	15	8,02	23	9,62	18	9,62	16	8,55
Female	53	63,09	7	8,33	9	8,52	8	8,52	7	8,33
Age (Years Old)										
20 – 35	98	58,33	7	31,81	15	46,87	9	34,61	12	52,17
35 - 55	56	33,33	11	50	13	40,62	12	46,15	6	26,08
55 – Over	14	8,33	4	18,18	4	12,5	5	19,23	5	21,73
Marital Status										
Single	78	46,42	6	27,27	14	43,75	11	42,30	16	69,56
Married	27	16,07	9	40,90	9	28,12	6	23,07	3	13,04
Divorced	63	37,5	7	31,81	9	28,12	9	34,61	4	17,39
Education Levels										
Primary	69	41,07	13	59,09	15	46,87	9	34,61	7	30,43
College	48	28,57	6	27,27	9	28,12	12	46,15	12	52,17
University	51	30,35	3	13,63	8	25	5	19,23	4	17,39
Employment										
Unemployed	72	42,85	6	27,27	8	25	10	38,46	7	30,43

Student	15	8,92	5	22,72	7	21,87	6	23,07	5	21,73
Employed	13	7,73	2	9,09	4	12,5	5	19,23	3	13,04
Self – Employed	47	27,97	3	13,63	3	9,37	3	11,53	7	30,43
Retired	21	12,5	4	18,18	10	31,25	2	7,69	1	4,34

Annex II — Table 2 : Evaluation of Curative Therapies

Curative Therapies	Surgical Resection		Liver Transplantation		Chemoembolization		Radioembolization		Local Abblation	
	n	%	n	%	n	%	n	%	n	%
Patient	0	0	0	0	0	0	0	0	0	0

Annex III — Table 3: Evaluation of Systemic Therapies

Result of Systemic Therapies	Sorafenib		Regorafenib		Nivolumab		Traditional Drugs		Ipilimumab	
	n	%	n	%	n	%	n	%	n	%
Patient	76	28,04	0	0	0	0	0	0	0	0

Annex IV — Table 4: Evaluation of prevalence of Liver Metastasis and Cancer Death

Result of Liver Metastasis and Cancer Death	Liver Metastasis		Cancers Death		Intensive Care Unit	
	n	%	n	%	n	%
Patient	123	45,38	69	25,46	23	8,48

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Profil Radio-Clinique et Electrique des Hypertrophies Ventriculaires Gauches à l'Hôpital Militaire de Ngaoundéré Cameroun

Yene Zang Bernard Severin[a,b] , Samen Sileu Chimène Nathalie[a]

Affiliations

s. [a] Hôpital Militaire de Ngaoundéré, Cameroon

t. [b]School of Health and Biomedical Sciences, Kesmonds International University of America

Corresponding Author:

Yene Zang Bernard Severin

Email: sahelseverin@gmail.com

Paper ID: IQRJ-V05I02-26005019

ABSTRACT

L'hypertrophie ventriculaire gauche se définit par une augmentation de la masse ventriculaire gauche laquelle comprend la masse des cardiomyocytes, ainsi que Celle des tissus interstitiels qui la composent. Elle est responsable des troubles de rythme, d'angor fonctionnel, d'anomalies de la compliance ventriculaire pouvant évoluer vers une insuffisance cardiaque. L'objectif notre étude était d'établir le profil Radio-Clinique et Electrique des patients atteints d'hypertrophies ventriculaires gauches. Il s'agit d'une étude transversale, descriptive et analytique avec recrutement prospectif, à l'hôpital militaire de Ngaoundéré. Étaient inclus, les patients des deux sexes, consentent de participer mais ayant réalisé une Radiographie du thorax, un électrocardiogramme, une échographie et présentant une HVG en échocardiographie. Les données recueillies étaient Anthropométriques, cliniques, radiographiques, électriques et écho cardiographiques. Nous avons colligé 34 dossiers répondant à nos critères d'inclusions, ils venaient de plusieurs régions du Cameroun dont 61.8% résidaient dans la région de l'étude avec une prédominance urbaine 67.6%. La tranche d'âge la plus représentative était celle de 60 à 70 ans. Le sexratio était de 1.1 avec une prédominance masculine 52.9%. La couche sociale la plus affecté par l'HVG est celle des ménagères 44.1%, la plupart des patients atteints d'HVG se sont consulter pour la dyspnée. L'HTA était la principale étiologie. L'HTA était de grade I représentative dans 42,6% des cas. A la radiographie 85% des patients ayant une d'HVG échographique présentaient une cardiomégalie, avec une tendance OR= 0.2 et une association statiquement non significative (P >0.05), le signe récurant d'HVG était l'enfoncement de l'apex du ventricule gauche dans le diaphragme 58.8%. L'ECG présente un rythme cardiaque sinusale 91.1%, une fibrillation atriale chez 23.5% des patients, la durée de QRS était normale 79.4%, avec un axe normal de 61.8%. Une proportion 82% des patients avaient un indice de Sokolow Lyon supérieure à 35mm et présentaient également une HVG à l'écho cardiographique ainsi l'association était statistiquement significative (P< 0.001). Avec une HVG modérée dans 47% des cas. L'ECG révèle 61.7% des anomalies du segment ST-T antérolatérale. Les manifestations de l'HVG sont présentes en radiographie, l'ECG et à l'échocardiographie d'ailleurs, les différentes prévalences obtenues, sont 30.9% à l'ETT. 25.45% à l'ECG et 26.4% à la radiographie. Elles se traduisent par un ensemble d'aspects radiographiques, électriques et écho cardiographique. L'ETT reste le diagnostic de référence par rapport à l'ECG mais toutes ces caractéristiques nous ont permis d'établir le profil radio-clinique et électrique des hypertrophies ventriculaires à l'hôpital militaire de Ngaoundéré dont sa connaissance permet d'améliorer la prise en charge des HVG.

Keywords : *SeroPrevalence, Viral Hepatitis B and C, Pregnant women, Cameroon*

1. INTRODUCTION

Le corps humain est un organisme complexe composé de milliards de cellules qui, ensemble, forment des tissus, des organes, les appareils et les systèmes. Ces cellules peuvent subir des altérations fonctionnelles ou des désorganisations au cours de leur cycle évolutif. Parmi celles-ci figurent les pathologies du système cardiovasculaire. Les maladies cardiovasculaires (MCV) représentent un problème majeur de santé publique en raison de leur fréquence et leur gravité croissantes. Elles constituent aujourd'hui la principale cause de décès dans le monde. En 2008, l'organisation mondiale de la santé (OMS) estimait à 17.3 millions de nombres de décès lié aux MCV, et ce chiffre devrait atteindre 23.6 millions en 2030 **(DT NAIBE et al (2016).** Parmi les facteurs de risques indépendants d'évènements cardiovasculaires, on retrouve les hypertrophies ventriculaires gauches

(HVG). L'hypertrophie ventriculaire gauche se définit par une augmentation de la masse ventriculaire gauche laquelle comprend la masse des cardiomyocytes, ainsi que Celle des tissus interstitiels qui la composent. Elle est responsable des troubles de rythme, d'angor fonctionnel, d'anomalies de la compliance ventriculaire pouvant évoluer vers une insuffisance cardiaque **(DJIENTCHEU NGUEMALEU 1991)** A l'échelle mondiale, la prévalence de l'HVG varie selon la méthode de diagnostic. En échocardiographie, elle est estimée entre 35 - 40 % chez des patients hypertendus. En Europe, et particulièrement en France, sa prévalence varie de 7 .5% à

20.5% selon le critère de sokolow- Lyon, et de 11 à 25.4% pour le produit de corneille, elle varie de 14.9 à 75.5%

(NIAKARA et al 2001). En Afrique, la prévalence de l'HVG est plus élevée en raison de la forte charge des maladies cardiovasculaires, particulièrement l'hypertension artérielle non diagnostiquée ou mal contrôlée. Selon les données disponibles, de nos jours, sa prévalence qui de 28.4% en 2000 à 53.3% en 2024 chez les patients hypertendus Africain. Au Togo elle est de 46.2 % après évaluation par les indices de sokolow –Lyon et de Cornell A, 68 % en échocardiographie Chez les patients hypertendus. Chez les sujets obeses l'HVG est de 44.7%. Au Congo sa prévalence est définie en fonction de la nature de l'HVG, pour les HVG concentrique elle est de 25.5% et 45,3 % excentrique. Ces données peuvent aller au-delà dans les zones rurales ou l'accès aux soins est limité. Au Cameroun les études qui fournissent quelques informations sur la prévalence et la mortalité dues aux MCV sont pour la plupart rétrospectives et hospitalières selon PANCHA MBOUEMBOUE et al. 2015, sur une étude observationnelle portant sur des modifications électrocardiographiques et échographiques dans une population d'hypertendue camerounaise, l'HVG représente 55.5 % des MCV en échocardiographie et 22% en électrocardiographique (**OLIVIER PANCHA et al 2015)). Au** regard de la prévalence dans le monde, en Afrique et au Cameroun, l'HVG est un problème de santé publique. Dans le contexte du Cameroun ses données restent limitées particulièrement dans les régions comme Adamaoua et bien d'autres ou les infrastructures médicales, le manque de moyen financier et même la pénurie des médecins cardiologues restent un obstacle pour un diagnostic précis mais aussi, Les outils de diagnostic recensés dans la même région, comme la radiographie,

L'électrocardiographie, l'échocardiographie fournissent les diagnostics divergents. Ces réalités relevées du terrain camerounais expriment un déficit de diagnostic des HVG au Cameroun. Pourtant son diagnostic fait appel à des examens complémentaires dont les plus simples demeurent la radiographie du thorax, pour évaluer la silhouette cardiaque, l'électrocardiogramme qui détecte les altérations électriques reflétant l'hypertrophie, et l'échocardiogramme qui mesure des

dimensions des ventricules gauches. Cependant l'accès limité aux technologies avancées ainsi que le manque de données locales dans de nombreuses structures sanitaires en particulier au Cameroun dans la région de l'Adamaoua pose un réel problème pour établir un protocole de prise en charge des HVG. Ainsi dans la période d'Avril à Juillet 2025 nous avions mené une étude sur le profil Radio-clinique et électrique des hypertrophies ventriculaires gauches à l'hôpital militaire de Ngaoundéré, dans une population Camerounaise qui a des particularités en termes de retard diagnostique donc le but était de décrire les aspects cliniques ,radiographiques ,écho cardiographiques et électriques ,déterminer la prévalence des HVG pour chaque modalités à fin d'établir le protocole et améliorer la prise en charge des patients atteints d'HVG au Cameroun. Ainsi pour mener à bien notre étude, nous nous posons la question ci-dessous.

3. MATERIALS & METHODS

IL s'agissait d'une étude transversale, descriptive et analytique à recrutement prospectif, réalisée au service de cardiologie de l'Hôpital Militaire de Ngaoundéré entre avril et juillet 2025.

La population d'étude était constituée de patients vus en consultation ou hospitalisés. Ont été inclus tous les patients des deux sexes ayant consenti à participer à l'étude, disposant d'une radiographie thoracique, d'un électrocardiogramme et d'une échocardiographie confirmant une hypertrophie ventriculaire gauche. Les patients ne présentant pas d'HVG à l'échocardiographie ont été exclus.

L'échantillonnage était consécutif et non probabiliste.

Les variables étudiées comprenaient les données anthropométriques (Age, poids, taille, IMC)

Les données sociodémographiques (Sexe, Profession,)

Les données cliniques (dyspnées, douleurs thoraciques, palpitations, syncope) ainsi que les données radiographiques (Indice cardio-thoracique, Signe radiologique d'HVG), électrocardiographiques

(Indices de sokolow –Lyon : S(V1) + S (V5 Ou V6) ≥ 35mm, Fréquence cardiaque(FC), Durée de P/Axe P, Durée de QRS/ Axe de QRS, Intervalle PR, Intervalle QT/QTc), échocardiographique

(MVG/taille (g/m^2) elle sera calculée selon la convention de l'American society of échocardiography

(ASE) épaisseur relative de la paroi (ERP), FR%, FEV, type d'HVG).

La collecte a été réalisée à partir des dossiers médicaux, des registres hospitaliers et d'une fiche d'enquête standardisée.

L'analyse statistique a été effectuée à l'aide du logiciel SPSS version 21. Le test du Chi-deux de Pearson a été utilisé pour la comparaison des proportions, avec un seuil de significativité fixé à $p < 0,05$.

4. RESULTATS ET DISCUSSION

Durant la période de l'étude, nous avons colligé 110 patients, (48 femmes et 62 hommes) dont 34 présentaient une HVG à l'échographique, considérée comme la modalité de référence

Pour l'aspect anthropométrique, La tranche âge la plus représentée était Celle de 60 à 70 ans (35, 3 %). Une légère predominance masculine était observée (52, 9 %), avec un sex-ratio de 1, 1. L'aspect clinique révèle cependant, la dyspnée et les palpitations comme étant les principaux motifs de consultations des HVG.

[See Annex — Figure 1: Répartition des 34 patients HVG échographique selon l'étiologie]

L'HTA représente l'étiologie la plus représentée des HVG.

[See Annex — Tableau1: Aspects radiographiques des Hypertrophies ventriculaires gauches]

Dans notre étude, la majorité des patients presentant une HVG échographique avaient une cardiomegalie avec une indice thoracique comprise entre 0.60 et 0.65 (42%). Le signe radiographique le plus frequent était l'enfoncement de l'apex du ventricule gauche dans le diaphragm (58.8%)

[See Annex — Tableau 2: Aspects électrocardiographiques des Hypertrophies ventriculaires gauches]

Le rythme sinusal était majoritaire chez les patients presentant une HVG (91.1%). L'HVG moderée selon l'indice de sokolow –Lyon representait la forme électrique la plus frequente (47%). Les anomalies du segment ST-T anterolaterale constituaient l'anomalie électrique associée la plus observée (61.7%)

[See Annex — Tableau 3: Aspects échocardiographiques des HVG]

Les formes moderées d'HVG étaient prédominantes aussi bien chez les femmes (56.25%) que chez les hommes (66.66%). L' hypertrophie concentrique était le type morphologique le plus frequente, observée chez des femmes (68.75%) et 88.9 % des hommes. La majorité des patients avaient une fraction d'ejection ventriculaire preservée (61.76%)

[See Annex — Tableau 4: Correlations diagnostiques entre radiographie thoracique, ECG, et échocardiographie.]

Une tendance OR (13.07) observée entre l'électrocardiogramme et l'échocardiographie avec P-value. Significative (P= 0.001) était supérieure à celle observée entre la radiographie du thorax et l'échocardiographie dont la tendance OR = 2 une association statistiquement non significative (P=0,07).

[See Annex — Tableau 5: Prévalence des HVG selon les méthodes diagnostiques]

L'échocardiographie était la methode de diagnostique la plus performante pour la detection des HVG avec une prevalence de 30,9% superieure a celle obtenue par la radiographie thoracique (26,4%) et electrocardiogramme selon indice de sekolow-Lyon (25,5%).

Aspects anthropométriques

La majorité de nos patients résidaient dans la région de l'Adamaoua 61.8%, dans la ville de Ngaoundéré. Ces résultats peuvent s'expliquer par le fait que notre étude s'est déroulée à Ngaoundéré. Plusieurs travaux ont mise en évidence la corrélation entre urbanisation et le développement des facteurs de risque cardiovasculaire.

Dans notre série, la tranche d'âge la plus représentée était celle de 60-70 ans une proportion près de 35.3%.

Cette observation peut s'expliquer par une le prolongement de l'espérance de vie liée aux différents programmes de lutte contre les pathologies du système cardiovasculaire. Nos données ne se rapprochent pas de ceux de KONIKO, de Dr KONE Pacôme au CHUGT à Bamako plus que SARR au Sénégal qui retrouvaient respectivement 48,7% 49.5% et 60%. **(KONE 2025, SARR 2016).**

La prédominance masculine était de 52.9 % avec un sexe ratio de 1.1. Cette observation est légèrement égale à des résultats de KONE 52.2% avec le même ratio 1.1**.** Et proche de celle de Cheick OUMAR 54.50% mais diffère des résultats de KINIKO et de TCHEDRE, Ces résultats pourraient s'expliquer par la faiblesse de notre échantillon. **(KONIKO 2023, SARR 2016).**

Toutes les couches sociales sont touchées par les HVG, dans notre étude on trouve un taux élevé chez les ménagères 44.1%, une proportion

largement supérieure à celle de Cheick OUMAR KONATE et KONE 30.07% et 32,7%. **(KONE 2025, C. OUMAR)**

Dans notre étude la proportion des mariés est de 67.6% ces résultats pourraient s'expliquer par le stress des responsabilités familiales.

Aspects cliniques

La dyspnée d'effort constitue le premier signe fonctionnel de découverte des HVG ainsi que les palpitations ceci ne concorde pas totalement avec les résultats de Cheick OUMAR, certes la dyspnée d'effort constitue également le premier signe fonctionnel 43.70% mais alors ses résultats révèlent les douleurs thoraciques 30.84%.

L'HTA est la principale étiologie pathologique des HVG dans notre étude 59% avec l'HTA de grade I 35.5% aussi vrai que nos résultats concordent à ceux de Cheick OUMAR **(C. OUMAR).** Mais qui présentent l'HTA de grade III comme le niveau de PA le plus représentatif avec 43% des cas supérieur aux 35% retrouvés par BOUARE au CHUPG et Abakar B. au Sénégal qui était de 21,21%. Ces chiffres peuvent s'expliquer par le fait que tous nos patients ont été recrutés dans une unité de cardiologie qui venait de terminer une campagne de lutte et de suivi de l'HTA pouvant ainsi réduire les chiffres tensionnels d'ailleurs nos chiffres tensionnels sont inférieurs à ceux de KONE l'étude de Dakar quant à elle s'est réalisée sur une population spécifique d'imams.

Nous tenons à souligner que la plupart des études réalisées dans le monde se penchent à l'HVG chez l'hypertendu mais dont étiologie comme le diabète, la sédentarité, l'insuffisance rénale ne sont pas à négliger. **(TCHEDRE 2023)**.

La pression artérielle systolique maximale était de 260 mmHg comparable aux résultats trouvés par KONE où la pression systolique maximale était de 280 mmHg. La pression artérielle diastolique maximale était de 160mmHg.

(KONE 2025)

Aspects radiographiques

Une proportion de 85% des patients ayant une cardiomégalie, avaient une HVG à l'échographie avec une prédominance de 42% des ICT de (0.60-0.65).La prévalence est de 26.4%, ce résultat est plus spécifique car il exclut les faux positifs radiologiques ainsi, elle est une prévalence des HVG certaines ,bien que l'association n'est statistiquement significative (P > 0.05) ce résultat peut s'explique par une faiblesse de l'échantillon mais aussi les cardiomégalies radiologiques surviennent tard après l'HVG et peu dénoter la survenue tardive ou en stade évolutif. **(TCHEDRE 2023)**

Aspects électriques

Le rythme cardiaque était sinusal chez 91.1% des patients atteint d'HVG électriques ce résultat concorde avec celui retrouvé par KONE 93,1 % mais à des proportions différentes. La fibrillation atriale était le trouble de rythme le plus fréquemment rencontré chez près de 23.5 des patients HVG. Nous pensons que l'HVG est avant tout une anomalie de la masse myocardique qui peut ne pas affecter le système de conduction, ceci dit le nœud sinusal n'est pas modifier et continue d'imposer un rythme normal. Les troubles de rythme ne surviennent dans les formes sévères ou associées à une atteinte auriculaire ou de fibrose myocardique. **(KONE 2025). Dans** notre étude la durée du complexe QRS était normale chez 79.4% des patients HVG avec une déviation normale sur 61.8% des patients. Ces résultats s'expliquent par un absence d'élargissement du complexes QRS mais plutôt une augmentation de l'amplitude du QRS signe des HVG isolé. **(TCHEDRE 2023)** 82% des patients ont un indice de sokolow –Lyon supérieur à 35mm avec une prévalence des HVG électrique de 25.4% cette prévalence est supérieure à celle retrouvée en France avec la même indice 7.5 à 20.5 % inférieure à celle retrouver au Togo 46.2% et celle de l'étude de KONE à 46,5%.

L'association était significative entre la présence d'une HVG électrique et une HVG échographique (P<0.05).

Aspects échocardiographiques

Dans notre étude, 56.25% des femmes avait une HVG modérée et de type concentrique 68.75%. Chez les hommes 66.66 % HVG modérée et 88.89% HVG concentriques,

Les résultats de notre étude sont largement supérieurs à ceux retrouvées par A. NIAKARA sur une population HTA **(NIAKARA 2001).**

Ces résultats peuvent s'expliquer selon KONE par le faite, qu'une augmentation de la tension pariétale entraîne donc une hypertrophie concentrique compensatoire. Ce mécanisme s'exerçant chez l'hypertendu, qui est l'étiologie principale des HVG.

Chez les patients atteints d'HVG 61.76% avaient une fraction d'éjection préservée dont supérieure ou égale à 50%, ces résultats seront non seulement compatibles à la littérature aussi qu'elles sont inférieures aux données obtenues par KONE et TCHEDRE respectivement 83% et 73%, la FEVG reste préservée grâce à l'hypertrophie des parois ventriculaires. **(TCHEDRE 2023)**

5. CONCLUSION

Au terme de notre étude dont l'objectif général était d'établir le profil Radio-clinique et électrique des hypertrophies ventriculaires gauches à l'hôpital militaire de Ngaoundéré, il ressort que L'aspect anthropométrique des HVG se caractérise par une prédominance des personnes de sexe masculin (52.9%), des résidents des zones urbaines, les femmes de profession ménagère (44.1%). Sur le plan clinique elle est marquée par des signes telles que les dyspnées, les palpitations, les douleurs thoraciques, l'HTA est la principale étiologie des hypertrophies ventriculaires gauches avec une proportion de 59% mais il existe aussi d'autre étiologie comme le diabète, l'obésité, l'insuffisance rénale. A la radiographie du thorax elles sont caractérisées par des cardiomégalies avec un enfoncement de l'apex du Ventricule gauche dans le diaphragme. Les aspects électriques des HVG sont cependant, des rythmes cardiaques sinusal 91.1%, les troubles de rythmes comme la fibrillation atriale 23.5%, des complexes QRS normal, un indice de Sokolow Lyon supérieur à 35 mm dans 82% des cas d'HVG.

Le profil ainsi établi dans ce travail en découle des différents aspects radiographiques, cliniques et électriques avec des prévalences telles que 30.90% en échocardiographie, 25.45% en électrocardiogramme et 26% en radiographie.

Ainsi L'échocardiographie demeure la modalité de référence pour le diagnostic de l'HVG mais l'apport combiné de la radiographie thoracique, la clinique, l'électrocardiogramme et de l'échocardiogramme renforce la fiabilité du dépistage et permet une meilleure prise en charge des patients. Pour cela une étude devra être menée avec un échantillon plus vaste dans une longue durée sur le profil radio clinique, échocardiographiques et électrique des HVG.

REFERENCES

BOOMBHI J et al prévalence et mortalité des maladies cardiovasculaires en milieu hospitalier camerounais : cas de deux hôpitaux de référence de la Ville de Yaoundé

DT NAIBE et al (2016) Cardiologie tropicale: Fardeau des maladies cardiovasculaires à Ndjamena

A Bondues Département de cardiologie, hôpital Erasme ULB: mise au point d'une hypertrophie ventriculaire gauche: comment identifier la cause?

DJIENTCHEU NGUEMALEU Hypertrophie ventriculaire gauche chez l'hypertendu : régression sous inhibiteurs d'enzyme de conversion de l'inhibiteur calcique

MALONGA MUNAMA JACQUES A. 2002, étude diagnostique de l'hypertrophie

ventriculaire gauche à l'électrocardiographie, à la radiographie et à l'échographie chez les patients hypertendus, de Kinshasa

NIAKARA et al. Hypertrophie ventriculaire gauche du noir Africain hypertendu : Etude échocardiographie chez 452 Sujets.

Marwick TH, Gilbert TC, Aurigemma G, Chirinos J, Derumeaux G, Galderisi M et al. Recommandations

on the Use of Echocardiography in Adult Hypertension: A Report from the European Association of Cardiovascular Imaging (EACVI) and the American Society of Echocardiography (ASE). J Am Soc Echocardiography. 2015; 28(7):72754.

Lang RM, Badano LP, Mor-Avi V, Afilalo J, Armstrong A, Ernande L et al. Recommendations for cardiac chamber quantification by echocardiography in

adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. J Am Soc Echocardiogr. 2015;28(1):1- 39.e14.

Lang RM, Badano LP, Mor-Avi V, et management of hypertrophic al. Recommendations for cardiac cardiomyopathy of the European chamber quantification by Society of Cardiology (ESC). Eur Heart echocardiography in adults : an update J 2014;35:2733.

Journal de la recherche scientifique de l'université de Lomé : prévalence de l'hypertrophie ventriculaire gauche les patients hypertendus dans trois structures médicales de LOME.

OLIVIER PANCHA MBOUEMBOUE et al, modification électrocardiographiques et echocardiographiques chez les hypertendus camerounais.

Mise au point d'une hypertrophie ventriculaire gauche comment identifier la cause.

NICOLAS B. DAYAL a et HAJO MÜLLER a Rev Med Suisse 2017 ; 13 : 1106- 12 Découverte d'une hypertrophie ventriculaire gauche à l'échocardiographie adulte.

Leonid STERNIK et Alexey Kryvenko revue 23.04.2024 Hypertrophie du ventricule gauche.

Lang R.M., Badano L.P., Mor-Avi V. et al. Recommendations for cardiac. Chamber quantification by echocardiography in adults: an update from the American Society of Echocardiography and the European Association of Cardiovascular Imaging. Eur Heart J Cardiovasc Imaging. 2015;16(3):233-270

Simone G., Devereux R.B., Roman M.J.et al. Relation of obesity and gender to left ventricular hypertrophy in normotensive and hypertensive adults. Hypertension. 1994;23(5):600-606.

C.OUMAR hypertrophie ventriculaires gauches électrique et echographique.à propos de 389 cas dans le service de cardiologie de l’hôpital Gabriel Touré.

E. Cornu, I. Belmihoub, N. Burnichon, C. Grataloup, F. Zinzindohoué, S. Baron, E. Billaud, M. Azizi,

A.-P. Gimenez-Roqueplo, L. Amar, Phéochromocytome et paragangliome, Volume 6306, Issue 11,

11/2019, Pages e21-e22, ISSN 0248- 8663, http://dx.doi.org/10.1016/j.revmed.2019.07.008

TCHEDRE, T. (2023). Aspects électrocardiographiques et échocardiographiques de l'hypertendu de sexe masculin au service de cardiologie du CHU-GT [Thèse de doctorat en médecine]. Université des Sciences, des Techniques et des Technologies de Bamako, Faculté de Médecine et d’Odontostomatologie, Bamako, Mali.

KONIKO, K. (2023). Aspects électrocardiographiques et échocardiographiques de l'hypertendu de sexe féminin dans le service de cardiologie du CHU-GT [Thèse de doctorat en médecine]. Université des Sciences, des Techniques et des Technologies de Bamako, Faculté de Médecine et d’Odontostomatologie, Bamako, Mali.

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Yene Zang Bernard Severin , Samen Sileu Chimène Nathalie. (2026). Profil Radio-Clinique et Electrique des Hypertrophies Ventriculaires Gauches à l'Hôpital Militaire de Ngaoundéré. IQ Research Journal, 5(2), IQRJ-V05I02-26004019. www.iqresearchjournal.com

ANNEXES

Annex I —

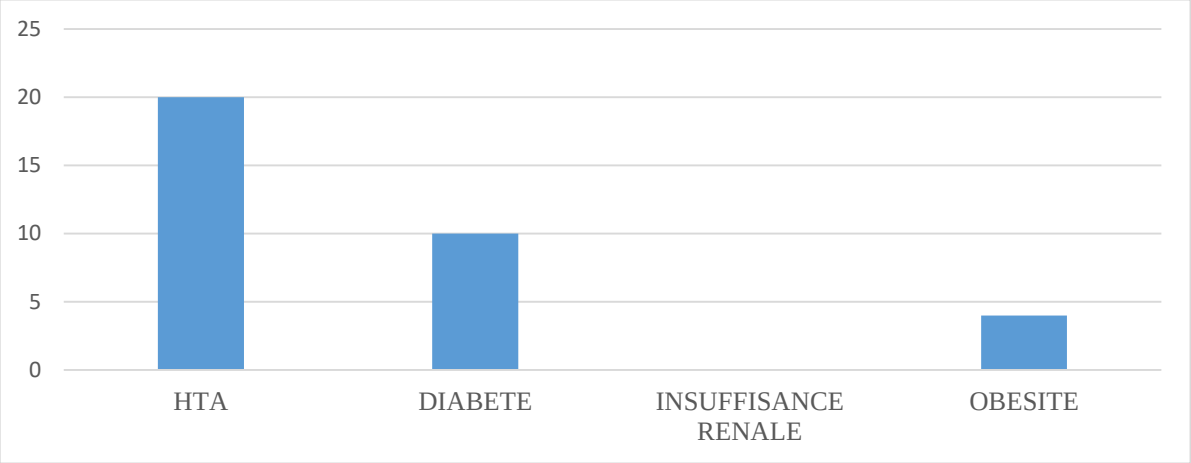


Figure 1 : Répartition des 34 patients HVG échographique selon l'âge

Annex II — Tableau 1 : Aspects radiographiques des Hypertrophies ventriculaires gauches

Variables	Modalités	Effectif (n)	Fréquence (%)
Indice cardio-thoracique (ICT)	< 0,50	5	15,0
	0,51 – 0,55	3	9,0
	0,55 – 0,60	4	11,0
	0,60 – 0,65	14	42,0
	> 0,65	8	23,0
	Total	34	100
Signes radiographiques d'HVG	Enfoncement de l'apex du VG dans le diaphragme	20	58,8
	Allongement de l'arc inférieur gauche déplacé vers le bas	9	26,4
	Rapprochement du VG vers la limite latérale gauche du thorax	5	14,8
	Total	34	100

Annex III — Tableau 2 : Aspects électrocardiographiques des Hypertrophies ventriculaires gauches

Variables	Modalités	Effectif (n)	Fréquence (%)
Rythme cardiaque	Rythme sinusal	31	91,1
	Rythme non sinusal	3	8,9
	Total	34	100
Durée du QRS	Normale	27	79,4
	Anormale	7	20,6
	Total	34	100
Axe électrique QRS	< -45°	13	38,2
	-45° à +110°	21	61,8
	> +110°	0	0
	Total	34	100
Importance de l'HVG	< 37 mm	10	29,4
	Modérée (38–40 mm)	16	47,0
	Importante (41–45 mm)	6	17,6
	Très importante (>45 mm)	2	6,0
	Total	34	100

Annex IV — Tableau 3 : Aspects échocardiographiques des HVG

Variables	Modalités	Effectif (n)	Fréquence (%)
Femmes : Classe d'HVG	Légère	5	31,25
	Modérée	9	56,25
	Sévère	2	12,5
	Total	16	100
Hommes : Classe d'HVG	Légère	2	11,11
	Modérée	12	66,66
	Sévère	4	22,22
	Total	18	100
FEVG	Réduite	8	23,52
	Modérément altérée	5	14,70
	Préservée	21	61,76
	Total	34	100

Annex V — Tableau 4 : Correlations diagnostiques entre radiographie thoracique, ECG, et échocardiographie

Variables compares	Présence d'HVG	Absence d'HVG	OR / p-value
Cardiomégalie radiographique vs HVG échographique	29	56	OR=2 ; p=0,07
ICT normal vs HVG échographique	5	20	
HVG électrique vs HVG échographique	28	20	OR=13,07 ; p=0,001
Absence d'HVG électrique	6	56	

Annex VI — Tableau 5 : Prévalence des HVG selon les méthodes diagnostiques

Méthode diagnostique	Critères	HVG (n)	Prévalence (%)
ETT	Critères échocardiographiques	34	30,9
ECG + ETT	Sokolow-Lyon >35 mm	28	25,5
Radiographie + ETT	Cardiomégalie	29	26,4

Entrepreneurial Education for a Changing Society

IQ RESEARCH JOURNAL
www.iqresearchjournal.com

KESMONDS RESEARCH INSTITUTE

Original Article IQRJ: Volume 005, Issue 002, May 2026 www.iqresearchjournal.com

Received : 20/02/2026___Revised : 20/04/2026___Accepted : 30/04/2026___Published : 02/05/2026

Quality Assurance and Calibration of Medical Devices Practices: Case Study of Government Regional Referral Hospitals in Uganda

Moses Lopuka[a,b] and Atanga Desmond Funwie[a]

Affiliations

u. [a] Institute of Engineering Sciences and Technology, Kesmonds International University

v. [b] Uganda Cancer Institute, Uganda

Corresponding Author:

Lopuka M. & Atanga DF.

Email: moseslopuka@gmail.com
atanga@kesmonds.edu.cm

Paper ID: IQRJ-V05I02-26005020

ABSTRACT

The accuracy of medical devices directly influences clinical decision-making and overall patient outcomes. To ensure that medical devices are accurate and safe all the time, it requires robust calibration and quality assurance practices coupled with maintenance. However, this topic is underexplored in studies regarding medical devices in Uganda. Here, we examine the calibration and quality assurance best practices in regional, national, and specialized public hospitals in Uganda. Moreover, we hypothesized that calibration and QA status directly influence medical device usage. To achieve this, we conducted quantitative and qualitative research in a cross-sectional study design in 17 regional referral hospitals, 3 national referral hospitals, and 5 specialized hospitals. A structured questionnaire was administered to 42 participants, which included biomedical engineers, technicians, and maintenance officers. It covered thematic areas of examining the existence of SOPs & QA protocols, availability of test tools, equipment downtime during calibration, calibration support, and training. The results of the study demonstrated that calibration and acceptance testing of donated equipment were performed on-demand. Nearly 43% of respondents had no SOPs and QA protocol, while 26% were unsure of it. Additionally, 20 of 42 respondents recorded the unavailability of test tools. Furthermore, about 75% of the respondents' sentiment revealed that calibration status directly affects use. This was consistent with a 71% score for a more than 1 month period on average time a medical device remains grounded as it waits for calibration. Overall, the calibration and QA practices of medical devices in public hospitals in Uganda are still in their infancy. It requires administrative, technical, and financial support. This study will become a precursor to calling for more effort, guidance, and strengthening the calibration and QA practices among biomedical engineers in Uganda.

Keywords: *Biomedical Engineers, Medical devices, calibration, quality assurance, test tools.*

1. INTRODUCTION

Medical gadgets are crucial to a healthcare system, particularly for accurate diagnosis, illness monitoring, and treatment, as well as for patient rehabilitation [1]. Clinical decision-making and overall patient outcomes are directly impacted by their operational performance. Ferda I. et al. (2026) state that this trend is substantially documented in affluent nations where patient health outcomes are consistent across demographic groupings. Because of the rigorous best practices for managing and maintaining medical devices, including quality assurance, calibration, and routine maintenance. As a result, medical devices are used extensively.

Medical gadgets are crucial to a healthcare system, particularly for accurate diagnosis, illness monitoring, and treatment, as well as for patient rehabilitation [1]. Clinical decision-making and overall patient outcomes are directly impacted by their operational performance. Ferda I. et al. (2026) state that this trend is substantially documented in affluent nations where patient health outcomes are consistent across demographic groupings. Because of the rigorous best practices for managing and maintaining medical devices, including quality assurance, calibration, and routine maintenance. As a result, medical devices are used extensively.

Muhammad K. et al. (2020) reviewed the literature on Nigerian medical imaging quality assurance. The results showed that inconsistent calibrations and quality assurance caused disparities in image quality and clinical judgements. The impact of medical device quality assurance and calibration on the use of medical devices, however, was not specifically addressed in the study. The confidence in medical device calibration knowledge and skills among healthcare professionals (HCP) in Ghana was investigated in a comparable cross-sectional study by Benjamin A. et al. (2025). According to the authors, clinical engineers and technicians were more confident than other HCP. Additionally, trained HCPs reported feeling more confident than their untrained counterparts.

However, the authors did not clearly examine the best practices and impact of medical device quality assurance and calibrations on its use.

There is a research gap in identifying the best practices in medical device quality assurance and calibration practices specific to sub-Saharan Africa, despite the fact that several studies have taken advantage of the difficulties, skilled capacity gap, and factors affecting maintenance, quality assurance, and calibration of medical devices in sub-Saharan Africa. We evaluated the best practices in medical device calibration and quality assurance across Uganda's regional referral hospitals in order to close this gap. Additionally, we assessed how quality control and calibrations affected the use of medical devices. Here, we postulated that, in addition to maintenance, calibration and quality assurance procedures have a direct impact on the use of medical devices.

In fact, we saw uncalibrated or subpar quality assurance and calibration procedures as quiet, deadly murderers that are overlooked throughout the medical device lifetime. It's also critical to keep in mind that issues with medical equipment are directly linked to poor patient outcomes [2].

Medical device calibration is one component of complete maintenance from the perspective of clinical engineering. According to Kumar R. et al. (2023), medical device calibration is a systematic process that verifies and adjusts medical device measurements to a recognised standard. This procedure guarantees that medical equipment consistently deliver precise measurements and outcomes within the permitted tolerance range or at all times.

According to John G. *et al.,* (2025), a typical calibration includes the following:

1) Purpose and scope;

2) Roles and responsibilities, e.g., in-house calibration and supplier-based calibration;

3) Frequency of calibration;

4) The use of risk-based scenarios for decisions (refers to second-level document for risk management) and the impact on the DMR;

5) Required equipment and standards;

6) Limits for accuracy and precision;

7) Limits of uncertainty and calculations;

8) Preliminary examinations and operations;

9) Calibration process description;

10) Remedial action for product as linked with the nonconforming corrective action process; and

11) Documentation requirements for not only internal process, but also those of the calibration supplier.

Calibration and Quality Assurance (QA) procedures are essential to the medical device lifecycle in a hospital setting. While quality assurance methods aim to reduce errors and increase safety and performance compliance, calibration ensures that medical equipment consistently produces reliable and accurate findings. As a result, both guarantee accurate and safe medical devices.

Together with the knowledge and experience of medical professionals, the accuracy and utility of medical devices are essential for accurate diagnosis and patient treatment. Kumar R. et al. (2025) state that "proper functionality of medical devices is crucial for patients in a large number of serious medical situations." Therefore, it is crucial to carry out as thorough and independent testing of medical device capabilities as is practical in order to obtain the most reliable and accurate diagnosis and patient treatment.

The purpose of this case study is to examine the quality assurance and calibration processes for medical devices in Ugandan government regional referral hospitals. We will then evaluate how calibration and quality control affect the use of medical devices. We postulated a relationship between the calibration and quality assurance status of medical devices and their use in hospital settings.

3. MATERIALS & METHODS

Research design

We investigated medical device calibration and quality assurance best practices (set procedures) at regional referral hospitals in Uganda using both quantitative and qualitative research methods in a cross-sectional study design. There were five-point Likert scale items in the survey, ranging from strongly disagree (1) to strongly agree (5). Other questions required a yes, no, or maybe response. Finally, there were open-ended enquiries that needed succinct, exact answers.

The overall survey questionnaires were structured as follows:

1) General information which included; hospital name, hospital level, participant's primary role, and number of years in practice.

2) Calibration test tools and resources; which assessed availability of test tools such as: patient monitor simulators, defibrillator analyzers, electrical safety analyzer, etc. In this section, we also examined external support to provide calibration services as well as availability of UPS or voltage stabilizers for high-risk medical devices.

3) Calibration & quality assurance procedures. This section was a multiple-choice grid with a 5-point Likert scale. It analyzed; planned preventive maintenance frequency, calibration status, acceptance testing of donated medical devices, spare

parts availability, inventory tracking, and documentations. Here, we analyzed the calibration and quality assurance practices based on widely applied clinical engineering practices.

4) Open end questions which explored; whether donated medical devices were provided with user/service manuals, issuance of calibration stickers, availability of standard operating procedures (SOPs), participants opinions on medical device calibration status and utilization, and major challenge in ensuring medical device accuracy.

Study population.

The targeted study participants were biomedical technicians and engineers who are full-time, part time or volunteering at regional, national and specialized public hospitals in Uganda. There are 17 regional referral hospitals in Uganda scattered in the Northern, Northeastern, Eastern, Central, Southwestern and Western parts of the country. Each regional referral hospital is estimated to serve 2 million people with an average 400-bed capacity, according to Sherry R.*et al.,* (2022). The national referral (3) and specialized hospitals (5) were included because of the limited number of biomedical engineers and technicians employed in public hospitals in Uganda. On average, there are two biomedical engineers/technicians per public hospital in Uganda. The regional, national and specialized public hospitals provide medical services that are not available at district level hospitals and lower health centers.

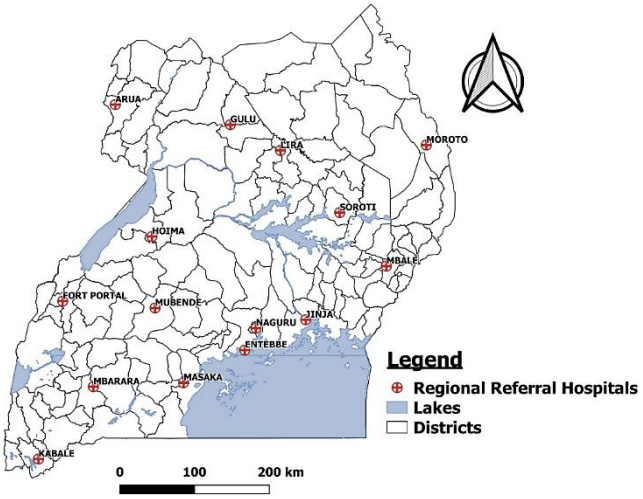


Figure 1: Location of regional referral hospitals in Uganda. Adopted from Sherry. R. et al., (2022).

Inclusion criteria.

The study participants must meet the criteria as follows:

a) The study targeted all engineering and technical cadres who are primarily responsible for the planning, acquisition, maintenance, management and disposal of medical devices in the respective public hospital.

b) The study participant must be a full-time, part-time, contract-based or a volunteer employed at a public regional, national or specialized hospital in Uganda.

c) The study participant must voluntarily agree to verbally consent to the study.

d) The study participant must have worked in the respective hospital for more than 6 months, which is a recommended probation period.

Exclusion criteria.

The exclusion criteria for the study were set as follows:

a) The participant is not directly responsible for planning, acquisition,

maintenance, management and disposal of medical devices in the respective public hospital.

b) The participant is not an employee at public regional, national or specialized hospital in Uganda.

c) The participant withdrew his or her consent at any time of the study.

d) The participant has not completed more than 6 months' probation period in the respective public hospital.

Data analysis.

The study collected data through administering a structured questionnaire to the study participants. The questionnaire was designed in google forms and pretested prior to publishing the link to the selected target participants. Furthermore, participants were limited to only one response to minimize data duplication. In an event of any digital challenges, a paper-based copy of the questionnaire was issued to the respective study participant. Responses to the questionnaire were verified for completeness and duplication for onward analysis. The collected dataset was migrated to Spreadsheet, 2016 for analysis. In some sections, the 5-point Liker scale responses were converted to numerical figures for quantitative analysis.

Privacy and confidentially.

An informed consent was obtained from the participants. The participants were debriefed on the study objectives and their right to withdraw from the study at any time without any penalty. Furthermore, the participant's personal information such as: names, age, gender, phone number, marital status, date of birth, ethnicity, and email address were not collected. This was to mitigate identification of study participants. The data collected were securely stored in passworded computer with a cloud back up option.

4. RESULTS & DISCUSSION

General information.

With the exception of the Ministry of Health, district/general hospitals, smaller health facilities, and private hospitals, regional, national, and specialised public hospitals in Uganda employ an average of fifty biomedical engineers and technicians. Of these, 42 people responded. Regional, national, and speciality public hospitals were where the responders were dispersed. The majority of responders (21, or 50%) were from regional referral hospitals. Nearly 59.5% of the respondents are biomedical engineers, and their years of experience ranged from 1 to 19.

42 Total respondents	**50%** Regional Referral Hospital	**33%** Specialized Hospital
Appx. **6yrs** Median years in practice	Appx. **60%** Biomedical Engineers	**17%** National Referral Hospital

Figure 2: Shows different hospital level in the study.

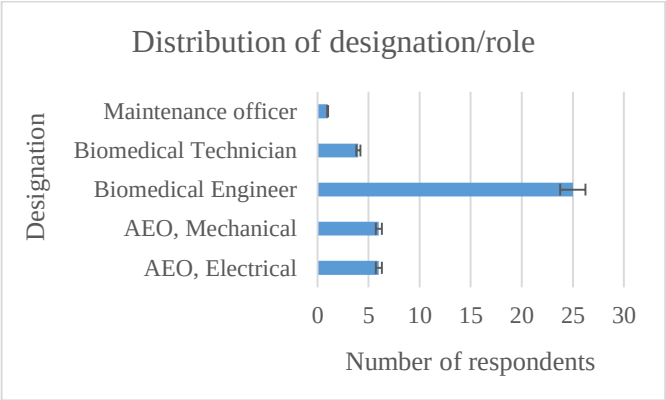


Figure 3: Shows different cadres or profession primarily responsible

for medical devices. Where AEO means Assistant Engineering officer

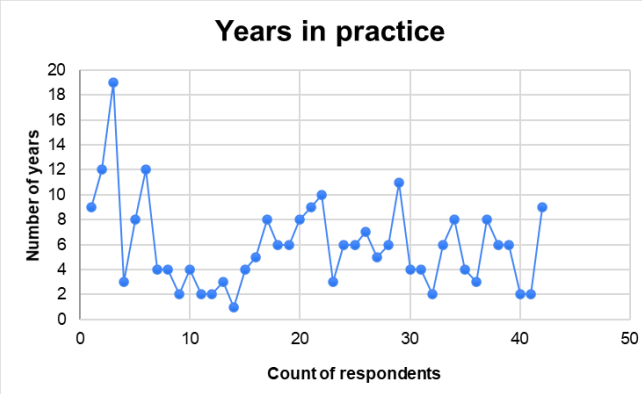


Figure 4: Shows distribution of years in practice among the count of respondents.

Test equipment and resources

The availability of test tools (simulators and analysers), such as electrical safety analysers, patient monitor simulators, and infusion pump analysers, is examined in this section. According to relevant standards, these test instruments are used to confirm that medical equipment are accurate within a permissible tolerance range.

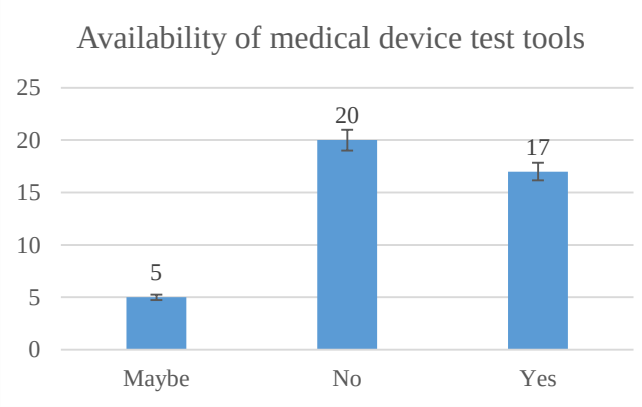


Figure 5: Availability of test tools.

The majority of responders (28.6%, n = 12) indicated the availability of gas flow analysers, followed by patient monitor simulators (23.8%, n = 10). The least accessible test tools among the 42 respondents were the ultra-sound phantom scoring (2.4%, n=1), water quality tester, and defibrillator analyser. For primary calibration help, hospitals without test equipment mostly relied on other institutions. Private outsourcing accounted for the majority (42.9%).

Additionally, about 26% of the respondents said that even the internal biomedical staff did not offer any external calibration support. Only 19% of respondents, however, said they were capable of internally calibrating medical equipment. According to 73.8% of respondents, all high-risk equipment was connected to a dedicated UPS in addition to the calibration services. This method simplifies quality assurance procedures by lowering errors brought on by unpredictable power supply.

Calibration and quality assurance.

In order to reduce the recurrence of errors and/or faults, we examined important calibration and quality assurance practices in this section, including calibration frequency, a risk-based approach to planned preventive maintenance, the implementation of a standard operating procedure for calibration, and incident reporting tools. It was found that, in contrast to planned preventive maintenance, which was primarily performed quarterly, calibration and acceptance testing of donated equipment was primarily conducted on-demand.

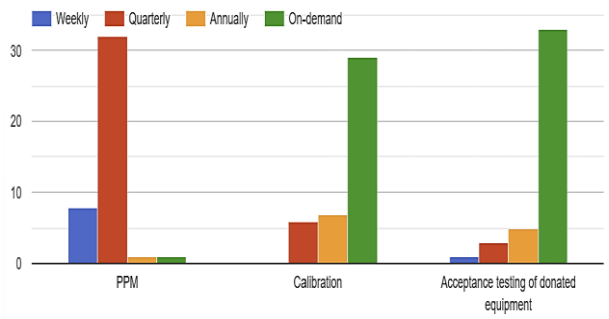


Figure 3: Planned preventive maintenance, calibration and acceptance testing frequency.

Majority of the respondents implement quarterly planned preventive maintenance

following a risk-based approach as described by J. Tobey Clark. *et al.,* (2009). Regarding standard operating procedures and QA protocols, the highest percentage of respondents (43%) do not have SOPs and QA protocols in place. Only 31% of the respondents have SOPs and QA protocols while the remaining 26% are unsure of SOPs and QA protocols. Conversely, a significant representation of 81% of the respondents have a formal incident reporting system for medical device malfunction or near misses.

From the perspective of capacity building, 28.6% of the respondents received training in calibration and or quality assurance practices. In addition, 69% of the respondent acknowledged no dedicated budget for calibration and quality assurance services or tools. Furthermore, most of the respondents disagree on availability of sufficient spare parts for medical devices in stock.

In general, the results from this portion of calibrating capacity and quality assurance showed deficiencies in almost every component. Many facilities lacked regular operating procedures, qualified personnel, a dedicated budget, and adequate calibration tools. There is a systemic resource gap since only 16.7% of facilities have a specific budget, and 71.4% of medical devices are grounded for more than a month as they wait to be calibrated.

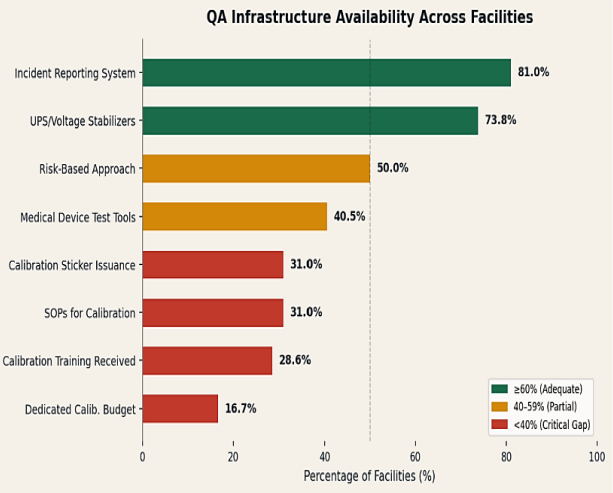


Figure 4: Quality Assurance infrastructure availability.

Most of the hospitals use Excel sheet for medical device tracking.

Impact on medical device utilization

This was a subjective response on how calibration and quality assurance practices can directly affect medical device utilization. Particularly, respondents provided feedback on an average time a medical device remains grounded as it waits for calibration. Furthermore, participants rated on a scale of 1 – 5 with 5 stars being the highest, the effect of calibration and quality assurance on medical device utilization.

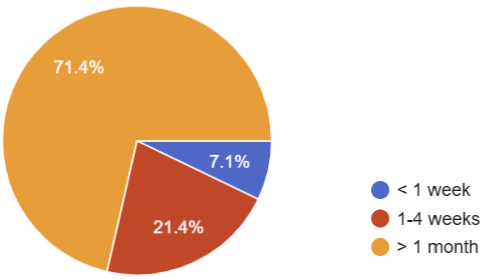


Figure 5: Average time medical devices remains grounded as it waits for calibration.

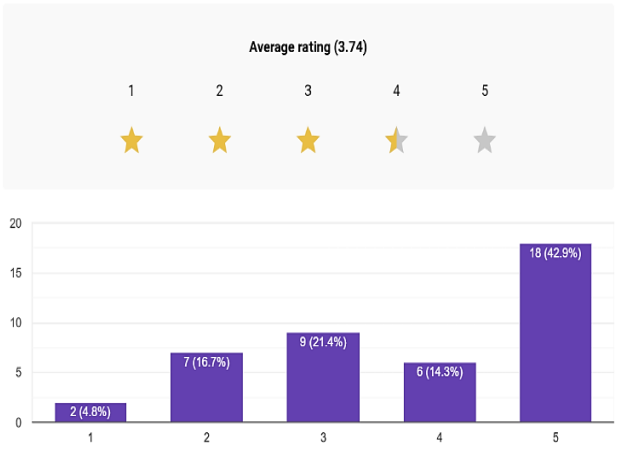


Figure 6: Sentiment rating on effects of calibration & QA activities on medical device utilization.

The graphs below show the respondents perceived impact of quality assurance and calibration on medical device utilization.

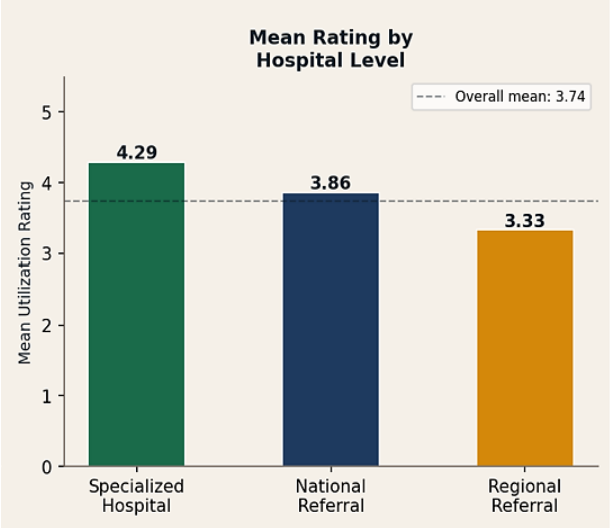


Figure 7: mean rating by hospital level

Role	n	Mean Rating
Biomedical Technician	4	4.25
Biomedical Engineer	25	4.16
AEO, Electrical	6	3.17
Maintenance Officer	1	3.00
AEO, Mechanical	6	2.33

Figure 8: Mean utilization rating by role

The correlation between medical device utilization impact rating and quality assurance and calibration practices were analyzed. The Spearman rank correlations were computed. Ordinal variables encoded as: Yes =2, Maybe = 1, No = 0.

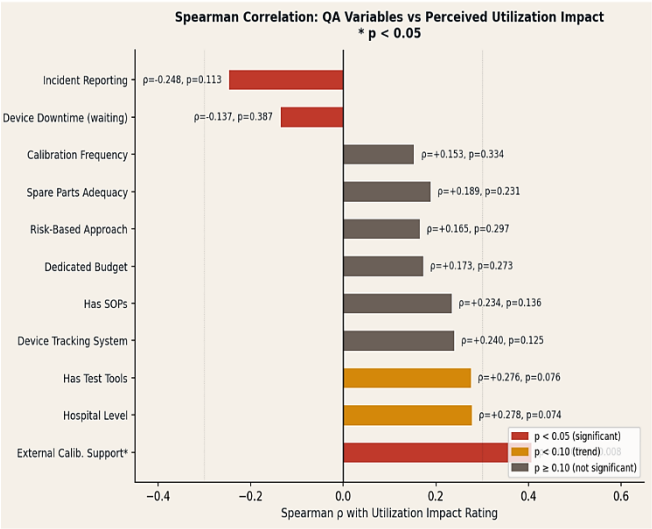


Figure 9: Spearman correlation on QA variables Vs perceived utilization impact.

The external calibration support was the highest significant correlation of utilization (rho = 0.407, p=0.008). Hospitals with in-house support perceived greater utilization impact than those relying on private outsourcing or receiving no support.

In addition, calibration budget was the strongest structural predictor of device downtime. Hospitals with a budget: 14.3% grounded more than 1 month. Hospitals without: 82.8% grounded more than 1 month. Chi-square = 14.342, df = 4, p=0.006

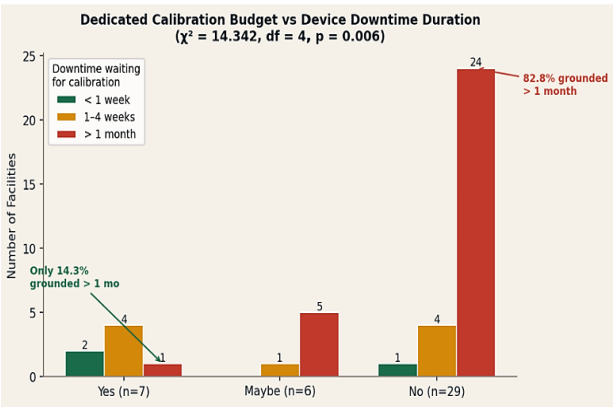
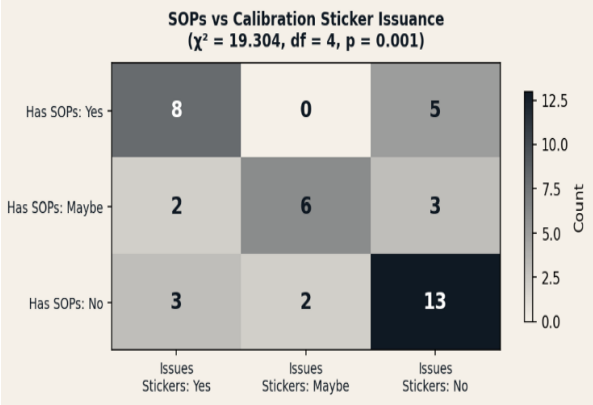


Figure 10: Budget vs medical device downtime duration

Furthermore, the presence of SOPs was strongly associated with calibration sticker issuance indicating that documented procedures drive observable compliance behaviours.



Lastly, the dual burden of inadequate spare parts (71.4%) and poorly documented donated medical equipment creates a compounding effect where hospitals cannot perform calibration even when willing, and cannot calibrate to appropriate standards without service manuals and test tools. Only 21.4% agree or strongly agreed that donated medical devices arrive with technical or service manuals.

Overall, the study hypothesis that medical equipment use in Ugandan hospitals is impacted by quality assurance and calibration procedures was rated as moderate. The direction of each link is consistent with the study hypothesis, even though only one predictor (external calibration support) met traditional significance standards at this sample size. The chi-square associations between the budget and downtime (p=0.0006) and SOPs and stickers (p=0.001) are strong. Strong respondent unanimity is shown in the mean utilisation impact rating of 3.74/5, with 42.9% choosing the highest grade.

The 'Uganda context'

The purpose of this section's open-ended, structured questions was to pinpoint the main issues and potential areas for development with medical equipment calibration and quality assurance procedures in Uganda's public hospitals. First, we investigated whether service manuals and calibration status stickers are included with donated medical equipment. While 28.6% of respondents disagreed that donated equipment lacked repair manuals, nearly 33% of respondents were unsure. Calibration stickers were not provided for any tested or calibrated medical devices, according to half of the respondents.

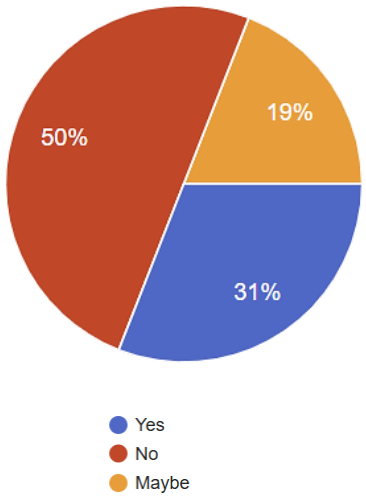


Figure 11: shows issuance of calibration stickers

Secondly, the respondents provided numerous challenges associated with calibration and quality assurance practices in public hospitals in Uganda. These included the following:

a) Limited or no funding at all for calibration services, especially when the medical device is required to be shipped outside or bring in a technical expert from oversea.

b) Limited or no test tools to verify the accuracy of medical devices to determine whether calibration is required.

c) Insufficient administrative support for technical capacity building and training on calibration and quality assurance practices.

d) Most medical devices are supplied without technical or service manuals for references on how to calibrate the model-specific medical device.

e) Unclear regulatory pathways and regulatory overlap among government agencies and technical working groups such as: National Drug Authority (NDA), Uganda National Bureau of Standards (UNBS), National Advisory Committee on Medical Devices (NACME).

f) No national reference accredited calibration laboratory, except for selected laboratory equipment like biosafety cabinets.

Lastly, the respondents pinpointed changes that could improve the medical device calibration and quality assurance practices in public hospitals in Uganda. These included: administrative support to trained specialised biomedical engineers on calibrations, adoption of computerize maintenance management systems (CMMS) that is comprehensive to the entire equipment lifecycle, procurement of test tools and dedicated budget line for calibration and quality assurance activities.

Discussions

This study explored medical device calibration and quality assurance best practices in Uganda's public hospitals. From the estimated total of 50 biomedical engineers and technicians in regional, national and specialized hospitals, we received responses from 42 personnel. This demonstrates the keen interest regarding the topic. Biomedical engineers formed the largest respondents also suggest a growing workforce and absorption into public hospitals. Initially, the role of medical device maintenance and management were held by technicians, many of whom were diploma holders with background in electrical or mechanical engineering.

Respondents greatly acknowledged the importance of medical device test tools with majority recognizing the different kinds of test tools and its application. The initial small number of respondents providing in-house calibration and quality assurance support to their respective hospitals could be attributed to a JICA project in collaboration with ministry of health, health infrastructure division (MoH-HID). The project was themed '*Improvement of health services through health infrastructure management (II): testing and calibration equipment/tools.*' In 2021, the project procured assorted medical device test tools and trained selected personnel in performance and safety testing of medical devices. Further study needs to evaluate the outcome of the project, especially in capacity building, training and instilling calibration and QA practices.

The highest number of respondents provided uninterruptable power supply for critical equipment demonstrating the importance in ensuring calibration accuracy. For instance, unstable power can reset calibration parameters in medical devices. Specifically, any power fluctuation during CT scanner QA and calibration can result into faulty reference data. Hence, making the equipment unreliable even after immediate QA and calibration. Thus, this leads to calibration inaccuracy and quality assurance protocols.

Interestingly, calibration activities and acceptance testing of donated equipment were mainly performed on-demand. This could be attributed to lack of test tools in many public hospitals, and untrained personnel. In addition, it also suggests that the biomedical engineers are extremely reluctant in performing calibration

and QA activities albeit availability of test tools. In principle, as a norm of best practices, medical devices must be tested for accuracy after every maintenance. However, this crucial practice is not yet adopted by many biomedical engineers at public hospital, which is consistent with the result observed in figure 6 under calibration and quality assurance frequency.

Subsequently, majority of the respondents had no SOPs and QA protocols in place. This exacerbates the reluctances from the biomedical engineers towards calibration and QA practices, especially those with the resources and tools. Limited or no SOPs is understandable in hospitals without any calibration resources and or support. However, this does not exempt developing a QA protocol for medical devices. These protocols may include: incoming medical device inspection, acceptance testing, inventory and documentation management, incident management, and among others.

Despite the limited effort to develop a comprehensive QA protocol for medical device covering its lifecycle, there is mushrooming interest in developing components like incident reporting tool. This was evident from the 81% score of respondents with medical device incident reporting system for malfunction and near misses in place. Perhaps, there is a growing effort that needs more refresher training, dedicated budget and resources to empower the biomedical engineers.

Besides calibration and QA best practices, this study subjectively examined medical device calibration status & QA activities to its utilization. Moreover 71.4% of the respondents indicated that medical devices remained grounded for more than 1 month as it waits calibration. Therefore, this simply means the medical device remains unavailable for use during that time, hence, directly affecting its utilization. Furthermore, respondents provided an average sentiment rating of 3.74 on a scale of 1 – 5; with 5 being the highest stars. The above average sentiment rating demonstrates that calibration & QA activities potentially affect medical device utilization. However, this study was limited in scope to further evaluate this hypothesis. This study simply pinpointed that there is likely relationship between calibration status and utilization of medical devices. Another study may be required to objectively provide insights rather than relying on subjective responses.

5. CONCLUSION

The study revealed that calibration and QA best practices are still in infancy with limited test tools available in public hospitals in Uganda. Additionally, majority of medical devices remains out of services for more than one month awaiting calibration. This was hindered by numerous challenges from administrative, technical and financial will. There is need for more emphasis to the biomedical engineers to test medical device after every maintenance, at incoming inspection and acceptance testing, especially for donated equipment. Moreover, the biomedical engineers need to start developing QA protocols for entire equipment lifecycle. This may require less finances compared to procurement of test tools. Lastly, the government through ministry of health needs to recruit more biomedical engineers to reduce the workload. Furthermore, provide specialty technical training for biomedical engineers other than recruitment of maintenance generalist.

REFERENCES

[1]WHO, ;Medical equipment maintenance programme overview WHO Med. Clin. North Am., vol. 74, no. 5, pp. 1113–1120, 2011.

[2]P. Howitt et al.;Technologies for global health, Lancet, vol. 380, no. 9840, pp. 507–535, 2012, doi: 10.1016/S0140-6736(12)61127-1.

[3]R. T. Ssekitoleko et al. Status of Medical Devices and their Utilization in 9 Tertiary Hospitals and 5 Research institutions in Uganda, Glob. Clin. Eng. J., vol. 4, no. 3, pp. 5–15, 2022, doi: 10.31354/globalce.v4i3.127.

[4]S. C. E. Anyangwe and C. Mtonga,Inequities in the Global Health Workforce: The Greatest Impediment to Health in Sub-Saharan Africa, Int. J. Environ. Res.

Public Health, vol. 4, no. 2, p. 93, 2007, doi: 10.3390/IJERPH2007040002.

[5]J. G. Meara et al., Global Surgery 2030: evidence and solutions for achieving health, welfare, and economic development, Lancet (London, England), vol. 386, no. 9993, pp. 569–624, Aug. 2015, doi: 10.1016/S0140-6736(15)60160-X.

[6]I. H. Marks, H. Thomas, M. Bakhet, and E. Fitzgerald, Medical equipment donation in low-resource settings: A review of the literature and guidelines for surgery and anaesthesia in low-income and middle-income countries, BMJ Glob. Heal., vol. 4, no. 5, pp. 1–9, 2019, doi: 10.1136/bmjgh-2019-001785.

[7]R. Y. Hsia, N. A. Mbembati, S. MacFarlane, and M. E. Kruk, Access to emergency and surgical care in sub-Saharan Africa: The infrastructure gap, Health Policy Plan., vol. 27, no. 3, pp. 234–244, 2012, doi: 10.1093/heapol/czr023.

[8] Medical device donations: considerations for solicitation and provision. https://apps.who.int/iris/handle/10665/44568?locale-attribute=en (accessed Jul. 20, 2025).

[9]D. Emmerling, P. W. Sholar, and R. A. Malkin,A sustainability evaluation of a biomedical technician training program in Honduras, Health Technol. (Berl)., vol. 8, no. 4, pp. 291–300, Sep. 2018, doi: 10.1007/s12553-018-0241-7.

[10] Operation Manual for Regional Medical Equipment Maintenance Workshops and Medical Equipment Maintenance Guidelines | Ministry of Health Knowledge Management Portal. http://154.72.196.19/publications/health-infrastructure/operation-manual-regional-medical-equipment-maintenance-workshops (accessed Jul. 21, 2025).

ACKNOWLEDGEMENTS

Firstly, I would like to acknowledge the presence of the Almighty God for the opportunity granted and the blessing every day. Also, I would like to recognize the guidance, advice and training I received from Professor Atanga D. Funwie, Professor Norbert Tcwaffe, Professor Akah Roland Tiagha, Professor Franklin Tchakounte , Dr Pauline W. Gitiha, Frida Ondoru and the entire technical team from Kesmonds International University. For sure, it was an interactive and meaningful study.

Not leaving a stone unturned, I would also like to give special thanks to senior in profession Eng. Tadeo Byabagambi, Asst. Commissioner Biomedical Ministry of Health and all other colleagues in the profession for the academic courage and assistance rendered to me. Furthermore, my sincere gratitude goes to all government hospitals where I conducted my research. Thank you for rendering me with the required authentic data to conclude my university assignment.

CONFLICTS OF INTEREST

The authors declare no conflict of interest in relation to this work.

HOW TO CITE

Moses Lopuka and Atanga Desmond Funwie. (2026). Quality Assurance and Calibration of Medical Devices Practices: Case Study of Government Regional Referral Hospitals in Uganda. IQ Research Journal, 5(2), IQRJ-V05I02-26004020. www.iqresearchjournal.com