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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

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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.

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CONFLICTS OF INTEREST

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

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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

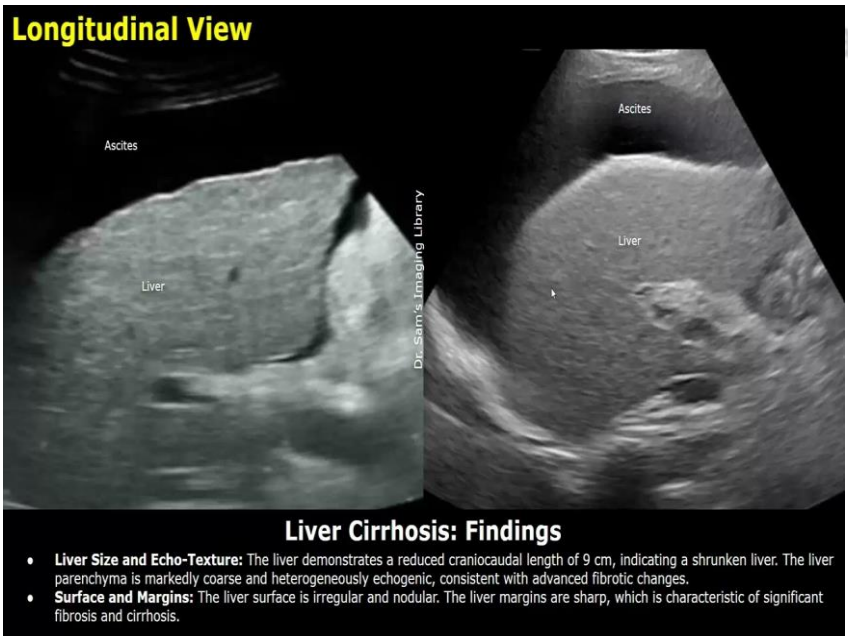

Figure 1. *Liver Cirrhosis and Complications by Ultrasounds*

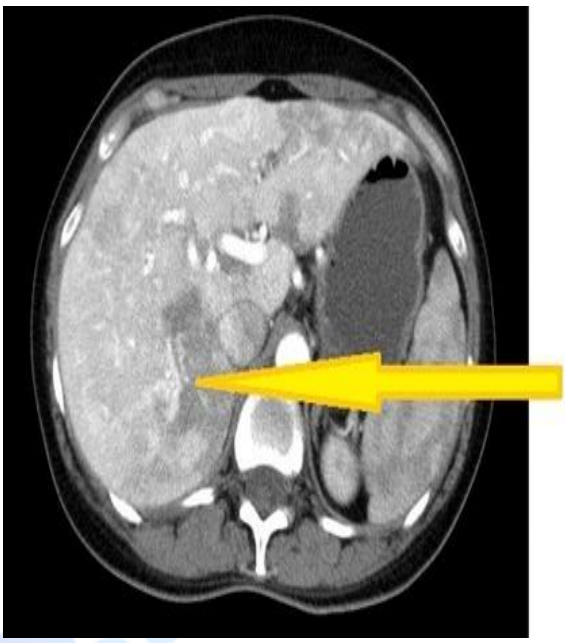

Figure 2. *Liver Cirrhosis and Complications by Contrast – Enhanced CT Scan*