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Colour Doppler Ultrasound of the Hepatic Veins and its Association with Child Pugh's Score and Oesophageal Varices

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Abstract: ***Background: Liver cirrhosis is characterized by extensive fibrosis and the formation of regenerative nodules, resulting from repeated episodes of hepatocyte necrosis and degeneration. This condition leads to irreversible hepatic scarring, significantly impairing liver function and giving rise to various severe complications, including portal hypertension, ascites, Hepatic encephalopathy, hepatorenal syndrome, and esophageal varices, and spontaneous bacterial peritonitis. Among these, portal hypertension represents one of the most critical complications of liver cirrhosis, often manifesting as esophageal varices, gastric varices, and portal hypertensive gastropathy. The clinical assessment of chronic liver disease is commonly performed using the Modified Child-Pugh Scoring System. Portal hypertension can be evaluated through both invasive and non-invasive methods. Doppler ultrasound, a non-invasive imaging modality, has demonstrated potential in assessing liver hemodynamics. Parameters such as hepatic vein waveform, splenoportal index, and damping index provide valuable insights into the severity of liver disease and the associated risk regarding esophageal varices. Objectives: To evaluate the association between hepatic venous doppler parameters with severity of cirrhosis assessed with Child Pugh's score and oesophageal varices. Methodology: A cross-sectional study was conducted on 51 patients to evaluate the association between hepatic venous Doppler parameters and the severity of cirrhosis, assessed using the Child-Pugh score, as well as the presence of esophageal varices. The study included patients aged 18 to 70 years who were diagnosed with cirrhosis. Exclusion criteria comprised coexisting cardiac or respiratory diseases, hepatocellular carcinoma, non- cirrhotic portal fibrosis, and a history of endoscopic variceal ligation or sclerotherapy. A comprehensive evaluation was performed, incorporating patient history, physical examination findings, laboratory investigations, and Doppler ultrasound results. Patients were stratified into three groups—Child-Pugh class A, B, and C—based on the Child-Pugh scoring system, which includes characteristics in the laboratory (serum bilirubin, serum albumin, and international normalized ratio [INR] of prothrombin time) and clinical findings. Data were recorded using a standardized template and analyzed using Microsoft Excel. Statistical analysis was performed using unpaired t-tests and one-way ANOVA, with a p-value of <0.05 considered statistically significant. Results: The study enrolled 51 patients with a male-to-female ratio of 1.4:1 (58.8% males, 41.2% females), categorized into three age groups: 20–40 years (27.5%), 41–60 years (49.0%), and 61–70 years (23.5%). The majority of patients (54.9%) were classified as Child- Pugh Class C, followed by Class B (27.5%) and Class A (17.6%). Ascites was present in 49.0% of patients, significantly associated with the Child-Pugh classification (P = 0.0036), being most common in Class C. Encephalopathy, observed in 7.8% of patients, was exclusively found in Class C, though there was no statistically significant correlation. (P = 0.1682). Hepatic waveforms included biphasic (43.1%), monophasic (41.2%), and triphasic (15.7%), with a significant correlation to Child-Pugh classification (P = 0.0015). The damping index >0.6 was noticeably more common in Class C patients (P < 0.001). Abnormal splenomegaly was present in 56.9% of patients and significantly associated with Child-Pugh class (P = 0.0097). The splenoportal index varied across groups, but no statistical significance was found. Esophageal varices were observed in 60.8% of patients, significantly associated with both Child-Pugh classification (P = 0.0299) and hepatic waveform patterns (P = 0.0041). Conclusion: This study emphasizes the utility of hepatic venous Doppler ultrasonography in assessing liver cirrhosis severity and related complications, such as esophageal varices. Male patients predominated, with the highest prevalence in the 41–60 age group. Doppler parameters correlated strongly with the Child-Pugh classification, with a transition from triphasic to monophasic waveforms indicating worsening of both portal hypertension and cirrhosis. The Doppler index (DI) was significantly elevated in advanced cirrhosis (Child-Pugh Class C), marking it as a reliable non-invasive indicator. While the splenoportal index (SPI) showed limited clinical relevance, esophageal varices were closely linked to Child-Pugh scores and Doppler findings. These results affirm the role of Doppler ultrasonography as a complementary tool to traditional cirrhosis staging methods.***

Keywords: Oesophageal Varices, Portal Hypertension, Cirrhosis, Child-Pugh Score, Non-invasive methods, Doppler ultrasound, hepatic vein waveform, splenoportal index, damping index

1. Introduction

Cirrhosis Overview

According to the World Health Organization (WHO), cirrhosis is "a diffuse process characterized by fibrosis and conversion of normal liver architecture into structurally abnormal nodules" Liver Cirrhosis is the ultimate stage of chronic liver disease, characterized by large-scale fibrosis and the formation of regenerating nodules, which are the result of repeated episodes of necrosis and degeneration of hepatocytes. Both the liver parenchyma and the hepatic vasculature, including the structural abnormalities of the hepatic vein, are altered in liver cirrhosis. Additionally, it is acknowledged that cirrhosis of the liver results in persistent aberrant circulation brought on by intrahepatic shunt and hyperdynamic circulation.

Global Burden of Cirrhosis

Liver cirrhosis is a significant reason of mortality and morbidity across the world. It's the 11th leading cause of death and 15th leading cause of morbidity. Liver Cirrhosis was responsible for nearly 1.32 million deaths globally in 2019 according to the Global Burden of Disease Study.

Various complications associated with Cirrhosis

Cirrhosis leads to the irreversible scarring of hepatic tissue, profoundly impairing liver function and giving rise to a multitude of severe complications, including portal hypertension, ascites, esophageal varice, gastric varices, portal hypertensive gastropathy, and spontaneous bacterial peritonitis. Moreover, cirrhosis significantly elevates the risk of hepatocellular carcinoma, further compounding the disease burden. Portal hypertension is one of the most serious complications of liver cirrhosis, defined as "a wedged hepatic vein pressure or direct portal vein pressure of more than 5 mmHg greater than the inferior vena cava pressure or surgically measured portal venous pressure of greater than 30 cm water." Portal hypertension can be manifested with portal hypertensive gastropathy, gastric varices and esophageal varices. If portal pressure upsurges greater than 12 mm Hg, esophageal varices will be formed. The frequency of esophageal varices in patients with upper gastrointestinal bleeding is from 2 to 9%.

Even with advancements in diagnosis and treatment, up to 20% of people may still die from acute variceal hemorrhage. Moreover, it is the second most frequent reason why people due to cirrhosis.

Clinical Assessment

Clinical assessment of chronic liver disease is done by Modified Child Pugh's and Model for end-stage liver disease (MELD) Scoring system. Child and Turcotte first presented the scoring method for cirrhosis prediction in 1964, and Pugh later updated it in 1974. The Child-Pugh classification system is widely used to evaluate the prognosis of cirrhosis. It incorporates five clinical and laboratory parameters (bilirubin, albumin, INR, ascites, and encephalopathy) to classify the severity of cirrhosis into three classes: A, B, and C. Additionally, serum biomarkers such as FibroTest, HepaScore, and the aspartate aminotransferase-to-platelet ratio index have been proposed as alternatives to liver biopsy, although their specificity and accuracy remain suboptimal due to their dependence on non-hepatic factors. Previous studies have suggested a correlation between Child-Pugh classification and the presence of Esophageal Varices, but there is limited data on how non-invasive Doppler ultrasound parameters correlate with this classification system.

Evaluation Methods

Portal hypertension is evaluated by invasive and non-invasive methods. Invasive methods include direct portal vein pressure and HVPG measurement while Non- invasive methods are Ultrasonography, colour and spectral Doppler evaluation, Computed Tomography (CT) scan and Magnetic Resonance Imaging (MRI). HVPG is considered the gold standard for diagnosis of portal hypertension. Currently, the most commonly used parameter for portal pressure is the HVPG. The normal HVPG is 1-5 mmHg. HVPG $\geq$ 10 mmHg is clinically significant and is predictive of the emergence of cirrhosis complications while the HVPG above 12 mmHg is a risk for variceal rupture. However, HVPG measurement is intrusive, costly, and not always accessible. Thus, there is a need for non-invasive methods to accurately diagnose and predict portal hypertension and oesophageal varices.

Doppler Ultrasound

Doppler ultrasound is a non-invasive imaging technique that has shown promise in evaluating liver hemodynamics. Parameters such as hepatic vein waveform and splenoportal index are derived from doppler ultrasound measurements and have been investigated for their potential to predict portal hypertension and its complications. The hepatic vein waveform reflects the blood flow pattern within the hepatic veins. There are three types of Hepatic vein waveform: - Monophasic: uniform waveform, Biphasic: no reversed stream with or without diminished phasic fluctuation and Triphasic: typical shape. A triphasic waveform with two negative and one positive wave is seen in healthy humans. Monophasic and Biphasic Hepatic vein waveform are accompanied with severe portal hypertension. While the splenoportal index is a composite measure of spleen size and portal vein velocity. Damping Index (DI) is a quantitative method for measuring the little changes of HV waveform. These parameters can provide valuable insights into the severity of liver disease and the risk of Esophageal varices.

Research Question:

- What is the association between hepatic venous Doppler parameters and the severity of cirrhosis as assessed by the Child-Pugh score?
- How do hepatic venous Doppler parameters correlate with the presence and severity of esophageal varices?
- How do both hepatic venous Doppler parameters and esophageal varices collectively correlate with the severity of cirrhosis as assessed by the Child-Pugh score?

2. Aim & Objectives

Aim:

To evaluate the association between hepatic venous doppler parameters with severity of cirrhosis assessed with Child Pugh's score and oesophageal varices.

Objectives:

- To evaluate the association of hepatic venous doppler with oesophageal varices.
- To evaluate the association of both with severity of cirrhosis as assessed with Child Pugh's score.

3. Background & Review of Literature

The histological development of regenerating nodules encircled by fibrous bands in response to chronic liver injury, which results in portal hypertension and end-stage liver disease, is known as cirrhosis.

Epidemiology:

With almost 2 million deaths annually, chronic liver disease (CLD) is a leading cause of morbidity and mortality globally. Furthermore, since 1980, the global cirrhosis mortality rate has increased by 46%. Diffuse fibrosis of the liver parenchyma and the transformation of normal hepatic architecture into structurally aberrant nodules characterize cirrhosis of the liver, the last pathway for various forms of CLDs. Cirrhosis and liver cancer are currently the 11th and 16th leading causes of death worldwide, accounting for 1.16 million and 788,000 deaths annually, respectively.

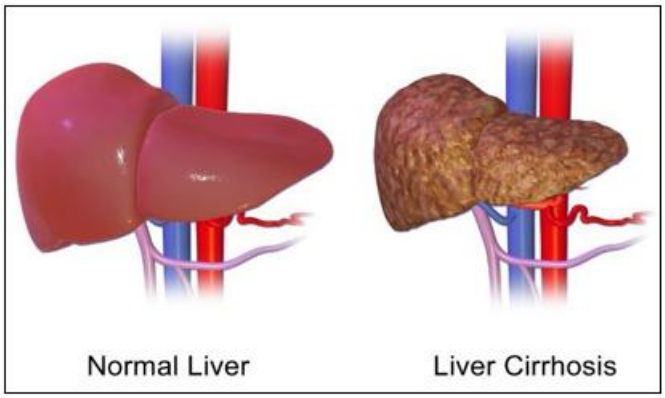


Figure 1: Schematic diagram showing difference between normal liver and liver cirrhosis

Etiology:
Viral hepatitis and alcohol are the main causes of liver cirrhosis. Hemochromatosis, Wilson's disease, autoimmune illnesses, fatty liver disease, various hereditary metabolic abnormalities, primary biliary cirrhosis, and primary sclerosing cholangitis are additional causes of liver cirrhosis.

Etiology of Cirrhosis
• Viral hepatitis • Alcohol • Autoimmune disease • Hemochromatosis • Wilson's disease • Primary biliary cirrhosis • Primary sclerosing cholangitis

Chart 1: Etiology of cirrhosis

Alcohol:
A significant risk factor for liver disease in general and liver cirrhosis in particular is alcohol consumption. In actuality, alcohol usage is responsible for roughly half of the morbidity and mortality burden of liver cirrhosis. Although alcohol use plays a role, liver disease is becoming more widely acknowledged as a complex disease process.

Different codes have been established for categories of liver disorders that are thought to be largely caused by alcohol due to the significance of alcohol in the etiology of liver disease. As a result, the International Classification of Diseases (ICD-10) acknowledges a number of stages of alcoholic liver disease (ICD-10, K70), which can vary from relatively mild and reversible stages like alcoholic hepatic steatosis (fatty liver) (K70.0) and alcoholic hepatitis (K70.1) to more severe and irreversible stages like alcoholic liver cirrhosis (K70.3) and alcoholic hepatic failure (K70.4). It has been determined that alcohol usage, especially long-term heavy use, plays a critical role in the development of many illnesses. But because liver disorders are complex, alcohol consumption may contribute to the development of all forms of cirrhosis, and even one drink daily may have an impact on the prevalence of liver cirrhosis.

Viral hepatitis:
Hepatitis is defined as inflammation of the liver that can result from a variety of causes, such as heavy alcohol use, autoimmune disorders, drugs, or toxins. However, the most frequent cause of hepatitis is due to a viral infection, referred to as "viral hepatitis."

The majority of cases of viral hepatitis result from hepatotropic viruses A, B, C, D, and E. It is unclear whether the hepatitis G virus (HGV) is pathogenic in humans. Other less common causes of viral hepatitis are cytomegalovirus (CMV), Epstein-Barr virus(EBV), herpes simplex virus (HSV), and varicella-zoster virus (VZV). These are non-heterotropic viruses that usually do not primarily target the liver and rarely cause hepatitis in the immunocompetent state.

Auto immune diseases:
A rare kind of chronic liver inflammation, autoimmune hepatitis starts as acute hepatitis and develops into chronic liver disease. Without a strong index of suspicion and sufficient test support, it can be challenging to diagnose because of its wide range of clinical manifestations, which include acute hepatitis and chronic liver illnesses such alcoholic liver disease and chronic viral hepatitis.

Based on the antibodies implicated, autoimmune hepatitis is classified as either autoimmune hepatitis-1 or autoimmune hepatitis-2. Because autoimmune hepatitis is so susceptible to immunosuppressive drugs, it is important to keep a high suspicion index for the condition because early detection can be crucial to improving the prognosis.

Fatty liver disease:
NAFLD includes both nonalcoholic fatty liver (NAFL) and nonalcoholic steatohepatitis (NASH) which is diagnosed when there is evidence of inflammatory activity and hepatocyte injury in a steatotic liver tissue. NAFLD has become the most frequent reason for long-term liver damage. The global prevalence of NAFLD was estimated to be about 24% . Cirrhosis is an important factor for liver-related morbidity and mortality in patients with NAFLD. Minor deposition of fat can occur in the liver of healthy adults, deposition of fat in at least 5% of hepatocytes is considered pathologic.

Metabolic disorders:
Patients with liver cirrhosis often experience protein-energy malnutrition, which is intimately associated with problems of energy and protein metabolism. It has been shown that throughout the protein metabolism process, the albumin's half-life in the blood increases, while its synthesis and breakdown rates decline. Although there is disagreement over energy expenditure in relation to energy metabolism, it is generally agreed that patients with liver cirrhosis have lower respiratory quotients than healthy individuals. The prognosis of patients is also strongly correlated with these metabolic abnormalities.

Hemochromatosis:
Systemic iron overload of hereditary origin, or hemochromatosis, is characterized as a decrease in the concentration of the iron-regulating hormone hepcidin or a decrease in the binding of hepcidin to ferroportin. Ferroportin is the sole known cellular iron exporter, and hepcidin controls its activity. Homozygous mutations in HFE, which codes for the hereditary hemochromatosis protein, notably the C282Y mutation, cause the most

prevalent kind of hemochromatosis. Cirrhosis and persistent liver disease might result from this iron overload.

Wilsons's disease:
Impaired copper metabolism is a hallmark of Wilson's disease (WD), which is caused by a faulty ATP7B protein product. Its clinical outcomes range from an asymptomatic condition to neurological and mental symptoms, chronic liver disease with or without cirrhosis, and fulminant hepatic failure. To avoid missing WD cases, particularly less florid ones with only a slight rise of transaminases or limited neuropsychiatric involvement, a high degree of suspicion is warranted.

Primary biliary cirrhosis
The three hallmarks of primary biliary cirrhosis (PBC) are circulating anti-mitochondrial antibodies, nonsuppurative destructive cholangitis, and interlobular bile duct destruction. PBC is an autoimmune, slowly progressing, cholestatic liver disease. Fatigue, pruritis, jaundice, xanthomas, osteoporosis, and dyslipidemia are among the most noticeable clinical symptoms. Primarily, ursodeoxycholic acid is the treatment.

Primary sclerosing cholangitis:
Primary sclerosing cholangitis (PSC) is a chronic and progressive cholestatic liver disorder of unknown etiology. PSC is characterized by inflammation, fibrosis, and stricturing of intrahepatic or extrahepatic biliary ducts. PSC is usually a progressive disorder that leads to complications of cholestasis and liver failure. Median survival from the time of diagnosis to death without liver transplantation is around 10 years.

Symptoms:
The onset of symptoms of cirrhosis is usually insidious with fatigue, weakness, and muscle wasting, sometimes with abdominal distention and ascites, peripheral edema or variceal haemorrhage. Patients may have pruritus and jaundice depending upon the severity of the cirrhosis. Patients may improve markedly with stopping therapy, but the improvement is slow and there may be a period of worsening signs and symptoms when the medication is first stopped.

Pathology:
A complex network of cytokine-mediated signaling mechanisms that control fibrogenesis and HSC activation choreograph liver cirrhosis.

PDGF (Platelet derived growth factor)
Of all the polypeptide growth factors, PDGF is the most potent mitogen for HSCs. PDGF-A, -B, -C, and -D are the four members of the PDGF family. In fibrous tissues, PDGF and its receptors are noticeably overexpressed, and the more severe the liver fibrosis, the higher the activity of PDGF. A number of things, including chemicals, viruses, or mechanical harm to hepatocytes, can cause KCs to produce and release PDGF. When PDGF attaches to its particular receptor on the HSC membrane, it triggers the activation of related signal molecules and transcription factors, which in turn triggers the activation of its downstream target genes and HSCs.It has been demonstrated that PDGF reduces ECM degradation by upregulating the production of MMP-2, MMP-9, and TIMP-1 and by inhibiting collagenase activity.

TGF-β (Transforming growth factor-β):
In hepatic fibrosis, TGF-β is the most potent known inducer of fibrogenesis. In the liver, hepatocytes, Kupffer cells, liver sinusoidal endothelial cells (LSECs), and HSCs/myofibroblasts are the primary producers of TGF-β. There are six members of the TGF-β1 family, and it has been demonstrated that TGF-β1 is essential for the development and upkeep of liver fibrosis. Fibrotic livers have higher levels of TGF-β1 expression, which peaks at cirrhosis. TGF-β1 has a complex pro-fibrogenesis action that involves several factors. Its main function is to increase HSC activation, and the TGF-β1 autocrine loop in activated HSCs provides a significant positive feedback to the advancement of liver fibrosis.

TNF-α (Tumor necrosis factor-α):
Monocytes, macrophages, HSCs, and KCs are the primary producers of TNF-α. In these cells, it exhibits cytotoxic and proinflammatory properties. TNF-α is crucial for the stimulation of HSCs and the production of extracellular matrix during the liver fibrosis process. By downregulating the proapoptotic protein p53 and upregulating the antiapoptotic factors NF-κB, Bcl-XL, and p21WAF1, TNF-α can decrease the spontaneous apoptosis of activated rat HSCs. However, research indicates that TNF-α may cause apoptosis in HSCs, illustrating the complex and sometimes contradictory effects of TNF-α on HSCs and fibrosis.

Pathogenesis:
Encapsulation or replacement of damaged tissue by a collagenous scar is referred to as fibrosis. Liver fibrosis is caused by an aberrant continuation of fibrogenesis, or the creation and deposition of connective tissue, as a result of the normal wound healing response. Depending on host, environmental, and liver disease causes, fibrosis advances at different speeds. A more severe form of liver fibrosis, cirrhosis is characterized by hepatic vascular distortion.

The interchange between the hepatic sinusoids and the nearby liver parenchyma, or hepatocytes, is compromised as a result of the portal and arterial blood supply being diverted straight into the hepatic outflow (central veins). The fenestrated endothelia that border the hepatic sinusoids lie on top of the space of Disse, a sheet of permeable connective tissue that contains some mononuclear cells and hepatic stellate cells (HSC). Hepatocytes, which carry out the majority of the known liver functions, line the other side of the Disse space. In cirrhosis the space of Disse is filled with scar tissue and endothelial fenestrations are lost, a process termed sinusoidal capillarization. Vascularized fibrotic septa that connect portal tracts to one another and to central veins are histologically indicative of cirrhosis. This results in hepatocyte islands that are encircled by fibrotic septa and lack a central vein.

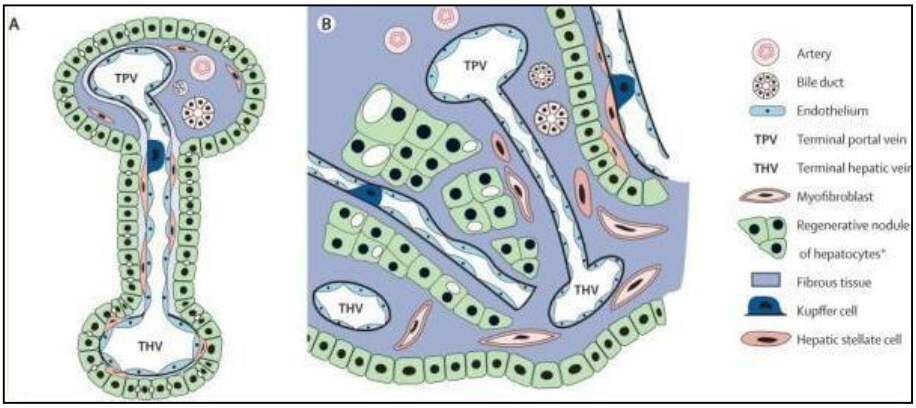


Figure 2: Changes in cirrhosis's architecture and vascular structure (A. Normal liver, B. Cirrhosis liver)

Complications associated with cirrhosis

Portal hypertension

Increased pressure in the portal vein, the blood artery that connects the liver to the spleen and gastrointestinal tract (splanchnic organs), is known as portal hypertension. Increased intrahepatic vascular resistance brought on by compromised hepatic sinusoidal circulation—which most commonly results from chronic liver disorders (CLDs)—causes portal hypertension. In addition to changes in cellular phenotypes linked to dysfunction of liver sinusoidal endothelial cells (LSECs), activated hepatic stellate cells (HSCs), and inflamed resident or infiltrating macrophages, CLDs cause structural changes of the liver through increased extracellular matrix (ECM) accumulation and turnover (fibrosis). Increased intrahepatic resistance brought on by these alterations raises portal vein pressure (portal hypertension), which is the first step toward CLD consequences. A hyperdynamic circulatory state develops as a result of portal hypertension's secondary induction of splanchnic and systemic arterial vasodilation, which exacerbates and fuels clinically harmful consequences. Complications from cirrhosis, including ascites and hemorrhaging gastro-oesophageal varices, as well as hepatic encephalopathy from portosystemic shunting, hepatorenal syndrome, and hypersplenism, are caused by portal hypertension.

Oesophageal Varices:

Patients with cirrhosis and portal hypertension may develop esophageal varices, which are portosystemic collaterals. A portion of cirrhosis patients (3–12%) each year develop esophageal varices, with 8–12% of patients showing signs of progression from mild to big varices. Small esophageal varices may also spontaneously recede, usually after alcohol cessation in cases of alcoholic cirrhosis.The most significant predictors of variceal bleeding are thought to be the size of the varices, the presence of red patches, and the degree of cirrhosis.

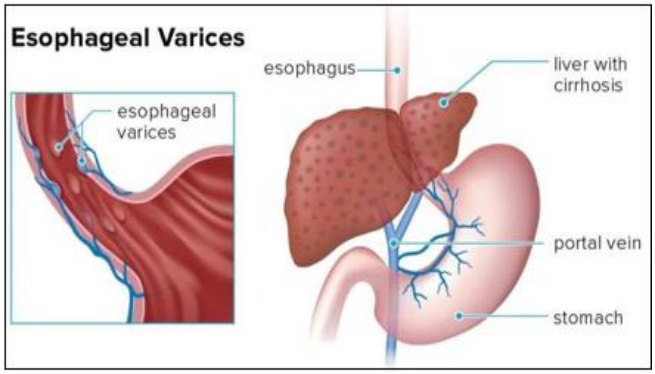


Figure 3: Esophageal varices

With different incidence across the globe, these are among the most frequent causes of acute upper gastrointestinal bleeding (UGIB). They are the main reason why people die from UGIB. A major economic and public health concern, acute variceal hemorrhage (AVB) is a potentially lethal consequence of clinically substantial portal hypertension (CSPH).

Classification of oesophageal varices:

While GV are categorized as either isolated gastric varices (IGV) or gastroesophageal varices (GOV), esophageal varices are categorized by size (small, medium, or large) and the presence of red wale markings.

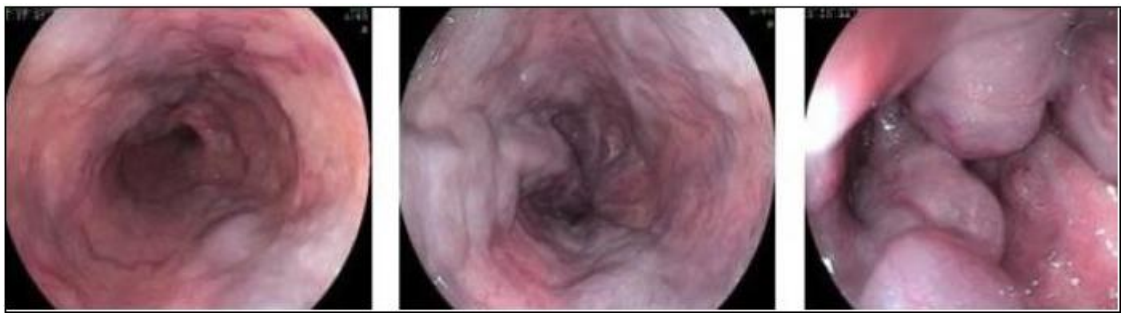


Figure 4: Oesophageal varices small medium and large respectively

Classification of esophageal varices	
Form	F1: varices having a straight shape that do not go away after insufflation F2: tortuous varices that are somewhat expanded and take up less than one-third of the esophageal lumen F3: large-sided varices that take up over one-third of the lumen of the esophagus
Fundamental color	White (CW) Blue (CB)
Red color sign (RC)	Red Wale Marking (RWM) Cherry Red Spot (CRS) Hematocystic Spot (HS) Diffuse Redness (DR
Location	Varices above the level of the tracheal bifurcation are known as locus superior (Ls). Varices near or at the level of the tracheal bifurcation are known as locus medialis (Lm). Varices in the region that includes the lower thoracic esophagus and abdomen are known as locus inferiorior (Li)
Esophagitis	Esophagitis positive (E+) Esophagitis negative (E−)

Chart 2: Classification of esophageal varices

Etiology:

The etiology is varied, and the frequency of etiology is the following: 60-65% of the hemorrhagic episodes are caused by esophageal varices, approximately 7% of gastric varices, 5-8% of hypertensive portal gastropathy, and 5-15% of gastric and duodenal ulcers. The mortality attributed to the hemorrhagic episode is substantial, estimated at 13-19% of overall mortality in cirrhosis. The mechanism of increasing portal pressure depends on the location and causes of portal hypertension. Causes of portal hypertension are: portal, splenic and superior mesenteric vein thrombosis, primary biliary cirrhosis, sclerosis cholangitis, cirrhosis, veno-occlusive disease, Budd Chiari syndrome, congestive heart failure, vitamin A toxicity, idiopathic portal hypertension and infiltrative disorders (lymphoproliferative and myeloproliferative diseases).

Hepatic Encephalopathy

Hepatic encephalopathy (HE) is defined as brain dysfunction due to acute, acute on chronic or chronic liver failure and/or portal-systemic shunting. It manifests as a wide spectrum of neuropsychiatric symptoms ranging from subclinical mental alterations to gross confusional state and coma in advanced stage. HE is probably the most frequent complication of cirrhosis, the prevalence of HE at the time of diagnosis of cirrhosis is 10–14%. Around 30–40% of cirrhosis patients develop HE at some time during their clinical course. The cumulative risk of developing recurrent HE is 40% at 1 year. The one-year mortality in patients with severe grade of HE is up to 42%. HE, which can manifest as many different neurological or mental diseases, from asymptomatic to coma, is a generic term for brain dysfunction brought on by hepatic insufficiency and/or portal-systemic shunting. The underlying cause of liver illness is not taken into account in this definition of HE. However, chronic liver diseases (CLDs), including viral hepatitis, primary biliary cholangitis, alcohol-related liver disease, and non-alcoholic fatty liver disease, can all harm the brain through mechanisms unrelated to liver loss or dysfunction. A liver transplant (LT) is considered to be able to reverse the metabolic disorder known as HE. However, several studies have demonstrated that HE is characterized by neuroinflammation and neuronal cell death, and that extended durations of overt HE might have irreversible effects. These manifest as ongoing neurological issues following LT. More significantly, regardless of the severity of the liver disease, HE has been related to a high risk of mortality, suggesting that it is a sign of hepatic insufficiency, but it may also have distinct implications for pathophysiology and prognosis.

Ascites

A significant side effect of cirrhosis, ascites affects 50% of patients during a 10-year follow-up period. Since ascites is linked to a 50% mortality rate over two years and indicates the need to explore liver transplantation as a treatment option, it represents a significant turning point in the natural history of cirrhosis. Cirrhosis is the underlying cause of ascites in 75% of individuals who present with it; the remaining 10% are caused by cancer, 3% by heart failure, 2% by tuberculosis, 1% by pancreatitis, and the last 1% by other uncommon causes. About 10–20% of people with one of the three most prevalent chronic liver diseases—alcoholic liver disease, chronic hepatitis C, or non-alcoholic fatty liver disease—develop cirrhosis over a ten to twenty-year period, and about 4% of the general population has abnormal liver function or liver disease.

Over the next few years, the burden of liver disease is predicted to rise significantly due to the increasing prevalence of alcoholic and non-alcoholic fatty liver disease, which will inevitably lead to an increase in cirrhosis complications.

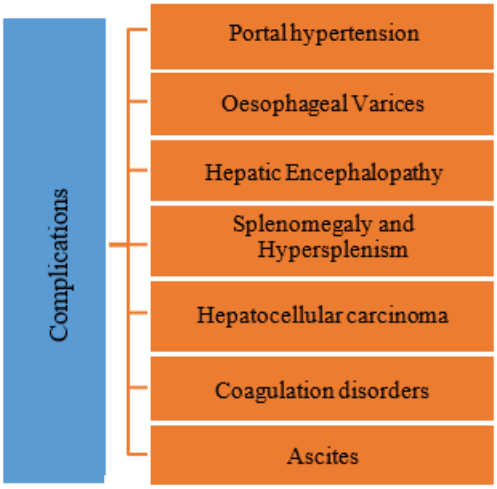


Chart 3: Various complications associated with Cirrhosis.

Splenomegaly and Hypersplenism

Hypersplenism and portal hypertension can result from liver cirrhosis. Due to thrombocytopenia, portal hypertension causes a tendency to bleed and raises the risk of variceal hemorrhage. Splenectomy and devascularization or shunt surgery were required for individuals with secondary hypersplenism, cirrhosis, and bleeding portal hypertension. The hepatitis C virus has been eradicated in individuals with

compensated cirrhosis as well as those without cirrhosis thanks to recent developments in interferon therapy. However, patients with hypersplenism and splenomegaly are suitable candidates for splenectomy because they are unable to undergo such treatment because of thrombocytopenia, leukocytopenia, or both.

Splenomegaly is a frequent finding in patients with liver disease, and it is an important sign of portal hypertension. Some 36% to 92% of patients with cirrhosis have splenomegaly. Some patients with splenomegaly due to cirrhosis or other causes develop thrombocytopenia, neutropenia, or anemia, either alone or in combinations. This condition is called hypersplenism, a term first introduced by Chauffard in 1907. Hypersplenism is characterized by a peripheral blood picture of cytopenia, splenomegaly, normal or hypercellular bone marrow, and a return of the peripheral blood picture to near normal after a splenectomy. However, fulfillment of all of these criteria is not necessary for the diagnosis. Practical criteria that defined hypersplenism as splenomegaly with a platelet count of <150.000/µL and/or a white blood cell (WBC) count of <3500/µL were suggested by Liangpunsakul et al. in 2003 and have largely been adopted.

Hypersplenism, or an overactive spleen, is a well-established syndrome. Banti first described a disorder characterized by anemia and splenomegaly not related to any hematological disorder in 1898. Osler reported on a group of patients with chronic splenomegaly and anemia without cirrhosis but with recurrent gastrointestinal hemorrhage that improved after splenectomy in 1900. Subsequently, a condition called noncirrhotic portal fibrosis, characterized by portal hypertension in the absence of cirrhosis, occurring with splenomegaly and hypersplenism was reported by authors in India, Japan, and elsewhere.

Hepatocellular carcinoma

Hepatocellular carcinoma (HCC) is the most common primary liver malignancy and is a leading cause of cancer-related death worldwide. The male to female HCC incidence ratio worldwide is 2.8:1. Hepatocytes, the fundamental parenchymal cells of the liver, are the source of HCC, a primary liver cancer. Over 80% of primary liver cancer cases worldwide are HCC. Numerous molecular malfunctions, including cell cycle dysregulation, DNA methylation changes, chromosomal instability, immunomodulation, epithelial-to-mesenchymal transition, an increase in HCC stem cells, and microRNA (miRNA) dysregulation, are all part of the complicated pathophysiology of HCC.

The usual sequence is liver injury, chronic inflammation, fibrosis, cirrhosis, and HCC. By activating PRRs like TLR, C-type lectin receptors, NOD-like receptors, and RIG-I-like receptors, the release of molecular mediators like DAMPS and PAMPS triggers an innate immune response and results in inflammation. Most HCCs (80–90%) are preceded by fibrosis and ultimately cirrhosis, which are caused by chronic, unresolved inflammation. Aflatoxin, alcoholic and nonalcoholic fatty liver disease, viral hepatitis, metabolic syndrome, and predisposing genetic factors are some of the causes of HCC, a complex disorder.

Coagulation disorders

There are two types of hemostasis: primary and secondary. The initial line of treatment for endothelial injury is primary hemostasis. Damage to the vascular endothelium causes platelets to become activated and local vasoconstriction to begin. This causes a temporal platelet clog to form and starts secondary coagulation or hemostasis. Multiple coagulation factors are sequentially activated during secondary hemostasis, which eventually leads to the creation of a stable fibrin clot over the platelet plug that has already formed. Ultimately, the fibrinolytic system will eliminate the blood clot that has developed. There are two types of coagulopathy: primary and secondary. Defects in platelets or blood arteries are examples of primary hemostasis disorders. Whereas, secondary diseases entail qualitative or quantitative abnormalities in clotting factors or their inhibitors. One of the main causes of public health issues that lead to morbidity or mortality on a global scale is coagulopathy. Between 5 and 10% of women of reproductive age seek treatment for prolonged bleeding during the menstrual month, while between 26 and 45% of the world's population who are deemed healthy have had nose and gum bleeding in the past.

Reduced synthesis of factors II, V, VI, IX, XI, XIII, fibrinogen, protein C, protein S, vitamin K deficiency from malnutrition or malabsorption, dysfibrinogenemia, enhanced fibrinolysis, diffuse intravascular coagulation, thrombocytopenia, impaired clearance of activated clotting factors, plasminogen activators, and fibrinogen degradation products are all factors that affect hemostasis in patients with severe liver disease. Clinical repercussions of this could include bleeding, thrombosis, and abnormal bleeding tests.

In individuals with decompensated liver cirrhosis, endothelial dysfunction, endogenous heparinoids, infections, and renal failure can all have an impact on coagulation.

In cirrhosis patients, endogenous heparinoids affect coagulopathy. Numerous coagulation tests, such as euglobulin lysis time, plasma fibrinogen, serum fibrinogen degradation product, plasma D-dimer, clotting time, prothrombin time, activated partial thromboplastin time, thrombin time, whole blood clot lysis, and factor assays for F XIII, protein C, protein S, and antithrombin III.

Clinical Assessment:

The Child-Pugh score is frequently used to forecast how cirrhotic patients would fare. To predict mortality in patients with cirrhosis, the Child-Pugh scoring system—also referred to as the Child-Pugh-Turcotte score—was created. It was first developed by Child and Turcotte in 1964 to help select patients who would benefit from elective surgery for portal decompression. Patients were divided into three groups: those with good hepatic function (A), those with moderately impaired hepatic function (B), and those with advanced hepatic dysfunction (C). Serum bilirubin, serum albumin, ascites, neurological illness, and clinical nutrition status were the five clinical and laboratory criteria that were employed in their initial scoring system to classify patients. Later, Pugh et al. changed the scoring system by replacing clinical nutrition status with prothrombin time. They also added changeable points according to increasing severity for each criterion.

Table 1: Child-Turcotte Pugh Classification for Severity of Cirrhosis

	Points*		
	1	2	3
Encephalopathy	None	Grade 1-2 (Or precipitant included)	Grade 3-4 (Or chronic)
Ascites	None	Mild to moderate (Diuretic responsive)	Severe (Diuretic refractory)
Bilirubin (mg/dL)	< 2	2-3	>3
Albumin (g/dL)	> 3.5	2.8-3.5	< 2.8
INR	< 1.7	1.7-2.3	> 2.3
*Child-Turcotte-Pugh Class and liver disease severity obtained by adding score for each parameter (total points)			
• Class A = 5 to 6 points (least severe) • Class B = 7 to 9 points (moderately severe) • Class C = 10 to 15 points (most severe)			

In addition to predicting mortality risk associated with other major procedures, the Child-Pugh score has been validated as a predictor of postoperative mortality following portocaval shunt surgery. This Child-Pugh score can be used to forecast the likelihood of all-cause death and development of further liver dysfunction-related problems, like variceal hemorrhage.

Diagnosis:

Fibrous septa between the portal fields are a histological characteristic of cirrhosis, which can be either micro- or macronodular in shape. Characteristic findings from laboratory testing, ancillary studies, and clinical examinations are used to diagnose the illness.

A hard liver upon probing, cutaneous indicators of liver disease, and specific risk constellations like metabolic syndrome, extensive alcohol use, exposure to hepatotoxic chemicals, and usage of hepatotoxic drugs are typical findings in cirrhosis.

Hepatic tissue inhomogeneity, hepatic surface irregularities, or caudate lobe enlargement are early indicators of cirrhosis in B-ultrasonography. Splenomegaly is a result of portal hypertension. Thrombocytopenia, impaired hepatic biosynthesis (as evidenced by, for example, low albumin and cholinesterase concentrations and an elevated international normalized ratio [INR]), and impaired liver detoxifying function (as evidenced by, for example, elevated bilirubin concentration) are all observed in advanced liver disease that is getting close to the stage of cirrhosis. Transaminase levels are often either slightly increased or within the normal range. No laboratory test has a clearly defined threshold value that can be utilized to decide when cirrhosis screening should be carried out. Liver biopsy is unnecessary, or even contraindicated, if the diagnosis of cirrhosis has been clearly established from the clinical findings and imaging studies (e.g., evidence of decompensation, with ascites and impaired hepatic biosynthesis).

If the cause of liver illness is unknown or the results of the aforementioned tests do not reveal the disease's stage, a liver biopsy is recommended. Transcutaneous liver biopsy is recommended in patients of suspected cirrhosis if the clinical findings raise questions about the diagnosis or if the biopsy is anticipated to provide information regarding the cirrhosis's origin that will influence the treatment decision.

Upper abdomen ultrasonography and gastroscopy are examples of ancillary studies. When cirrhosis is first diagnosed or suspected, esophagogastroduodenoscopy (EGD) should be done to show esophageal varices and determine the likelihood that they will bleed.

Histology is still used for the ultimate diagnosis because ultrasound, computerized tomography (CT), and magnetic resonance imaging (MRI) are not sensitive enough to identify cirrhosis. When there is a clear etiology and imaging shows an inhomogeous hepatic texture or surface, rarefied hepatic central vein, an enlarged caudate lobe, splenomegaly, or collateral veins, their specificity is high. However, compensated cirrhosis cannot be ruled out by normal radiographic results, and alternative etiologies including portal vein thrombosis, parasite infections, or hematological malignancies must be ruled out. Radiography's primary function is to identify and measure cirrhosis consequences, such as ascites, HCC, and portal vein or liver thrombosis.

Ultrasonography is widely accessible, affordable, and offers valuable insights into the architecture of the liver. Although they are also present in steatosis, nodularity and increased liver echogenicity are frequently observed in cirrhosis. Usually, the right lobe atrophy and the left, particularly the caudate lobes, hypertrophy. The caudate's breadth in relation to the right lobe, however, is not a reliable indicator of cirrhosis. Portal hypertension and vascular patency can be detected by ultrasound and Doppler ultrasonography of portal and central vein diameters and velocities. The appearance of echogenic microbubbles in the hepatic vein is examined by contrast ultrasonography. Fibrosis has an inverse relationship with their presence following antecubital injection. The first imaging modality for suspected HCC is ultrasound; however, nodular lesions should be validated by helical CT and/or MRI because ultrasound has a lower sensitivity and specificity to identify HCC than CT or MRI.

Even in the absence of ultrasonographic lesions, these more stringent methods are necessary for pretransplant evaluation or a high degree of suspicion, such as in patients whose alfa-fetoprotein levels are greater than 200 μg/L.

Although they are not yet widely accessible, power Doppler, harmonic imaging, and contrast ultrasonography enhance the identification of HCC by sensitively seeing aberrant vasculature.

While helical CT and MRI with contrast are the preferred modalities when HCC or vascular lesions are detected, conventional CT and MRI are not helpful in determining the severity of cirrhosis. When it came to detecting tiny HCCs that were between one and two centimeters in size, MRI outperformed helical CT. Hepatic iron and fat concentration in hemochromatosis and liver steatosis, respectively, have also been demonstrated to be accurately determined by MRI.

A promising method for measuring elasticity is called Fibroscan, which uses an intercostally positioned transmitter to measure the velocity of an elastic wave. Pulse ultrasonography measures shear wave velocity, which is correlated with fibrosis, or liver stiffness.

The Modified Child Pugh's and Model for End-Stage Liver Disease Scoring System is used to do the clinical evaluation of chronic liver disease. When it comes to measuring portal hypertension in cirrhosis, upper gastrointestinal endoscopy and hepatic venous pressure gradient (HVPG) measurement are the gold standards. HVPG measurement is costly, intrusive, and may cause problems for cirrhotics with coagulopathy. Non-invasive assessment of portal hypertension is required. Doppler for color Patients accept ultrasound as a safe, radiation-free, painless, affordable, and reproducible technique. A useful substitute for assessing the hepatic venous waveform, damping index, and predicting the degree of portal hypertension may be color and spectral doppler ultrasound.

Hepatic vein waveform (HVW):

Three types of hepatic vein waveforms (HVW) are distinguished: Triphasic: typical layout, Biphasic: with or without less phasic oscillation, no reversed flow, and A monophasic waveform is flat. Significant portal hypertension is linked to both biphasic and monophasic HVW. Cirrhosis is indicated by the hepatic veins becoming damp. Additionally, the splenoportal index and hepatic venous waveform are helpful non-invasive indicators for predicting the existence of oesophageal varices.

Hepatic venous pressure gradient (HVPG) measurement:

HVPG measurement has evolved from being mainly used with diagnostic purposes to be considered a useful tool to assess the severity and prognosis of chronic liver disease and LC, including the risk of the complications such as varices bleeding, ascites, encephalopathy, or hepatorenal syndrome.

HVPG measures this resistance using a minimally invasive technique guided by fluoroscopy. HVPG is the computed difference between a wedged and a free venous pressure, as determined by a catheter placed in the hepatic vein, as opposed to direct and more intrusive portal venous sampling.

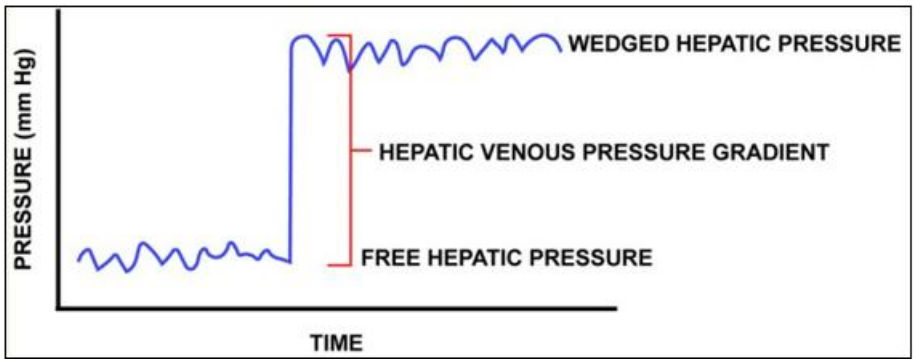


Figure 5: HVPG calculation.

Free hepatic venous pressure represents systemic venous pressure, while wedged hepatic venous pressure (WHVP) is a measure of pressure within the portal venous system. A gradient >5 mm Hg is indicative of portal hypertension, while an HVPG < 5 mm Hg is considered normal. Notably, patients with noncirrhotic causes of portal hypertension may have normal or slightly raised WHVP, which would result in a normal gradient even in cases of severe portal hypertension.

HVPG measures are helpful in assessing the chance that advanced cirrhosis-related varices will form and, if they do, how likely they are to bleed. Variceal development is predicted by values ≥10 mm Hg.

Colour doppler Ultrasound:

Doppler liver ultrasonography constitutes an effective and non-invasive means of evaluating the Doppler ultraound (DU) and color Doppler (CD) are routinely included in the study of the abdomen because they contribute greatly to the immediate differentiation of vascular and nonvascular structures.

Furthermore, DU can detect changes in the direction of blood flow, flow velocity, and organ perfusion. DU information concerning the hepatic vasculature has been obtained faster and more easily because of new advances in technology and instrumentation, producing a profound impact on the evaluation of hepatic cirrhosis (HC) and chronic hepatitis (CH). Duplex Doppler imaging and CD optimize flow information in patients with these conditions.

In the evolution of hepatic disease, it is useful and necessary to obtain the following DU information: (1) the flow direction in the portal vein, (2) the presence of portal hypertension and portosystemic collaterals, (3) occlusion of portal or splenic veins, (4) pre- and postprocedural assessment of transjugular portosystemic shunts (TIPS), and (5) vascular characterization of hepatocellular carcinoma (HCC) versus benign focal lesions. Colour Doppler ultrasound of the hepatic veins has emerged as a non-invasive technique for the diagnosis of portal hypertension and to predict oesophageal varices. The normal hepatic vein waveform is triphasic-retrograde A wave and antegrade S and D waves.

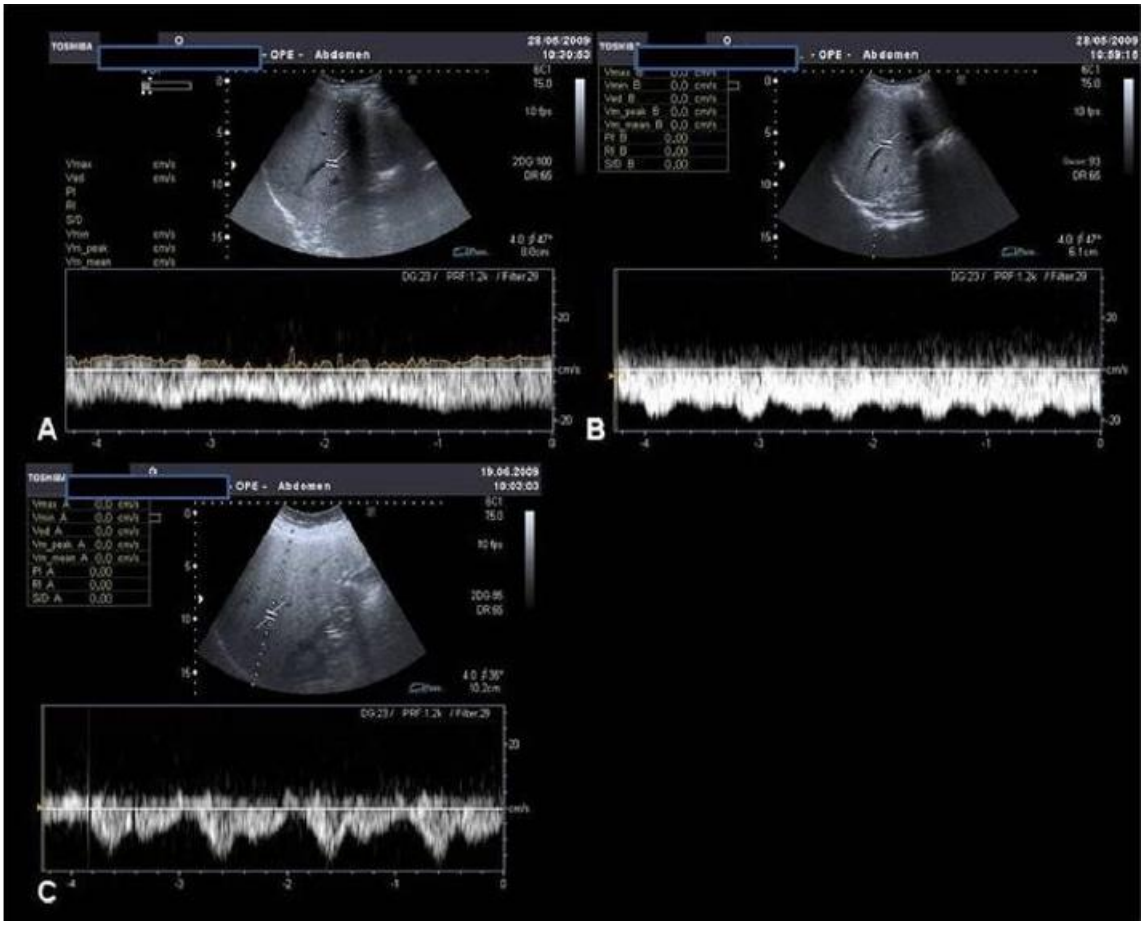


Figure 6: Spectral waveform monophasic or flat waveform, biphasic waveform without a reverse flow, and triphasic waveform (A, B, C).

Bhutto AR et al. in 2012 studied on the correlation of hepatic venous waveform changes with severity of hepatic dysfunction and grading of oesophageal varices. This cross-sectional analytical study involved 65 patients. Among these 51 (78.5%) were males while 14 (21.5%) were females. The average age was 47.39 +/- 10.91 years, with a range of 23-70 years. 32 patients (49.2%) were classified as Child-Pugh Class A based on their liver function, 23 patients (35.4%) as Class B, and 10 patients (15.4%) as Class C. In five cases (7.7%), the hepatic venous waveform was triphasic; in eighteen cases (27.7%), it was biphasic; and in forty-two cases (64.6%), it was monophasic. These waveforms were significantly correlated with hepatic dysfunction ($p < 0.012$), but not with oesophageal varices grading (p 0.29). Upper GI endoscopy revealed large grade varices in 37 (56.9%) patients, 17 (26.2%) patients had small grade varices while no varices were found in 11 (16.9%) patients.The study concluded that Hepatic venous waveform pressure changes have significant relation with severity of hepatic dysfunction but insignificant relation with grading of oesophageal varices. To develop indices with a higher predictive value, more research utilizing a mix of several Doppler characteristics is needed.

In 2016, **Antil N et al.** sought to predict the presence of oesophageal varices and the degree of portal hypertension in patients with cirrhosis by evaluating the hepatic venous waveform, damping index, and splenoportal index on color Doppler ultrasound. The study comprised 30 patients with chronic liver disease, 8 of whom were male and 22 of whom were female. Twenty-two (73.3%) patients had monophasic waveform. In four cases (13.3%), biphasic and triphasic waveforms were seen. Twenty-two patients (73.3%) had monophasic waveforms and majority of them were in class C. This distribution of hepatic vein waveform was statistically significantly with the Child Pugh's class ($p<0.05$). Twenty patients (66.7%) had value of Damping index more than >0.6 where majority of patients (18) belonged to class C and 2 in class B. There was a positive correlation between Child Pugh's total score and Damping index ($r=0.614$; $p<0.05$). There was weak positive correlation between splenoportal index and Child Pugh's score ($r=0.269$; $p=0.15$). The study concluded that Change in triphasic to monophasic waveform and DI >0.6 suggests severe liver dysfunction and is associated with severe portal hypertension. Hepatic venous waveform pressure changes, DI and SPI have no value in predicting presence of oesophageal varices.

The association between the degree of aberrant Doppler HV waveforms, as measured by the damping index (DI), the hepatic venous pressure gradient (HVPG), and the reaction to propranolol in cirrhosis patients was prospectively assessed by **Kim MY et al.** in 2007. Both the DI of the Doppler HV waveform and the HVPG were measured in 76 cirrhosis patients (69 males and 7 women), and their correlation was examined. In 66 out of 76 individuals, abnormal HV waveforms were seen (86.8%). DI and HVPG grade were strongly connected; that is, higher HVPG was associated with higher DI ($P<0.01$). DI>0.6 was substantially

more likely to be severe portal hypertension according to logistic regression analysis (odds ratio: 14.19, 95% CI: 4.07-49.55). The receiver-operating characteristic curve indicated a sensitivity of 75.9% and a specificity of 81.8% for the presence of severe portal hypertension based on the DI value of 0.6. The change in DI after propranolol treatment also showed a significant correlation with that of HVPG in 19 patients in the propranolol subgroup (P<0.01). The author came to the conclusion that Doppler ultrasonography's Damping Index of the HV waveform could be a non-invasive additional tool for assessing the degree of portal hypertension and how well patients with liver cirrhosis respond to propranolol.

Sudhamshu KC et al. in 2006 conducted a study on Doppler study of hepatic vein in cirrhotic patients and correlation with liver dysfunction and hepatic hemodynamics. Total 160 subjects were involved in the study (One hundred patients with liver cirrhosis and 60 non-cirrhotic controls). Hepatic vein waveforms were classified into three classical patterns. Flat waveform was uncommon. All non- cirrhotic controls showed triphasic waveforms of HV. In cirrhotic patients triphasic pattern was observed in 49 (49%) patients, biphasic in 48 (48%) patients and flat in 3 (3%) patients. Out of 3 patients, 2 were Child-Pugh A and were clinically stable. Hepatic encephalopathy and ascites were absent in all 3 patients. Mean hepatic vein velocity was significantly higher in cirrhotic patients (12.7 ± 6.4 vs 5.1 ± 2.1 and 6.2 ± 3.2 cm/s; P < 0.0001). The poorer the grade of cirrhosis, the higher was the mean velocity. Maximum forward velocity was never greater than 40 cm/s in controls. Degree of ascites was found to be highly correlated with mean velocity. “Very high” group (≥ 20 cm/s) presented clinically with moderate to massive ascites. Correlations between right portal flow and mean velocity was significant (P < 0.0001, r = 0.687). The study stated that Doppler waveforms of hepatic vein, which is independent of liver dysfunction, should be obtained during normal respiration. The shift in hepatic circulation linked to the advancement of liver cirrhosis is reflected in the mean hepatic vein velocity. It can be applied as a novel metric to evaluate liver cirrhosis.

In 2024, **Khan R et al.** conducted research on the relationship between the Child-Pugh score, splenoportal index, damping index, and hepatic vein waveform patterns in patients with liver cirrhosis. Of the 52 patients in the final cohort, 39 were men (75%) and 13 were women (25%). 55.3 was the average age. From Child-Pugh Class A (0.45 ± 0.10) to Class C (0.75 ± 0.15), the damping index increased significantly (p=0.003). Additionally, there was a noteworthy increase in the splenoportal index from Class A (1.4 ± 0.3) to Class C (2.0 ± 0.5) (p=0.015). The sensitivity and specificity of the damping index (> 0.6) in predicting higher Child-Pugh scores (B + C) were 52.6% and 85.7%, respectively, with a positive predictive value of 90.9%. The study found strong correlations between the severity of liver cirrhosis, as assessed by the Child-Pugh score, and Doppler ultrasound parameters such as hepatic vein waveforms and the damping index. Doppler ultrasound, therefore, presents itself as a precise, non-invasive alternative for evaluating the severity of liver disease, potentially replacing more invasive procedures.

A 2011 study by **Mittal P et al.** assessed the relationship between color Doppler results and the degree of portal hypertension in cirrhosis patients. There were fifty patients in the trial group. Based on the Child Pugh categorization, the patients were split up into three groups (Child' A, B, and C). The patients in this study were 45 years old on average. The largest percentage of patients (42%) were in the Child's C category. Out of fifty individuals, six (12%) had non-hepatopetal flow (hepatofugal/bidirectional), four (8%) had continuous hepatofugal flow, and two (4%) had bidirectional flow throughout the study. Bidirectional or hepatofugal flow was only observed in patients in the Child's C group. Two patients (4%) developed portal cavernoma development, while three patients (6%) had portal vein thrombus, with no flow found in the major portal vein. The most prevalent varices identified by color Doppler were splenic varices, which were observed in 82% of cases. On color Doppler, one or more collaterals were found in 84% of cases overall. Patients who had splenic varices had a substantially lower mean PVV (14.05 ± 4.13) than those who did not (17.77 ± 1.68; P < 0.05). A significant decrease in PVV was also linked to ascites (P < 0.01). Compared to those without esophageal varices (14.74 ± 4.25), those with esophageal varices had a lower mean PVV (14.62 ± 2.62). Nevertheless, P > 0.05 indicated that this difference was not statistically significant. According to the study's findings, Color Doppler is a great tool for identifying and describing the intricate hemodynamics of portal hypertension in cirrhosis, and it correlates with the disease's clinical stage.

In 2002, **Vyas K et al.** conducted research on the evaluation of portal hemodynamics in liver cirrhosis using laser Doppler velocimetry and ultrasound color Doppler. The study included 10 healthy volunteers (7 males) and 28 patients with liver cirrhosis (24 men). At the point where the hepatic artery crosses the portal vein, color Doppler was used to evaluate portal venous blood flow (PVBF) and portal flow velocity (PFV), while laser Doppler velocimetry was used to measure GMBF. Patients with cirrhosis had significantly lower PVBF (379.5 [102.9] mL/min), PFV (5.3 [1.1] cm/sec), and GMBF (3.5 [0.8] volts) than controls. Patients in Child classes B and C had considerably lower PVBF and PFV than patients in class A. Patients with ascites had significantly lower PVBF, PFV and GMBF than those without; values were also lower in patients with PHG than in those without. History of bleeding had no relation with PVBF and PFV. GMBF showed good correlation with PVBF (r=0.58, p<0.001) and with PFV (r=0.48, p<0.01). In cirrhosis of liver, PVBF, PFV and GMBF are significantly lower, and the changes increase with increasing severity of liver disease.

In order to anticipate major varices in patients with cirrhosis, **Joseph T. et al.** conducted a study in 2011 on the Doppler assessment of hepatic venous waves. 51 cirrhosis cases in all were investigated. The group consisted of seven women and forty-four males. The group's average age was 48.1, with a range of 24 to 78. The mean model for end-stage liver disease (MELD) score was 7.781 for the 51 cases, of whom 16 were in Child-Pugh A, 24 in Child-Pugh B, and 11 in Child-Pugh C. Alcohol was the cause of cirrhosis in 26 instances, HBV in 10, HCV in 4, hemochromatosis in 1, and cryptogenic in 10. Four instances had triphasic hepatic venous waveforms, twenty-six had biphasic waveforms, and twenty-one had

monophasic waveforms. Thirty instances had little varices, while twenty-one cases had extensive varices. The size of the varices was not correlated with changes in hepatic venous waveforms. But when it came to identifying big varices, the sensitivity of loss of the typical triphasic pattern was great (95.23%). While the specificity (10%) and positive predictive value (42.6%) were low, the negative predictive value (75%) was likewise high.

Changes in hepatic venous waveforms did not correspond with the severity of liver disease as measured by the Child-Pugh and MELD scores. Other indicators such as platelet count, serum salt, serum creatinine, serum albumin, prothrombin time, and splenic size did not correlate with hepatic venous waveforms. The author came to the conclusion that in patients with cirrhosis, the loss of the triphasic hepatic venous waveform is a highly sensitive indicator of substantial variations.

Relevance of the Study

Liver cirrhosis is a significant global health burden, characterized by high morbidity and mortality rates. Accurate assessment of disease severity is critical for optimal management and prognostication. Non-invasive diagnostic modalities offer a promising alternative, with the potential to improve patient care through more accessible and safer evaluations. However, its role in correlating with established severity markers, such as the Child-Pugh score and the presence of esophageal varices, remains underexplored.

4. Methodology

Study Design
A Cross sectional study.

Study Duration
October 2023 to October 2024.

Study Setting
Department of Radiodiagnosis, Govt. Medical College, Kozhikode.

Study Subjects Inclusion Criteria
- Inpatients and outpatients diagnosed with cirrhosis
- Age group of 20-70 years.

Exclusion Criteria
- Patients with co-existent cardiac or respiratory disease.
- Patients with hepatocellular carcinoma, non-cirrhotic portal fibrosis.
- Past history of endoscopic variceal ligation/sclerotherapy.

Sample Size:
The formula used to determine sample size is given below:
Sample size N = (4 p q) ÷ D2
Where,
P= Prevalence = 13.3 Q= 100 – P = 86.7
D= Absolute error

With a precision of 10% using the above formula, the sample size comes to around 46. Considering drop out as 10%, N = 51.

Methodology
The patient's history, physical examination results, laboratory tests, and Doppler US examination results were all combined into a composite evaluation. One operator obtained all the parameters. The patients gave their written informed consent, and the university approved the study. Due to their incapacity to comply completely during the examination, patients with grade 3 and grade 4 encephalopathy were not included.

A combination of clinical evidence, including jaundice, ascites, muscle atrophy, cutaneous spider angiomas, ecchymosis, palmar erythema, and flapping tremors; laboratory evidence, including elevated prothrombin time and decreased serum albumin; and ultrasound findings, including coarsened echo texture and irregular liver surface, were used to diagnose cirrhosis with portal hypertension. Not every patient received an endoscopy.

Using the Child-Pugh criteria, which were based on the evaluation of a combination of laboratory parameters (serum bilirubin, serum albumin, and prothrombin time international normalized ratio [INR]) and clinical parameters, the patients were split into three groups: Child's A, Child's B, and Child's C cirrhosis groups.

Using an intercostal technique, color Doppler was utilized to assess the peak venous velocity (PVV) and the hepatopetal/ nonhepatopetal (hepatofugal or bidirectional) direction of the flow in the major portal vein while the individuals were in the supine position during suspended inspiration. The angle of insonation was always less than 60 degrees for the velocity measurement. To achieve the proper angle of insonation, the intercostal technique was chosen. The flow path should be as parallel to the US beam direction as feasible in order to provide a sufficient Doppler signal. The intercostal technique is the only way to accomplish this. Getting the right angle of insonation is crucial since portal hypertension frequently results in decreased portal velocity. The means were compared using the unpaired t test and one-way ANOVA; a P value of less than 0.05 was deemed statistically significant.

Plan of Analysis
Data were collected using a pre-designed template and compiled in an Excel spreadsheet. Descriptive analysis was performed on the outcome data. Baseline patient characteristics were presented as frequencies for categorical variables and as means and standard deviations or medians for continuous variables. The statistical analyses were carried out using SPSS ver. 10.0 All the measurements were expressed as mean ± SD. To compare means, Student's *t* test was performed. For multiple values, ANOVA (analysis of variance) with LSD (least square design) was used. $P < 0.05$ and $r > 0.6$ were considered statistically significant.

Subject Confidentiality
Data was collected on paper. All the patient specific data was kept in strict confidence. Patient identifiable data (name, contact, address, etc.) were not be presented in journal or any public forum. Informed consent process was initiated prior to the individual agreeing to participate in the study and

continuing throughout the individual's study participation. It is in a language understandable by each member of the study population.

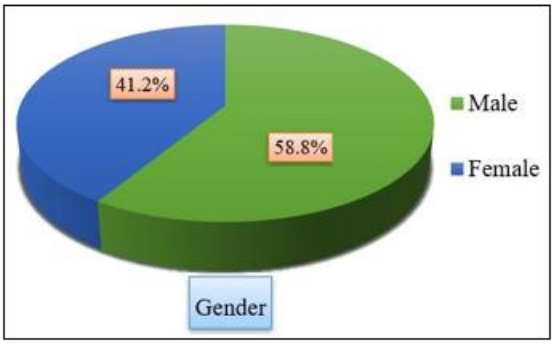
Ethical Considerations

The protocol of the present study was submitted to the Ethics Committee of the hospital for review. After getting their approval the study was initiated in the institution. Also prior to enrolling any patient in the study, a voluntary written informed consent was obtained for participation.

5. Results

Table 2: Distribution of patients on the basis of their gender.

Gender	Frequency	Percentage
Male	30	58.8
Female	21	41.2

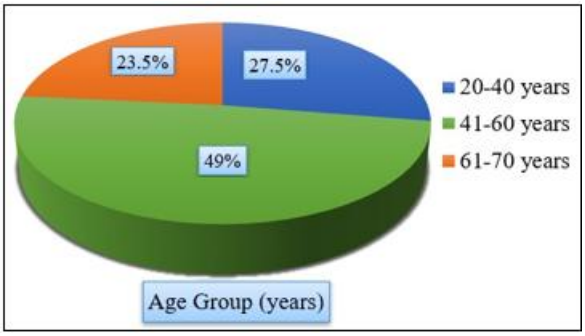


Graph 1: Distribution graph of patients on the basis of their gender

A total of 51 patients were enrolled in the study, comprising 58.8% males (n=30) and 41.2% females (n=21), resulting in a male-to-female ratio of 1.4:1.

Table 3: Distribution of patients on the basis of their age group (years).

Age Group	Frequency	Percentage
20-40 years	14	27.5
41-60 years	25	49.0
61-70 years	12	23.5

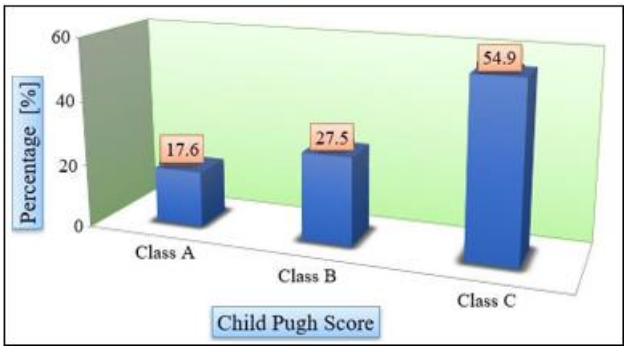


Graph 2: Distribution graph of patients on the basis of their age group (years).

The study population was categorized into three age groups: 20–40 years, 41–60 years, and 61–70 years. Among the 51 patients, 49.0% (n = 25) were aged 41–60 years, 27.5% (n = 14) were aged 20–40 years, and 23.5% (n = 12) were aged 61–70 years.

Table 4: Distribution of patients on the basis of their Child-Pugh score

Child Pugh Score	Frequency	Percentage
Class A	9	17.6
Class B	14	27.5
Class C	28	54.9



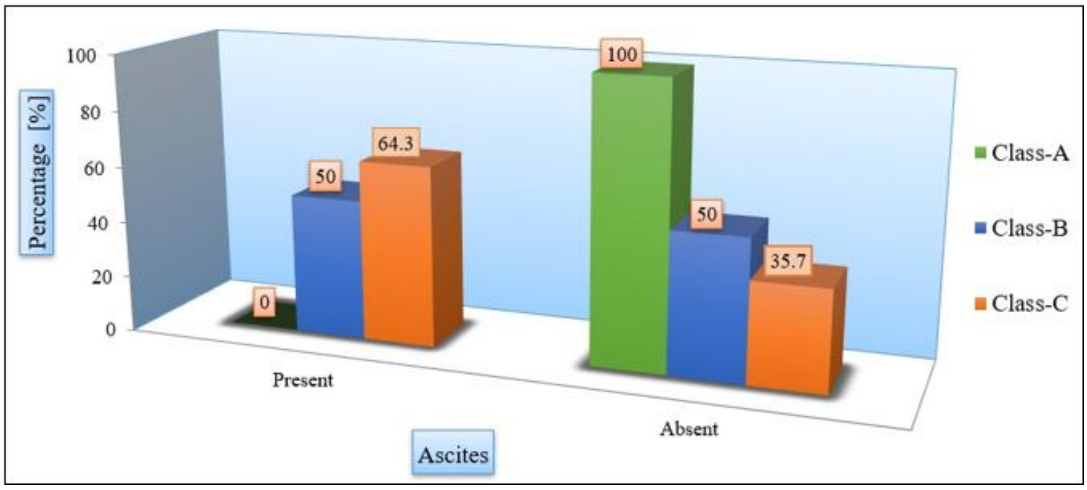
Graph 3: Distribution graph of patients on the basis of their Child-Pugh score.

Regarding the Child-Pugh classification, the majority of patients were categorized as Class C, accounting for 54.9% (n = 28), followed by 27.5% (n = 14) in Class B and 17.6% (n = 9) in Class A.

Patients were stratified into three categories according to the Child-Pugh classification: Class A (n = 9), Class B (n = 14), and Class C (n = 28).

Table 5: Distribution of patients based on ascites with respect to Child-Pugh class.

Ascites	Class-A (N = 9)		Class-B (N = 14)		Class-C (N = 28)		P-value
	N	%	N	%	N	%	
Present	0	0.0	7	50.0	18	64.3	0.0036
Absent	9	100.0	7	50.0	10	35.7	

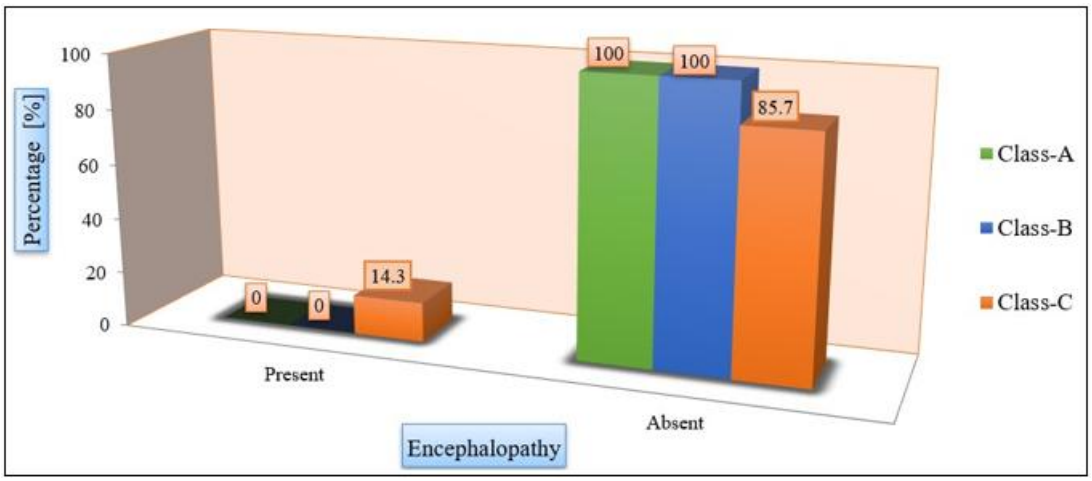


Graph 4: Distribution graph of patients based on ascites with respect to Child-Pugh class.

Out of 51 patients, ascites was present in 49.0% (n = 25) and absent in 51.0% (n = 26). Among the 28 patients categorized as Child-Pugh Class C, 64.3% (n = 18) had ascites, compared to 50.0% (n = 7) among the 14 patients classified as Class B. Notably, none of the 9 patients in Class A had ascites. A statistically significant association was observed between Child-Pugh classification and the presence of ascites (P = 0.0036).

Table 6: Distribution of patients based on Encephalopathy with respect to Child-Pugh class

Encephalopathy	Class-A (N = 9)		Class-B (N = 14)		Class-C (N = 28)		P-value
	N	%	N	%	N	%	
Present	0	0.0	0	0.0	4	14.3	0.1682
Absent	9	100.0	14	100.0	24	85.7	

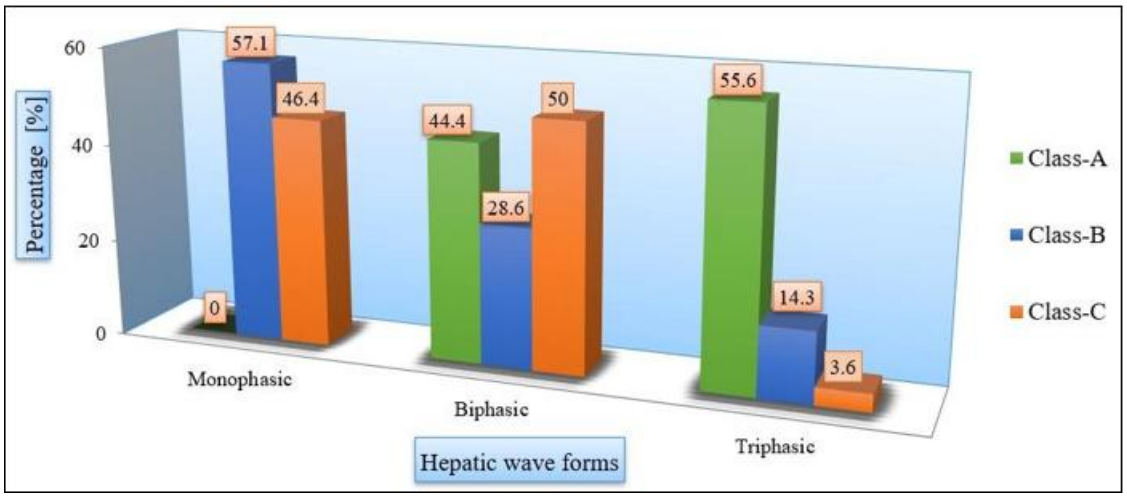


Graph 5: Distribution graph of patients based on Encephalopathy with respect to Child-Pugh class.

Encephalopathy was present in 4 patients (7.8%) within the study cohort, while absent in the remaining 47 patients (92.2%). Among the 28 patients categorized as Child-Pugh Class C, encephalopathy was observed in 14.3% (n = 4), whereas no cases of encephalopathy were reported in patients classified as Child-Pugh Class A or B. Despite this distribution, the association between Child-Pugh classification and the occurrence of encephalopathy was not statistically significant (P = 0.1682).

Table 7: Distribution of patients based on hepatic wave forms with respect to Child- Pugh class

Hepatic wave forms	Class-A (N = 9)		Class-B (N = 14)		Class-C (N = 28)		P-value
	N	%	N	%	N	%	
Monophasic	0	0.0	8	57.1	13	46.4	0.0015
Biphasic	4	44.4	4	28.6	14	50.0	
Triphasic	5	55.6	2	14.3	1	3.6	

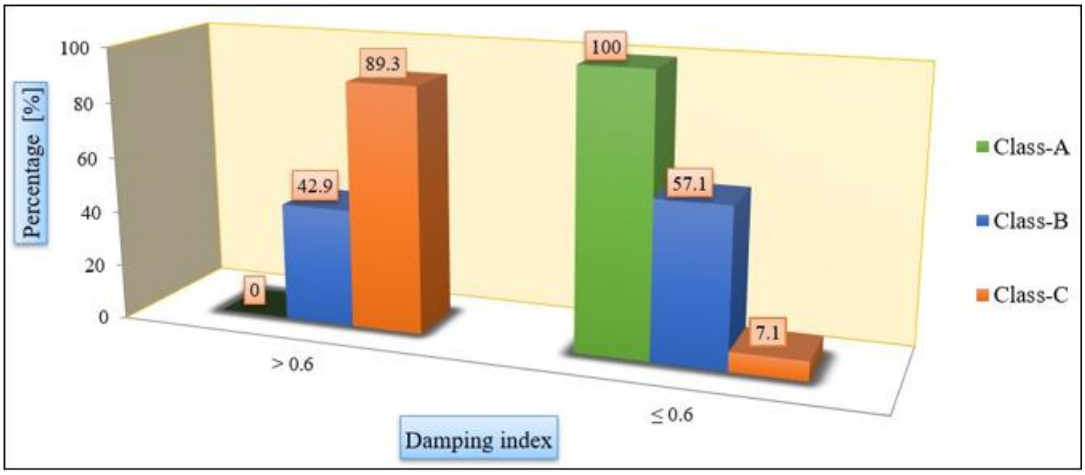


Graph 6: Distribution graph of patients based on hepatic wave forms with respect to Child-Pugh class

In the assessment of hepatic waveforms, the majority of patients had biphasic waveforms, accounting for 43.1% (n = 22), followed by monophasic waveforms in 41.2% (n = 21), and triphasic waveforms in 15.7% (n = 8). Stratified by class, among the 9 patients classified as Class A, 55.6% (n = 5) had triphasic waveforms, 44.4% (n = 4) had biphasic waveforms, and none presented with monophasic waveforms. In Class B patients (n = 14), monophasic waveforms were observed in 57.1% (n = 8), biphasic in 28.6% (n = 4), and triphasic in 14.3% (n = 2). Among the 28 patients classified as Class C, 50.0% (n = 14) had biphasic waveforms, 46.4% (n = 13) monophasic, and only 3.6% (n = 1) triphasic. A statistically significant association was observed between Child-Pugh classification and the hepatic waveform patterns (P = 0.0015).

Table 8: Distribution of patients based on damping index with respect to Child-Pugh class.

Damping index	Class-A (N = 9)		Class-B (N = 14)		Class-C (N = 28)		P-value
	N	%	N	%	N	%	
> 0.6	0	0.0	6	42.9	25	89.3	< 0.001
≤ 0.6	9	100.0	8	57.1	2	7.1	

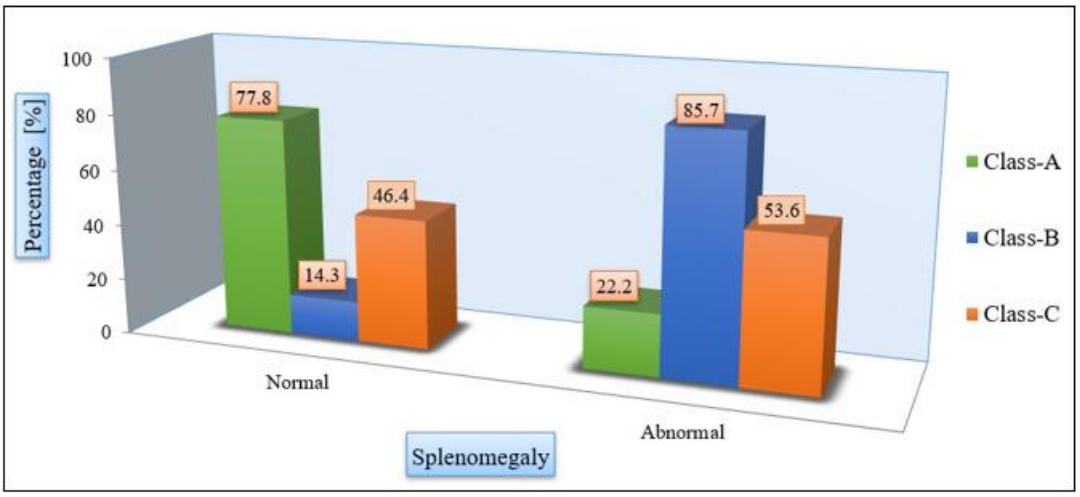


Graph 7: Distribution graph of patients based on damping index with respect to Child- Pugh class

The damping index was > 0.6 in 60.8% of patients (n=31) and ≤0.6 in 39.2% (n=20). Among patients classified as Class A (n=9), all (100.0%, n=9) had a damping index ≤0.6. In Class B patients (n=12), 57.1% (n=8) exhibited a damping index ≤0.6, while 42.9% (n=6) had a damping index >0.6. In contrast, the majority of Class C patients (n=28) had a damping index >0.6 (89.3%, n=25), with only 7.1% (n=2) having a damping index ≤0.6. A statistically significant association was observed between Child- Pugh classification and the damping index (P < 0.001).

Table 9: Distribution of patients based on splenomegaly with respect to Child-Pugh class

Splenomegaly	Class-A (N = 9)		Class-B (N = 14)		Class-C (N = 28)		P-value
	N	%	N	%	N	%	
Normal	7	77.8	2	14.3	13	46.4	0.0097
Abnormal	2	22.2	12	85.7	15	53.6	

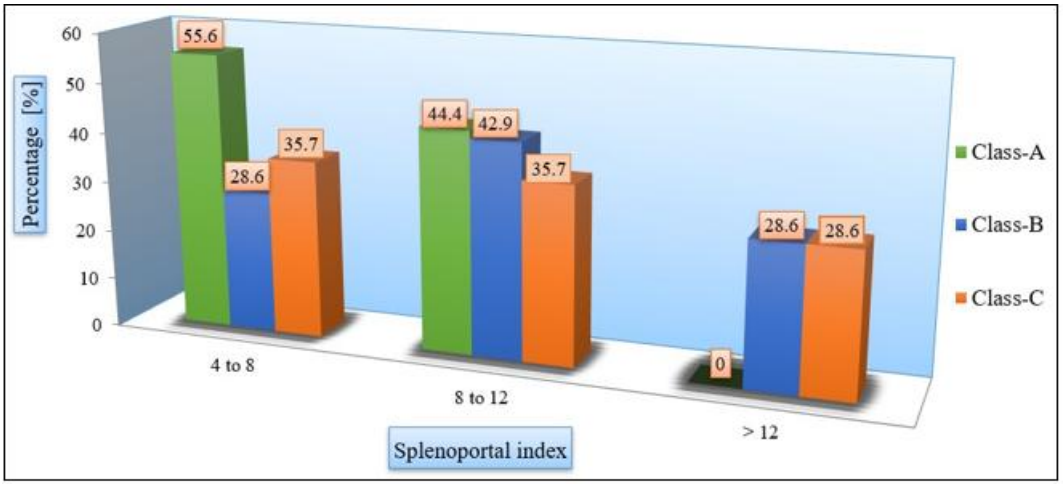


Graph 8: Distribution graph of patients based on splenomegaly with respect to Child- Pugh class.

In the study cohort, 56.9% (n = 29) of patients had abnormal splenomegaly, while 43.1% (n = 22) had normal splenomegaly. Stratification by Child-Pugh classification revealed that among Class A patients (n = 9), 22.2% (n = 2) had abnormal splenomegaly. In contrast, 85.7% (n = 12) of Class B patients (n = 14) and 53.6% (n = 15) of Class C patients (n = 28) had abnormal splenomegaly. A statistically significant correlation was observed between the Child-Pugh classification and the presence of abnormal splenomegaly (P = 0.0097).

Table 10: Distribution of patients based on splenoportal index with respect to Child- Pugh class.

Oesophageal varices	Class A (N=9)		Class B (N= 14)		Class C (N= 28)		P-value
	N	%	N	%	N	%	
4 to 8	5	55.6	4	28.6	10	35.7	0.4226
8 to 12	4	44.4	6	42.9	10	35.7	
> 12	0	0.0	4	28.6	8	28.6	

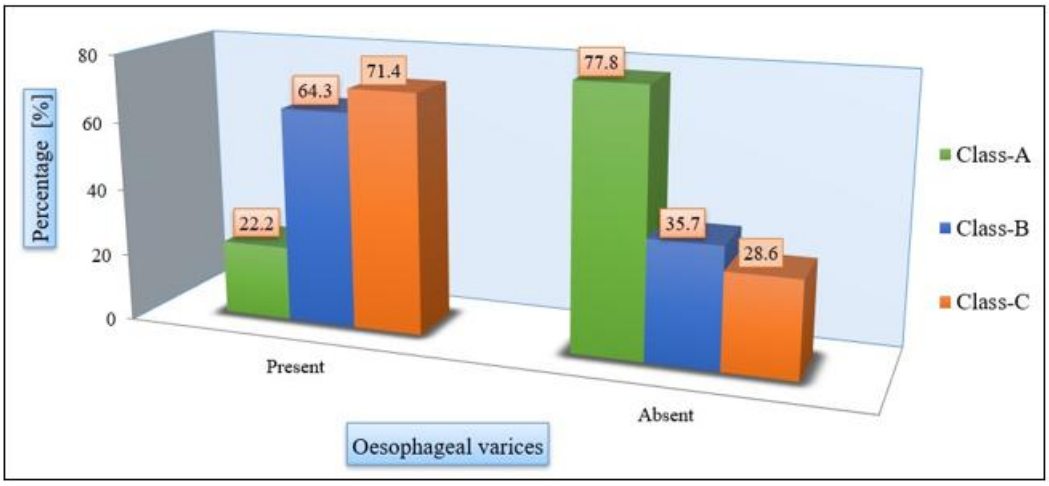


Graph 9: Distribution graph of patients based on splenoportal index with respect to Child-Pugh class.

The splenoportal index was categorized into three groups: 4–8 cm/sec (37.3%; n = 19), 8–12 cm/sec (39.2%; n = 20), and >12 cm/sec (23.5%; n = 12). Stratification based on the Child-Pugh classification observed that among the 9 Class A patients, 55.6% (n = 5) had a splenoportal index of 4–8 cm/sec, 44.4% (n = 4) had values between 8–12 cm/sec, and none had values exceeding 12 cm/sec. Among the 14 Class B patients, 28.6% (n = 4) were in the 4–8 cm/sec category, 42.9% (n = 6) were between 8–12 cm/sec, and 28.6% (n = 4) were >12 cm/sec. Similarly, of the 28 Class C patients, 35.7% (n = 10) were in the 4–8 cm/sec category, 35.7% (n = 10) were between 8–12 cm/sec, and 28.6% (n = 8) had a splenoportal index >12 cm/sec. However, no statistically significant association was observed between the Child-Pugh classification and the splenoportal index (P = 0.4226).

Table 11: Distribution of patients based on Esophageal varices with respect to Child- Pugh class.

Oesophageal varices	Class A (N=9)		Class B (N= 14)		Class C (N= 28)		P-value
	N	%	N	%	N	%	
Present	2	22.2	9	64.3	20	71.4	0.0299
Absent	7	77.8	5	35.7	8	28.6	

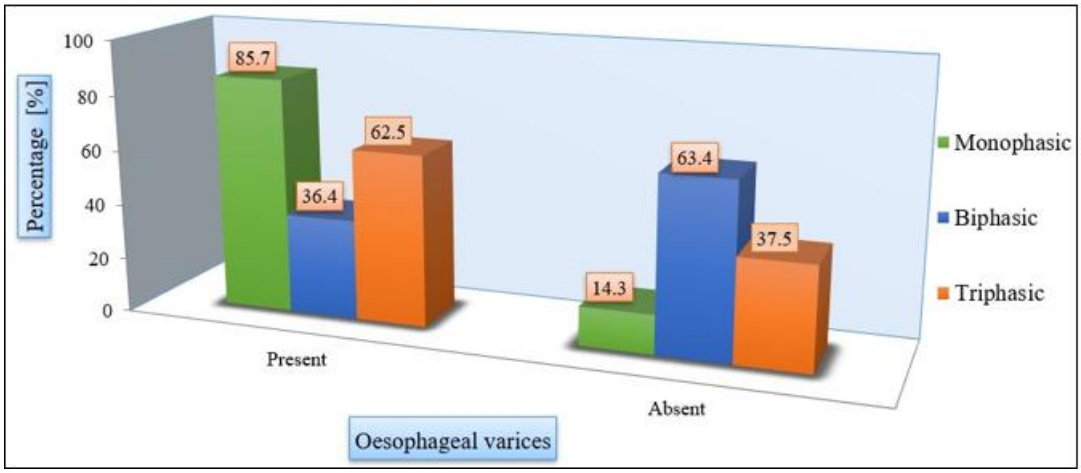


Graph 10: Distribution graph of patients based on Esophageal varices with respect to Child-Pugh class

Of 51 patients included in the study, esophageal varices were observed in 60.8% (n=31). Stratification according to the Child-Pugh classification demonstrated that esophageal varices were present in 22.2% (n=2) of Class A patients (n=9), 64.3% (n=9) of Class B patients (n=14), and 71.4% (n=20) of Class C patients (n=28). A statistically significant association was observed between the Child-Pugh classification and the esophageal varices (P = 0.0299).

Table 12: Distribution of patients based on Esophageal varices with respect to hepatic wave forms.

Oesophageal varices	Monophasic (N=21)		Biphasic (N=22)		Triphasic (N=8)		P-value
	N	%	N	%	N	%	
Present	18	85.7	8	36.4	5	62.5	0.0041
Absent	3	14.3	14	63.6	3	37.5	



Graph 11: Distribution graph of patients based on Esophageal varices with respect to hepatic wave forms

Of 51 patients included in the study, esophageal varices were observed in 60.8% (n=31). Stratification according to the hepatic wave forms demonstrated that esophageal varices were observed in 85.7% (n=18) of monophasic patients (n=21), 36.4% (n=8) of biphasic (n=22) and 62.5% (n=5) of triphasic (n=8). A statistically significant association was observed between the hepatic wave forms and the esophageal varices (P = 0.0041).

6. Discussion

Cirrhosis, a progressive liver disease characterized by fibrosis and architectural distortion, leads to significant alterations in hepatic hemodynamics. Portal hypertension, a major complication of cirrhosis, arises from increased resistance to blood flow within the portal venous system. This elevated pressure can result in life- threatening complications, including esophageal varices, ascites, and hepatic encephalopathy. The Child-Pugh score, a widely used staging system for cirrhosis, primarily assesses liver function and prognosis. However, it does not directly reflect the hemodynamic changes associated with portal hypertension. This limitation underscores the need for additional tools to assess the severity of cirrhosis and predict complications. Hepatic venous Doppler ultrasonography is a non-invasive technique that can provide valuable insights into hepatic blood flow dynamics. By analyzing the waveform patterns of hepatic venous blood flow, Doppler ultrasound can help assess the degree of portal hypertension and liver dysfunction. This study aims to investigate the correlation between hepatic venous Doppler parameters and the severity of cirrhosis, as assessed by the Child-Pugh score. Additionally, we aim to explore the association between Doppler parameters and the presence of esophageal varices. By understanding these relationships, we may identify novel non-invasive biomarkers for risk stratification and clinical decision-making in patients with cirrhosis.

This study observed a predominance of males (58.8%) among patients with cirrhosis, compared to females (41.2%). These findings align with prior epidemiological data, which consistently report higher cirrhosis prevalence in males across diverse populations. This gender disparity likely reflects differences in risk factor profiles, behavioral exposures, and potentially biological determinants of disease progression. One significant factor contributing to the male predominance is the higher prevalence of chronic alcohol consumption in men. Alcohol-related liver disease remains a leading cause of cirrhosis worldwide, and men are more likely than women to engage in heavy and prolonged alcohol use. Additionally, delayed healthcare engagement in men may contribute to later-stage diagnoses and a higher prevalence of advanced liver disease. In contrast, non-alcoholic fatty liver disease (NAFLD), autoimmune hepatitis, and certain metabolic conditions are more frequently associated with cirrhosis in women. Notably, sex hormones, particularly estrogen, may exert protective effects on liver fibrosis in premenopausal women. This antifibrotic effect may attenuate the progression of chronic liver disease in women, partially accounting for the observed gender differences in cirrhosis prevalence. These results align with **Mittal P et al.** who reported a similar trend, with 66% of cirrhosis cases occurring in males compared to 34% in females. **Abdelmonem EA et al.** also observed a male dominance of 66.7%, with females accounting for only 33.3% of cases. **Kumar S et al.** reported an even higher male prevalence, with 88% of cases in males and only 12% in females.

The age distribution of patients with cirrhosis in this study demonstrated that the highest prevalence (49.0%) was in the 41–60 years age group, followed by 27.5% in the 20–40 years group and 23.5% in the 61–70 years group. These findings suggest that cirrhosis predominantly affects individuals in middle adulthood, a trend commonly reported in the literature. The peak prevalence in the 41–60 years age group likely reflects the prolonged natural history of chronic liver disease, where etiologies such as alcohol-related liver disease, chronic viral hepatitis (HBV and HCV), and NAFLD progressively lead to cirrhosis over decades. This age group represents the critical point were cumulative hepatic injury manifests as clinically significant liver dysfunction or portal hypertension. **Antil et al.** also reported that cirrhosis was commonly observed in the age range of 36-45 years (43.3%), followed by the 26–35 years group (36.7%), and the lowest prevalence in the 46–56 years group (20.0%). Similarly, **Kumar S et al.** reported that the incidence of cirrhosis was maximum in the age group 31-50 years (60%). **Mittal P et al.** also reported that maximum of the patients was in the 31-40 years age group.

The Child-Pugh score is a widely used clinical system for assessing the severity of cirrhosis and predicting patient prognosis. It classifies patients into three groups— Class A, Class B, and Class C—based on the degree of liver dysfunction. Class A indicates well-compensated liver function, with the best prognosis, while Class C represents severe liver impairment, associated with a higher risk of complications and mortality. The Child-Pugh score plays a crucial role in clinical decision-making, including the management of cirrhosis-related complications and the prioritization of patients for liver transplantation. The distribution of Child-Pugh scores in this study reveals a predominance of patients classified as Child-Pugh C (54.9%), followed by Class B (27.5%) and Class A (17.6%). This distribution suggests that a significant proportion of patients in this cohort present with advanced liver dysfunction, highlighting the severity of cirrhosis in the studied population. The high percentage of patients in Class C reflects the advanced stage of liver disease in many individuals, which is often associated with complications such as portal hypertension, ascites, hepatic encephalopathy, and variceal bleeding. The predominance of Class C patients may be due to late-stage diagnosis, a common occurrence in cirrhosis when clinical manifestations of liver failure become evident. These results align with **Mittal P et al.** reported that majority (42%) of cases belong to class C, followed by class B (32%) and class C (26%). **Yasmin T et al.** reported that 25 (31%) patients presented in Child-Pugh Class A, 31(39%) with Class B and 24(30%) patients had Class C. In contrast, **Bhutto AR et al.** observed a higher proportion of patients in Child-Pugh Class A (49.2%) compared to Class B (35.4%) and Class C (15.4%). **Afif M et al.** also observed a higher proportion of patients in Child-Pugh Class A (51.8%) when compared to Class B (33.9%) and Class C (14.3%).

Hepatic Encephalopathy (HE) is a neuropsychiatric syndrome resulting from severe liver dysfunction, most commonly observed in patients with cirrhosis. A significant proportion of Class C patients (14.3%) experiencing HE, while no patients in Class A or Class B demonstrated signs of encephalopathy. This aligns with the understanding that HE is more commonly seen in patients with advanced cirrhosis, particularly those classified as Child-Pugh Class C, who exhibit severe liver dysfunction and a diminished ability to clear toxins such as ammonia from the bloodstream. The absence of encephalopathy in Class A and Class B patients is consistent with the fact that these individuals typically have better-preserved liver function and are less likely to accumulate toxic metabolites. This suggests that, at these stages of liver disease, the liver still maintains its detoxifying capacity, preventing the development of HE. **Antil et al.** reported that three patients with cirrhosis had hepatic encephalopathy and all were in class C. In contrast, **Nazemi S et al.** reported that no significant correlation between hepatic venous waveforms and the presence of encephalopathy (P=0.817).

Hepatic venous Doppler waveforms, including monophasic, biphasic, and triphasic patterns, reflect changes in liver function and the presence of portal hypertension. A monophasic waveform, marked by a single forward flow phase without distinct systolic or diastolic components, is often associated with advanced portal hypertension and severe cirrhosis, indicating increased resistance to hepatic venous outflow due to fibrosis or structural liver changes. A biphasic waveform, characterized by two phases with forward flow during systole followed by a reversal or reduced flow during diastole, is commonly observed in moderate portal hypertension or intermediate stages of liver disease. This pattern suggests moderate resistance to hepatic venous outflow without complete decompensation. In contrast, a triphasic waveform, comprising three distinct phases—systolic (S-wave), diastolic (D-wave), and

retrograde atrial flow (A-wave)—is the normal pattern found in healthy livers, reflecting minimal resistance to hepatic venous return. The transition from a triphasic to biphasic or monophasic pattern corresponds to increasing portal pressure and liver dysfunction, offering clinically relevant information for evaluating the progression of liver disease and associated hemodynamic changes. In Class-A, 55.6% of patients exhibited a triphasic waveform, which is typical of normal liver function, indicating that there was minimal resistance to hepatic venous outflow. This pattern is consistent with the well- preserved liver function seen in this group. In Class-B, 57.1% of patients had a monophasic waveform, which suggests increased resistance to hepatic venous flow, often associated with moderate portal hypertension. The biphasic waveform was observed in 28.6% of Class-B patients, reflecting a transitional state where hepatic venous resistance is elevated, but not to the extent seen in advanced cirrhosis. These findings align with the pathophysiological changes observed in Class-B cirrhosis, where liver function is compromised, and portal pressure is starting to rise but has not reached the levels seen in decompensated cirrhosis. In Class-C, 46.4% of patients displayed a monophasic waveform, and 50% had a biphasic waveform, while only 3.6% maintained a triphasic waveform. The dominance of monophasic and biphasic patterns in this group reflects significant hepatic dysfunction and advanced portal hypertension. The reduced incidence of the triphasic waveform in Class-C patients is consistent with the progressive worsening of hepatic venous outflow resistance and the liver's declining ability to manage blood flow. The absence of a triphasic pattern in most Class-C patients further supports the severity of cirrhosis, where decompensation and high portal pressure are prevalent. There was a statistically significant difference in Child Pugh score and hepatic wave forms (P=0.0015). As liver disease progresses from Class-A to Class-C, there is a clear shift from a normal triphasic pattern to more abnormal waveforms, reflecting increasing resistance and liver dysfunction. These findings are consistent with those of **Antil et al.** who reported that as the Child-Pugh score increased, the normal triphasic waveform progressively converted to biphasic and ultimately to monophasic. This demonstrated a significant correlation between hepatic venous waveforms and Child-Pugh classification ($p < 0.05$). Similarly, **Bhutto et al.** in their study involving 65 patients, concluded that hepatic venous waveforms showed a significant association with hepatic dysfunction ($p < 0.012$). **Yasmin T et al.** also observed that 88% of patients in Child-Pugh Class C, 23% in Class B, and 8% in Class A exhibited monophasic hepatic venous waveforms. Their study further emphasized that hepatic venous waveform progressively transitioned from triphasic to biphasic and then to monophasic with advancing grades of cirrhosis, demonstrating a significant correlation with hepatic dysfunction ($p < 0.022$). The exact mechanism behind the changing Doppler waveform patterns associated with portal hypertension remains unclear. Some researchers suggest that the change is due to parenchymal fibrosis and the infiltration of fat around the portal vein wall, which reduces the compliance of the vein. Others believe that interhepatic shunting, a pathogenic mechanism in cirrhosis, is responsible for altering the hepatic venous waveform pattern. **Bolondi et al.** reported that the underlying mechanism of these waveform changes may be related to liver fibrosis, which progressively reduces phasic oscillation in hepatic veins. However, studies by **Sudhamshu et al.** and **Joseph et al.** could not establish a correlation between the severity of liver disease and hepatic venous waveforms.

The damping index (DI) is a hepatic venous Doppler parameter used to evaluate the severity of liver dysfunction and portal hypertension. It is calculated as the ratio of the minimum to the maximum velocity of the hepatic venous waveform during a cardiac cycle. In healthy individuals, the DI is typically low due to the prominent phasic variations in the triphasic waveform. However, in conditions like cirrhosis and portal hypertension, where hepatic venous outflow is impaired, the waveform becomes attenuated, and the DI increases. A higher DI indicates greater hepatic venous resistance and is associated with advanced liver dysfunction, making it a useful non-invasive marker for assessing disease severity. The analysis of the damping index (DI) across different Child-Pugh classes revealed a significant association between DI values and the severity of cirrhosis ($P < 0.001$). In Child-Pugh Class A, all patients (100%) had a DI ≤ 0.6, consistent with the presence of a normal or minimally altered hepatic venous waveform, indicating preserved hepatic venous outflow and minimal portal hypertension. In Class B, 42.9% of patients exhibited a DI > 0.6, suggesting moderate hepatic venous resistance and early hemodynamic changes associated with worsening liver function. In Class C, the majority of patients (89.3%) had a DI > 0.6, reflecting significant hepatic venous outflow resistance and advanced portal hypertension, characteristic of severe liver dysfunction and cirrhosis. The progressive increase in the proportion of patients with a DI > 0.6 from Class A to Class C demonstrates the utility of the damping index as a non-invasive marker for evaluating the severity of cirrhosis. These results align with **Antil et al.** who reported that patients with DI > 0.6 were predominantly in Class C, reflecting significant hepatic venous resistance and advanced liver dysfunction. In contrast, those with DI ≤ 0.6 were more commonly in Classes A and B, indicating less severe disease. **Subodh D et al.** reported that fourteen patients (70%) had value of Damping index more than >0.6 where majority of patients (12) belonged to class C and 2 in class B. There was a positive correlation between Child Pugh's total score and Damping index.

Splenomegaly refers to an abnormal enlargement of the spleen, commonly associated with liver diseases such as cirrhosis. It results from increased portal venous pressure, leading to congestion in the splenic vein, a condition known as congestive splenomegaly. Splenomegaly is a hallmark of portal hypertension and may contribute to hypersplenism, characterized by anemia, leukopenia, and thrombocytopenia due to excessive sequestration of blood cells. Its presence is a key clinical indicator of advanced liver disease and often correlates with the severity of portal hypertension. The analysis of splenomegaly across Child-Pugh classes revealed a significant association between spleen enlargement and the severity of liver dysfunction ($P = 0.0097$). In Child- Pugh Class A, the majority of patients (77.8%) had normal spleen size, reflecting preserved liver function and minimal portal hypertension. However, in Class B, 85.7% of patients exhibited splenomegaly, indicating the presence of

significant portal hypertension as liver function deteriorates. In Class C, 53.6% of patients had splenomegaly, consistent with advanced portal hypertension and severe cirrhosis. The progression from normal spleen size in Class A to abnormal enlargement in Classes B and C highlights the role of splenomegaly as an indicator of worsening portal hypertension and liver dysfunction.

The Splenoportal Index (SPI) is a non-invasive measurement used to assess the degree of portal hypertension, commonly in patients with liver cirrhosis. It is calculated by dividing the splenic size (usually the longest diameter of the spleen) by the portal vein diameter. An elevated SPI indicates increased portal pressure, which is a hallmark of portal hypertension. An SPI value between 4 to 8 typically indicates mild portal hypertension, often observed in early stages of liver disease or less advanced cirrhosis.

Values between 8 to 12 suggest moderate portal hypertension, commonly seen in patients with more advanced cirrhosis. When the SPI exceeds 12, it is indicative of significant portal hypertension, which is often associated with severe cirrhosis and complications such as varices and splenomegaly. The index is useful for predicting the presence of complications such as varices or splenomegaly and can help in monitoring disease progression. It provides a valuable alternative to more invasive procedures, like liver biopsy, in evaluating the severity of liver disease and portal hypertension. In Class A patients, 55.6% had an SPI ranging from 4 to 8, suggesting a relatively mild degree of portal hypertension, which corresponds with the well-preserved liver function in this group. In Class B, 42.9% of patients also had an SPI in the 8 to 12 range, reflecting moderate portal hypertension. Interestingly, the distribution of SPI values in Class B patients was fairly balanced, with a notable 28.6% having an SPI > 12, which may indicate the onset of more significant portal hypertension as liver function starts to decline. In Class C patients, the majority (35.7%) had an SPI in the 4 to 8 range, while 35.7% also had values in the 8 to 12 range, and 28.6% showed an SPI > 12. This distribution suggests that as cirrhosis advances, there is increasing variability in the degree of portal hypertension, likely reflecting the complex nature of severe liver dysfunction and the presence of complications such as variceal bleeding or splenomegaly. The lack of a statistically significant difference in the SPI values across the Child-Pugh classes ($p = 0.4226$) indicates that while there is an increasing trend in SPI values with worsening cirrhosis, the index alone may not fully capture the degree of portal hypertension or correlate directly with liver dysfunction as classified by the Child-Pugh score. **Nazemi S et al.** reported that splenoportal indices across the different categorizes of liver disease severity according to the Child grading system. **Antil et al.** reported that there was weak correlation between the SPI value and Child Pugh's score ($r = 0.269$; $p = 0.15$).

Esophageal varices are dilated veins in the lower part of the esophagus, which develop as a result of portal hypertension, commonly caused by liver cirrhosis. In cirrhosis, the liver becomes scarred and damaged, obstructing blood flow, leading to increased pressure in the portal vein. This pressure is transmitted to the veins in the esophagus, causing them to enlarge and become fragile, increasing the risk of rupture and life-threatening bleeding. The severity of esophageal varices can range from small, asymptomatic veins to large, bleeding varices. The presence and size of esophageal varices are important indicators of cirrhosis progression and are closely monitored to manage the risk of bleeding, which can be life-threatening. In the present study, the presence of oesophageal varices was found to increase with the severity of cirrhosis, as indicated by the Child-Pugh classification. In Class-A patients, only 22.2% had oesophageal varices, reflecting the mild liver dysfunction and lower incidence of portal hypertension in this group. In contrast, Class-B patients had a significantly higher prevalence of varices, with 64.3% affected, suggesting that moderate liver dysfunction and portal hypertension are more common in this stage. Among Class-C patients, 71.4% exhibited oesophageal varices, consistent with the advanced portal hypertension and decompensated liver disease seen in this class. Also, a statistically significant ($P=0.0299$) association was observed between the presence of oesophageal varices and the severity of liver disease, with increasing frequency of varices as the Child-Pugh score rises. These results are consistent with **Tiwari PS et al.** who reported that among patients with Child class A, 48.3% had varices. Similarly, 94.2% and 100% of Child class B & C had varices respectively. There was a statistically significant relation between oesophageal varices and Child-Pugh score ($P<0.05$). **Bhattarai et al.** also demonstrated similar findings and reported that the prevalence of varices in CPC-A, CPC-B, and CPC-C was 16.6%, 19.1%, and 79.9%, respectively. This variation in the detection of varices across different CPC grades was statistically significant ($P < 0.05$). Similarly, the study by **Shrestha et al.** also highlighted a correlation between higher grades of varices and elevated Child-Pugh scores. In contrast, **Antil et al.** and **Bhutto et al.** reported a statistically insignificant relationship between presence of oesophageal varices and Child-Pugh score.

The relationship between oesophageal varices and hepatic venous waveform patterns reveals significant findings. Among patients with a monophasic waveform, 85.7% had oesophageal varices, indicating a strong association between this waveform pattern and the presence of varices. This is consistent with the advanced portal hypertension and liver dysfunction typically seen in patients with monophasic waveforms. In contrast, only 36.4% of biphasic waveform patients had oesophageal varices, suggesting a weaker but still notable correlation, likely reflecting an intermediate stage of liver disease and portal hypertension. Interestingly, 62.5% of triphasic waveform patients had varices, which is higher than expected, possibly indicating that even in early stages of liver disease, oesophageal varices can develop, though the overall liver function remains less impaired. The p-value of 0.0041 indicates a statistically significant association between hepatic venous waveforms and the presence of oesophageal varices, highlighting the potential utility of waveform analysis as a predictor of variceal development in cirrhosis. These findings suggest that as hepatic function deteriorates and portal hypertension progresses, changes in the hepatic venous waveform, particularly towards monophasic, may serve as an early marker for oesophageal varices in cirrhotic patients. **Abdallah et al.** reported that, almost all patients with esophageal varices had monophasic hepatic wave (96.2%),

while patients without esophageal varices had the three forms of waves with the highest percentage for triphasic waveform (40.9%). According to a study by Joseph et al., the presence of the triphasic pattern in a patient with cirrhosis was strongly predictive of the absence of major esophageal varices, while the absence of the pattern in the hepatic venous tracing was significantly predictive of their occurrence. In contrast, **Antil et al.** and **Bhutto et al.** reported that there was no statistically significant relationship hepatic vein waveform and oesophageal varices.

7. Limitations

This study, while highlighting the utility of hepatic venous Doppler ultrasonography in assessing cirrhosis severity and related complications, has certain limitations. First, the sample size was relatively small, which may limit the generalizability of the findings to broader populations. A larger cohort could provide more robust validation of the observed correlations. Second, the study was cross-sectional, capturing data at a single time point; a longitudinal design could better elucidate the progression of cirrhosis and Doppler parameter changes over time. Additionally, while Doppler ultrasonography is a non-invasive and widely available tool, its accuracy is operator-dependent and may vary with the skill and experience. Finally, variability in the measurement of Doppler parameters, particularly in patients with severe ascites or other anatomical distortions, which may affect the quality of Doppler waveform analysis. Future research addressing these limitations could enhance the applicability and clinical relevance of Doppler ultrasonography in cirrhosis management.

8. Conclusion

This study highlights the value of hepatic venous Doppler ultrasonography and associated parameters in assessing the severity of liver cirrhosis, its hemodynamic consequences, and complications such as esophageal varices. The demographic analysis revealed a male predominance among patients with cirrhosis, with the highest prevalence observed in the age group of 41–60 years The findings demonstrated a strong association between hepatic venous Doppler ultrasonography parameters and the severity of cirrhosis, as assessed by the Child-Pugh classification. A progressive transition from triphasic to monophasic waveforms was observed with worsening cirrhosis severity, reflecting increasing portal hypertension and hepatic venous outflow resistance. This transition demonstrates a statistically significant association with the Child-Pugh classification, affirming the clinical utility of Doppler ultrasonography in staging cirrhosis. The DI was significantly elevated in advanced stages of cirrhosis, with a strong correlation between higher DI values and Child-Pugh Class C, indicating its potential as a non-invasive marker for advanced liver dysfunction and portal hypertension. Although the splenoportal index (SPI) showed a trend toward elevation with worsening cirrhosis, its correlation with cirrhosis severity was not statistically significant, limiting its clinical utility. The presence of esophageal varices correlated significantly with both Child-Pugh scores and Doppler waveform patterns, emphasizing the role of portal hypertension in variceal development. Hepatic venous Doppler parameters, particularly waveform analysis and the DI, serve as valuable non-invasive tools for assessing cirrhosis severity and predicting complications like varices. These findings support their integration into routine clinical evaluation, complementing established methods like the Child-Pugh classification. Future research should focus on refining Doppler-based biomarkers and exploring their role in guiding management decisions, including the timing of interventions for variceal bleeding and liver transplantation.

9. Summary

Liver cirrhosis is characterized by fibrosis and regenerative nodules due to hepatocyte damage, leading to impaired liver function and complications like portal hypertension, ascites, varices, hepatic encephalopathy, and hepatorenal syndrome. Portal hypertension often manifests as gastropathy and varices. The Modified Child-Pugh Scoring System is commonly used to assess cirrhosis severity, while portal hypertension can be evaluated with both invasive and non-invasive methods. Doppler ultrasound, a non- invasive technique, assesses liver hemodynamics, providing insights into disease severity and the risk of esophageal varices. This study investigates the relationship between hepatic venous Doppler parameters, Child-Pugh scores, and esophageal varices.

A cross-sectional study was conducted with 51 cirrhosis patients. A comprehensive evaluation was performed, incorporating patient history, physical examination findings, laboratory investigations, and Doppler ultrasound results. Patients were stratified into three groups—Child-Pugh class A, B, and C—based on the Child-Pugh scoring system, which includes laboratory parameters (serum bilirubin, serum albumin, and international normalized ratio [INR] of prothrombin time) and clinical findings. Statistical analysis used unpaired t-tests and ANOVA, with significance set at $p < 0.05$.

The study included 51 patients with a male-to-female ratio of 1.4:1. Most were classified as Child-Pugh Class C (54.9%). Ascites was strongly associated with Child- Pugh class ($P = 0.0036$). Biphasic, monophasic, and triphasic hepatic waveforms correlated with Child-Pugh classification ($P = 0.0015$), and a damping index >0.6 was more frequent in Class C ($P < 0.001$). Esophageal varices were found in 60.8%, with a significant association to both Child-Pugh scores and hepatic waveform patterns.

This study highlights the role of Doppler ultrasonography in assessing cirrhosis severity and complications, confirming its value in cirrhosis staging.

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List of Abbreviations

1) MELD – Model for end stage liver disease.
2) INR – International normalized ration.
3) DI – Damping index.
4) CLD – Chronic liver disease.
5) ICD – International classification of diseases.
6) NAFLD – Non alcoholic fatty liver disease.
7) NASH – Non alcoholic steatohepatitis.
8) PBC – Primary biliary cirrhosis.
9) PSC – Primary sclerosing cholangitis.
10) PDGF – Platelet derived growth factor.
11) HSC – Hematopoietic stem cell.
12) TGF – Transforming growth factor.
13) LSCEC – Liver sinusoidal endothelial cells.
14) TNF – Tumor necrosis factor.
15) HCC – Hepatocellular carcinoma.
16) HVPG – Hepatic venous pressure gradient.
17) HVW – Hepatic vein waveform.
18) WHVP – Wedged hepatic venous pressure.
19) TIPS – Transjugular intrahepatic portosystemic shunt.
20) PFV – Portal flow velocity.
21) PVBF – Portal venous blood flow.
22) GMBF – Glomerular micro blood flow.

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Annexures

Annexure-1: Proforma

- Name:
- Age:
- Sex:
- Occupation:
- IP no:
- Address:
- contact no:
- Past medical and surgical history:
- Family history:
- Personal history:
- Drug & treatment history:
- Relevant clinical examination findings:
- S. Bilirubin (total) (mg/dl):
- S. Albumin (g/dl):
- INR:
- Ascites (absent/slight/moderate):
- Encephalopathy no encephalopathy/grade 1-2/ grade 3-4):
- Hepatic vein waveform (triphasic/biphasic/monophasic):
- Minimum velocity of hepatic venous flow:
- Maximum velocity of hepatic venous flow:
- Damping index:
- Splenoportal index:
- Endoscopic findings:

Annexure-2: Master Chart

S. No	1.Age	2.Sex	3.Ascites	4.Encephalopathy	5.Child Pugh Class	6.Hepatic Venous Waveform	7.Damping Index	8.Splenomegaly	9.Splenoportal Index	10.Oesophageal Varices
1	2	1	1	2	2	3	1	1	2	2
2	2	2	2	2	1	2	1	1	1	2
3	2	1	1	2	2	3	2	1	1	1
4	1	1	1	1	3	3	2	1	2	1
5	1	1	2	2	1	1	1	2	2	1
6	3	1	1	2	3	3	1	1	2	1
7	2	2	2	2	2	2	2	1	1	1
8	3	2	2	2	1	2	1	2	1	2
9	1	1	2	2	3	3	2	1	2	1
10	2	2	1	2	3	2	1	2	2	1
11	3	1	2	2	1	2	1	2	2	2
12	1	2	2	2	3	3	2	1	2	1
13	2	1	1	2	2	3	1	1	2	1
14	3	2	1	2	3	2	2	1	3	1
15	2	1	1	1	3	3	2	1	1	1
16	2	2	2	2	2	2	2	1	2	2
17	3	2	2	2	2	3	1	1	1	1
18	1	1	2	2	3	2	2	1	3	2
19	3	1	1	2	3	3	2	1	2	1
20	2	2	2	2	1	1	1	2	1	1
21	3	1	2	1	3	2	1	2	3	2
22	2	2	1	2	3	2	2	1	1	1
23	1	1	2	2	3	3	2	1	1	1
24	3	1	1	2	3	2	2	2	2	2
25	2	2	1	2	2	1	2	1	2	1
26	1	1	2	2	3	2	2	2	1	1
27	2	2	2	2	3	1	2	1	3	1
28	1	1	1	2	2	3	1	1	1	1
29	2	1	1	2	3	2	2	2	1	2
30	1	2	2	2	1	1	1	1	1	2
31	2	1	1	2	3	3	2	2	2	1

32	2	2	1	2	3	3	2	2	1	1
33	3	1	2	2	1	2	1	2	2	2
34	3	2	2	2	3	2	2	1	2	1
35	2	1	1	2	2	3	1	2	2	2
36	1	1	2	2	2	2	2	1	3	1
37	2	2	1	2	3	2	2	2	3	2
38	2	1	1	2	3	3	2	2	2	1
39	3	1	2	2	2	3	1	1	3	2
40	1	1	1	2	3	3	2	2	3	1
41	2	2	2	2	3	3	2	1	1	1
42	1	1	2	2	1	1	1	2	2	2
43	2	2	1	2	3	2	2	2	3	1
44	2	2	1	2	3	3	2	2	1	1
45	1	1	1	2	2	2	2	1	2	2
46	2	1	2	2	3	2	2	1	3	2
47	2	2	1	1	3	2	2	2	1	2
48	1	1	2	2	2	1	1	1	3	1
49	2	1	2	2	1	1	1	2	1	2
50	2	1	1	2	3	2	2	1	3	2
51	3	2	2	2	2	3	1	2	3	1

Key to Master Chart

1) Age group (in years)
   20-40 – 1
   40-60 – 2
   60-70 – 3
2) Sex
   Male – 1
   Female - 2
3) Ascites
   Present – 1
   Absent - 2
4) Encephalopathy
   Present – 1
   Absent - 2
5) Child Pugh class
   A – 1
   B – 2
   C - 3
6) Hepatic venous waveform
   Triphasic - 1
   Biphasic – 2
   Monophasic - 3
7) Damping index
   ≤0.6 – 1
   >0.6 - 2
8) Splenomegaly
   Present – 1
   Absent – 2
9) Splenoportal index
   4-8 – 1
   8-12 – 2
   >12 - 3
10) Oesophageal varices
   Present – 1
   Absent – 2

Annexure-3: Informed Consent Form

I ..... have understood the information given in the information sheet and is willing to participate in the study titled "Colour doppler ultrasound of the hepatic veins and its association with Child Pugh's score and esophageal varices" conducted by Dr Mohammed Rakheeb CG. The nature, objective, duration and expected effects of the study have been explained to me in....., a language in which I am conversant. I have been informed what I have to do as part of the study. I have had the time and opportunity to enquire about the study and I have been fully satisfied with the explanations

given.

- I am ready to participate voluntarily in this study.
- I agree to cooperate with the research staff and voluntarily undergo the procedures required in the study.
- I understand that I am at liberty to withdraw from this study at any time without justifying my decision to withdraw.
- I know that the results from this study may be forwarded to the appropriate authorities, presented in scientific meetings and published.
- By signing this consent form, I have not given up any legal rights which I am otherwise entitled to as a subject in this study.
- I know that I will get a copy of this consent form which is signed and dated.

Signature of Subject
Name of Subject
Date:

I confirm that I have explained the nature, purpose and expected effects of the study to the subject whose name is printed above

Signature of person providing information
Name of person providing information
Date:

സമ്മതപത്രം

ഞാൻ വിവരപ്പത്രത്തിൽ നൽകിയ വിവരങ്ങൾ മനസ്സിലാക്കി ഡോക്ടർ മുഹമ്മദ് റഫീഖ് സി ജി നടത്തിയ ഹെപ്പാറ്റിക് വെയ്ൻസിന്റെ കളർ ടോപ്പ്ലർ അൾട്രാസൗണ്ട് സ്കാനിങ്ങും ലിവർ സിറാഹോസിസ് എന്ന അസുഖത്തിന്റെ തീവ്രത രേഖപ്പെടുത്തുന്ന ചൈൽഡ് പഗ് സ്കോറും അന്നനാളത്തിലെ ഞരമ്പുകളുടെ വീർക്കലും തമ്മിലുള്ള ബന്ധവും" എന്ന പഠനത്തിൽ പങ്കാളിയാകാൻ സന്നദ്ധനാണ്. പഠനത്തിന്റെ സ്വഭാവം, ഉദ്ദേശം, ദൈർഘ്യം, പ്രതീക്ഷിക്കാവുന്ന ഫലങ്ങൾ എന്നിവ എനിക്ക് പരിചിതമായ ഭാഷയിൽ വിശദീകരിച്ചിട്ടുണ്ട്. പഠനത്തിന്റെ ഭാഗമായി എനിക്ക് ചെയ്യേണ്ട കാര്യങ്ങൾ അറിയിച്ചിട്ടുണ്ട്. പഠനത്തെ കുറിച്ച് അന്വേഷിക്കാൻ എനിക്ക് സമയവും അവസരവും ലഭിച്ചു, നൽകിയ വിശദീകരണങ്ങളിൽ ഞാൻ പൂർണ സംപ്തൃപ്തനാണ്. ഈ പഠനത്തിൽ സ്വമേധയാ പങ്കെടുക്കാൻ ഞാൻ തയ്യാറാണ്. ഗവേഷണ ഉദ്യോഗസ്ഥരുമായി സഹകരിക്കാനും പഠനത്തിൽ ആവശ്യമായ നടപടിക്രമങ്ങൾ നടത്താനും ഞാൻ സമ്മതിക്കുന്നു. പിൻവലിക്കാനുള്ള എന്റെ തീരുമാനത്തെ ന്യായീകരിക്കുന്ന ഏത് സമയത്തും ഈ പഠനത്തിൽ നിന്ന് പിൻവലിക്കാൻ ഞാൻ സ്വാതന്ത്ര്യത്തിലാണെന്ന് ഞാൻ മനസ്സിലാക്കുന്നു. ഈ പഠനത്തിന്റെ ഫലങ്ങൾ ഉചിതമായ മീറ്റിംഗുകളിൽ അവതരിപ്പിച്ചതും പ്രസിദ്ധീകരിച്ചതുമായ ഉചിതമായ അധികാരികളിലേക്ക് കൈമാറ്റം ചെയ്യാമെന്ന് എനിക്കറിയാം. ഈ സമ്മത ഫോം ഒപ്പിടുന്നതിലൂടെ, ഈ പഠനത്തിൽ ഒരു വിഷയമായി ഞാൻ അർഹിക്കുന്ന നിയമപരമായ അവകാശങ്ങളൊന്നും ഞാൻ ഉപേക്ഷിച്ചിട്ടില്ല. ഒപ്പിട്ടതും ഡേറ്റുചെയ്തതുമായ ഈ സമ്മത രൂപത്തിന്റെ ഒരു പകർപ്പ് എനിക്ക് ലഭിക്കുമെന്ന് എനിക്കറിയാം.

പങ്കെടുക്കുന്നയാളുടെ ഒപ്പ്

പേര്

മുകളിൽ അച്ചടിച്ചിരിക്കുന്ന വിഷയത്തിന് പഠനത്തിൻറെ സ്വഭാവം, ഉദ്ദേശ്യം, പ്രതീക്ഷിച്ച ഫലങ്ങൾ എന്നിവ ഞാൻ വിശദികരിച്ചിട്ടുണ്ടെന്ന് ഞാൻ സ്ഥിരീകരിക്കുന്നു

വിവരങ്ങൾ നൽകുന്ന വ്യക്തിയുടെ ഒപ്പ്, പേര്

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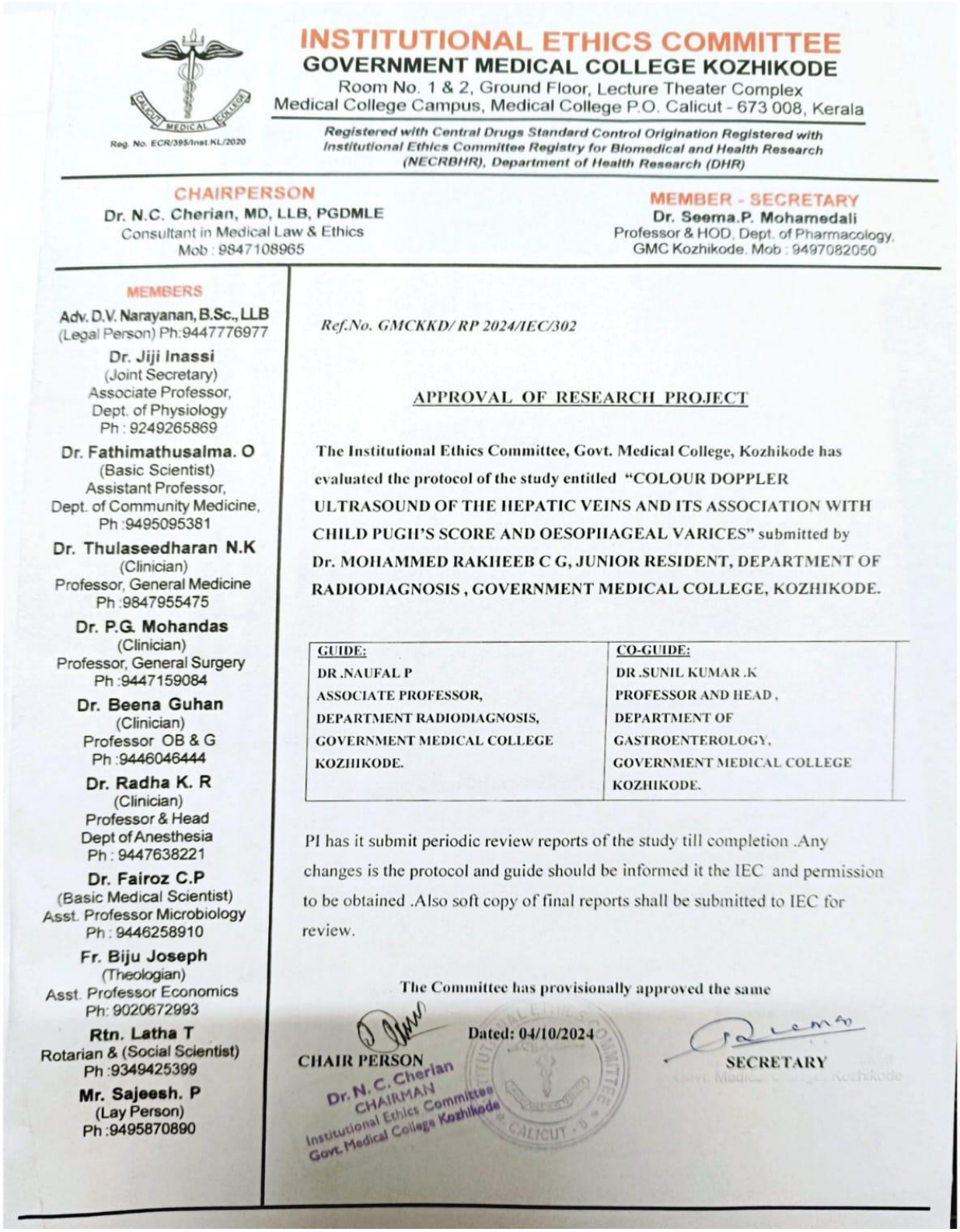

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APPROVAL OF RESEARCH PROJECT

The Institutional Ethics Committee, Govt. Medical College, Kozhikode has evaluated the protocol of the study entitled "COLOUR DOPPLER ULTRASOUND OF THE HEPATIC VEINS AND ITS ASSOCIATION WITH CHILD PUGH'S SCORE AND OESOPHAGEAL VARICES" submitted by Dr. MOHAMMED RAKHEEB C G, JUNIOR RESIDENT, DEPARTMENT OF RADIODIAGNOSIS , GOVERNMENT MEDICAL COLLEGE, KOZHIKODE.

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PI has it submit periodic review reports of the study till completion .Any changes is the protocol and guide should be informed it the IEC and permission to be obtained .Also soft copy of final reports shall be submitted to IEC for review.

The Committee has provisionally approved the same

Dated: 04/10/2024

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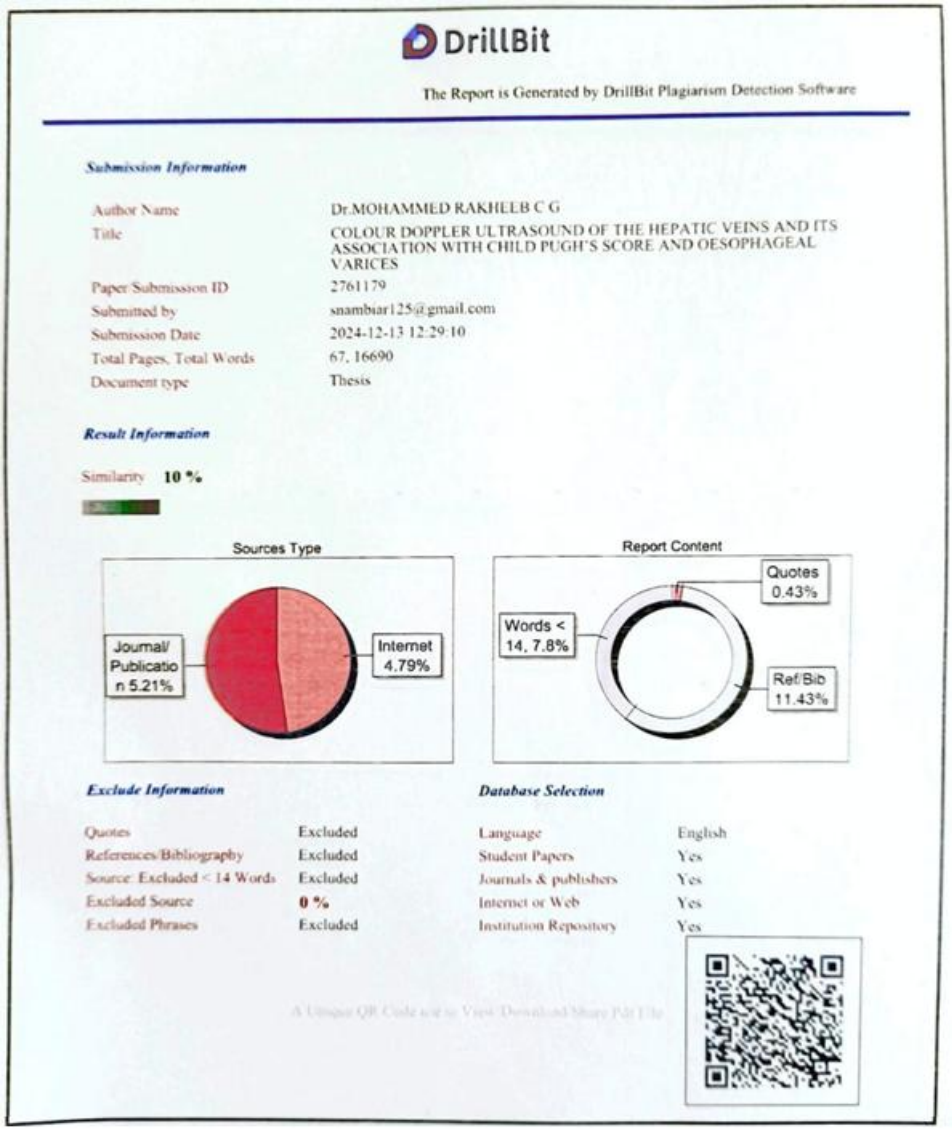

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