“ETIOLOGY, CLINICAL PROFILE, UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS AND OUTCOME OF PATIENTS PRESENTING WITH LIVER CIRRHOSIS WITH PORTAL HYPERTENSION.”

BY

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Dissertation Submitted to the

Shri Dharmasthala Manjunatheshwara University, Dharwad, Karnataka,

In partial fulfillment

of there quirements for the award of degree of

DOCTOR OF MEDICINE

IN

GENERAL MEDICINE

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ACKNOWLEDGEMENT

I am deeply indebted to many for guiding and helping me in my endeavor in making this dissertation a reality. I express my deep sense of gratitude to my respected teacher and guide **DR. HEMAMALINI GURURAJ,** Professor, Department of GENERAL MEDICINE, SDM college of medical sciences and hospital Dharwad, for their valuable guidance and constant encouragement throughout the course of this study.

I express my deep sense of gratitude to my respected teacher and co- guide **DR.PREETHAM HURKADLI,** Professor, Department of GASTROENTEROLOGY, SDM college of medical sciences and hospital Dharwad, for their valuable guidance and constant encouragement throughout the course of this study.

I am extremely grateful to Dr. KIRAN AITHAL, professor and Head, Department of GENERAL MEDICINE for his untiring and constant help and guidance at all time.

My sincere gratitude to and all my teachers of department of GENERAL MEDICINE for their constant support and valuable suggestions at every stage of this study. I am thankful to all my fellow postgraduates, seniors and juniors for their support. I would like to extent my gratitude toward our non-teaching technical staffs as well for their help during the study.

I am grateful to Dr.Niranjan Kumar, Professor and Medical Director, Dr. Rathnamala M. Desai, Professor and Principal, SDM College of Medical Sciences and Hospital Dharwad for permitting me to carry out this study. I will always be grateful to my parents and friends for their, support and encouragement at every step of my life. Finally, I am indebted to all my patients, without whom this study would not have been possible.

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LIST OFABBREVIATIONS USED

CBD	common bile duct
CHD	Common hepatic duct
CLD	Chronic liver disease
CPS	Child Pugh's scoring
INR	International Normalized Ratio
MELD	Model for end stage liver disease
TIPS	Trans jugular intra-hepatic Porto systemic shunt
UGIB	upper gastrointestinal bleed
WHO	World health organization

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ABSTRACT

BACKGROUND: Liver cirrhosis is a common problem faced by physicians worldwide and is also responsible for the 11th most common cause of death globally. Cirrhosis is by far the most frequent cause of portal hypertension. Portal hypertension opens porto-systemic collaterals and leads to the development of oesophageal varices There is an association between esophageal varices and portal hypertensive gastropathy. Large oesophageal varices are at risk of rupture, increasing preventable mortality and morbidity.

AIM AND OBJECTIVES: To study the common clinical presentation of patients with cirrhosis and to study the upper gastrointestinal endoscopy findings in patients with cirrhosis, portal hypertension and to study in-hospital mortality in patients with cirrhosis factors affecting the outcome.

MATERIALS AND METHODS: The study design is a prospective observational study. Had included 100 patients aged >18years age of either gender, diagnosed to have cirrhosis of liver with portal hypertension. All the laboratory parameters and the ultrasonography findings were assessed and analysed.

RESULTS: Liver cirrhosis most common among the 35-50 years with male predominance. Esophageal varices and portal hypertensive gastropathy have been the most common endoscopic findings present in patients with liver cirrhosis. The in-hospital mortality was 18% with septic shock being the most common cause. The portal hypertensive gastropathy correlated well with the presence of oesophageal varices , erosive gastritis and the gastric varices.

CONCLUSION: Liver cirrhosis is common among the age group of 36-50 years with male predominance and in 76% it was ethanol related. The most common clinical presentation is with jaundice and pedal oedema compared with the other clinical features. The common UGI findings were portal hypertensive gastropathy followed by grade 2 oesophageal varicesand 24% of them underwent the endoscopic procedure (23% EVL and 1% endotherapy). The in-hospital mortality of patients with cirrhosis was 18% with septic shock being the most common cause. The portal hypertensive gastropathy correlated well with the presence of oesophageal varices, erosive gastritis and the gastric varices suggesting a common pathophysiology in the formation.

Portal hypertensive gastropathy has a statistical association with anemia as it can cause acute and chronic bleeding leading to iron deficiency anemia, it had no statistical significance with biochemical parameters.

KEY WORDS: Portal hypertension, UGI, Liver cirrhosis, Gastropathy

INTRODUCTION

As per the world health organization (WHO) cirrhosis of liver, is "a diffuse process characterized by fibrosis and conversion of normal liver architecture into structurally abnormal nodules." The normal portal vein pressure is 5 to 10mmHg.

Portal hypertension is 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".[1,2]

Liver cirrhosis is a common problem faced by physicians worldwide and is also responsible for 11th most common cause of death globally. Cirrhosis is by far the most frequent cause of portal hypertension.[3] Portal hypertension opens porto-systemic collaterals and lead to development of esophageal varices and haemorrhoids. Oesophageal varices and portal hypertensive gastropathy were common endoscopic findings present in patients with liver cirrhosis, there was statistically significant association between oesophageal varices and portal hypertensive gastropathy. Large oesophageal varices are at risk of rupture, increase ng the preventable mortality and morbidity. The mortality rate is 20% when patients are treated optimally in hospital. Incidence of first variceal haemorrhage ranges from 20 to 40% within two years. [4,5]

Cirrhosis with portal hypertension is common scenario in India. This can be caused due to alcohol consumption, virus, metabolic diseases like diabetes mellitus. This disease is associated with severe morbidity and mortality because of associated complications, this study will provide us with a comprehensive data regarding etiology, clinical presentation and complications associated with cirrhosis and portal hypertension which will help in better management of patients.[5,6]

Cirrhosis is responsible for about 1.1% of all mortality as estimated by W.H.O. Portal hypertension- a major hallmark of cirrhosis is defined as a portal pressure gradient exceeding

5-10 mm Hg.[7] In portal hypertension, portosystemic collaterals decompress the portal circulation and give rise to varices. Development of esophageal varices and gastrointestinal bleeding represents a serious consequence in patients with portal hypertension. At the time of diagnosis of liver cirrhosis, esophageal varices are present in about 40% of patients with compensated disease and in 60% of those with decompensated disease and ascites.[8,9]

This study helped in assessing associated complications and factors influencing the hospital morbidity and mortality which will help in a better management of the patients with cirrhosis with portal hypertension.

OBJECTIVES

1. **Primary objective:** To study the common clinical presentation of patients with cirrhosis. to study the upper gastrointestinal endoscopy findings in patients with cirrhosis and portal hypertension.

2. **Secondary objective:** To study in hospital mortality in patients with cirrhosis and factors affecting the outcome.

REVIEW OF LITERATURE

RELAVENT ANATOMY

Henri Bismuth summaries that, although many of the advances in hepatic surgery have been linked to improvements in technology, there is no denying the impact of thorough knowledge of the internal anatomy of the liver on improved outcomes. [10]

This is largely due to the work of the French surgeon and anatomist, Claude Couinaud (1922–2008), who detailed his early work in *Le Foie: Études anatomiques et chirurgicales* (*The Liver: Anatomic and Surgical Studies*), in 1957, regarding segmental anatomy of the liver. Couinaud was able to closely examine the intrahepatic anatomy and demonstrated that hepatic functional anatomy is based on vascular and biliary relationships rather than external surface anatomy, improving the safety and feasibility of hepatic surgery today.[10,11]

BRIEF ANATOMY OF LIVER AND PORTAL SYSTEM

The liver is the largest organ, accounting for approximately 2% to 3% of average body weight. The liver has 2 lobes typically described in two ways, by morphologic anatomy and by functional anatomy.[12,13] Located in the right upper quadrant of the abdominal cavity beneath the right hemidiaphragm, it is protected by the rib cage and maintains its position through peritoneal reflections, referred to as ligamentous attachments.

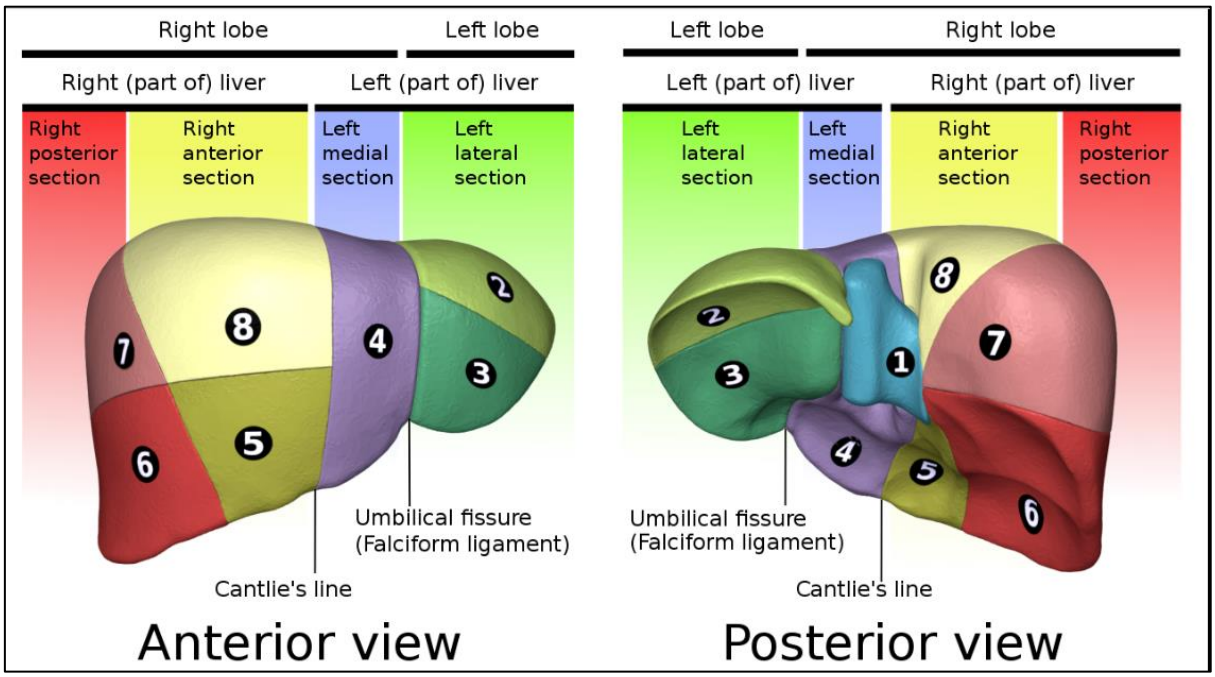


Figure 1: Anatomy of the Liver: Lobes and segments[14,15]

Above is the schematic self-explanatory image of anterior and posterior view of the Liver lobes and the segments.

Traditionally, four lobes are distinguished in the liver based on its external appearance: right, left, caudate, and quadrate.

- Anterior surface: The falciform ligament divides the liver into the right and left anatomic lobes.
- Inferior surface: The quadrate lobe is defined by the gallbladder fossa, porta hepatis, and ligamentum teres hepatis.
- The caudate lobe is delineated by the inferior vena cava groove, porta hepatis, and ligamentum venosum fissure.

Although these lobes are convenient and well known, these structures are not true functional lobes.

The true right and left lobes of the liver are of roughly equal size and are divided not by the falciform ligament, but by a plane passing through the bed of the gallbladder and the notch of the inferior vena cava. This plane, which has no external indications, is called the Cantlie line.

Based on arterial blood supply, portal venous blood supply, biliary drainage, and hepatic venous drainage, the liver is divided into right and left functional lobes, each of which is divided into two segments, and these are further subdivided into two subsegments.

Subdivision is based on the distribution of bile ducts 3 In these systems, the subsegments are assigned numbers from 1 to 8, with the caudate lobe being subsegment 1 and the others following in a clockwise pattern.[14,16]

The portal vein is formed from the confluence of the superior mesenteric vein and the splenic vein. At the hilum, the portal vein divides into right and left branches, upon which the right and left lobes of the liver are based.[16]

The hepatic artery commonly arises from the celiac trunk, although occasionally it arises from the superior mesenteric artery. A common variant is a left hepatic artery that branches from the left gastric artery and a right hepatic artery branch that arises from the superior mesenteric artery.

Within the hilum, the hepatic artery lies anterior to the portal vein and to the left of the bile duct. In the liver, the arteries, portal veins, and bile ducts are surrounded by a fibrous sheath, the Glissonian sheath, whereas the hepatic veins lack this structure.

Three major hepatic veins drain into the inferior vena cava, although in 60% to 85% of persons, the left and middle veins unite to enter the inferior vena cava as a single vein.

The extrahepatic biliary tree is composed of the common hepatic duct, cystic duct, gallbladder, and right and left hepatic ducts. The right and left hepatic ducts drain the right and left lobes of the liver, respectively.

The fusion of the right and left hepatic ducts gives rise to the common hepatic duct. The caudate lobe usually drains to the origin of the left hepatic duct or to the right hepatic duct. The cystic duct usually drains into the lateral aspect of the common hepatic duct below its origin. [16]

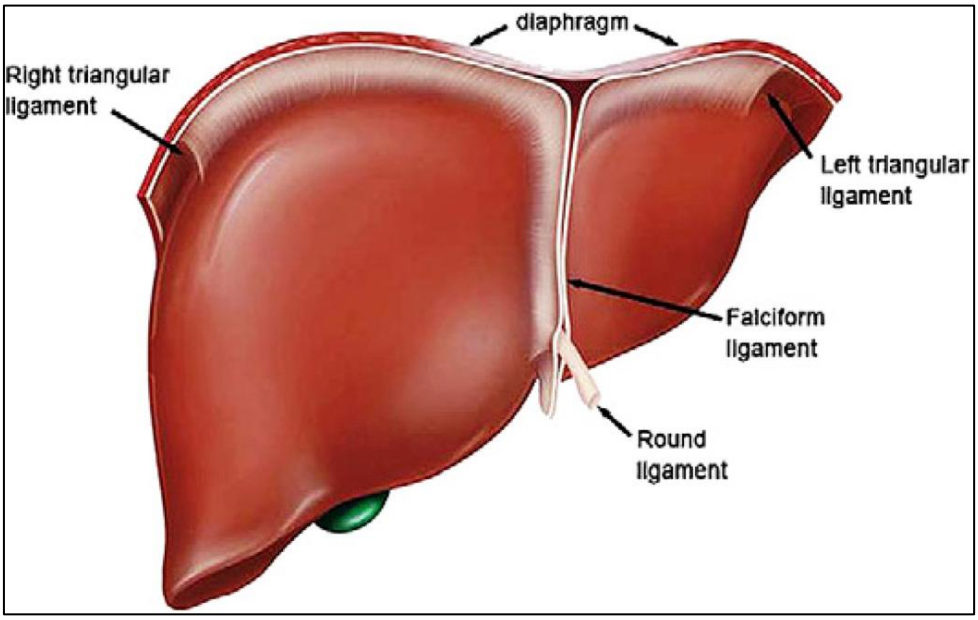


Figure 2: Ligaments attached to the liver[14]

BILIARY SYSTEM[14,16]

Bile, a digestive fluid produced and secreted by the liver, is transported by a series of branching bile ducts known collectively as the biliary tree. At the cellular level, several narrow tubular channels called canaliculi collect the bile generated by each hepatocyte. These canaliculi drain into an intralobular bile duct which collects all the bile from each lobule, the functional unit of the liver.

Intralobular ducts then drain into the interlobular ducts which are located between lobules. The interlobular ducts merge to form the two main bile ducts of the liver: the right hepatic duct (RHD) and the left hepatic duct (LHD).

Extrahepatically, the RHD and LHD coalesce to form the common hepatic duct (CHD) which travels within the hepatoduodenal ligament until coming into contact with the cystic duct, the bile duct which connects to the gallbladder.

The CHD and cystic duct merge to form the common bile duct (CBD). The hepatopancreatic ampulla, also called the hepatopancreatic duct or ampulla of Vater which is a spherical structure located at the site of the confluence of the common bile duct and pancreatic duct, marking the entry point of bile into the second portion of the duodenum.

This is controlled by the smooth muscle fibers of the sphincter of Oddi which opens at the duodenal papilla, allowing bile to flow into the small intestine. Alternatively, bile can travel into the gallbladder for storage via the cystic duct.[17-19]

Intrahepatic Ducts

- Canaliculi: Contain microvilli for increased surface area
- Intralobular ducts: These ducts are located alongside the hepatic artery and the portal vein. Together, these three structures are referred to as portal triads and are ensheathed by a layer of connective tissue known as Glisson's capsule
- Interlobular ducts
- Right hepatic duct: Drains right lobe of the liver (segments V, VI, VII, and VIII)
- Left hepatic duct: Drains Left lobe of the liver (segments II, III, IV).

The caudate lobe of the liver (segment I) is drained by small ducts from both the right and left lobes.

Extrahepatic Ducts

- Extrahepatic segments of the right hepatic and the left hepatic ducts
- Common hepatic duct. Approximately 4 cm in length
- Cystic duct: Outflow tract of the gallbladder. Approximately 7 mm in diameter. Contains valves of Heister
- Common bile duct: Normal width should be less than approximately 6 mm. Approximately 6.0 cm to 8.0 cm in length.

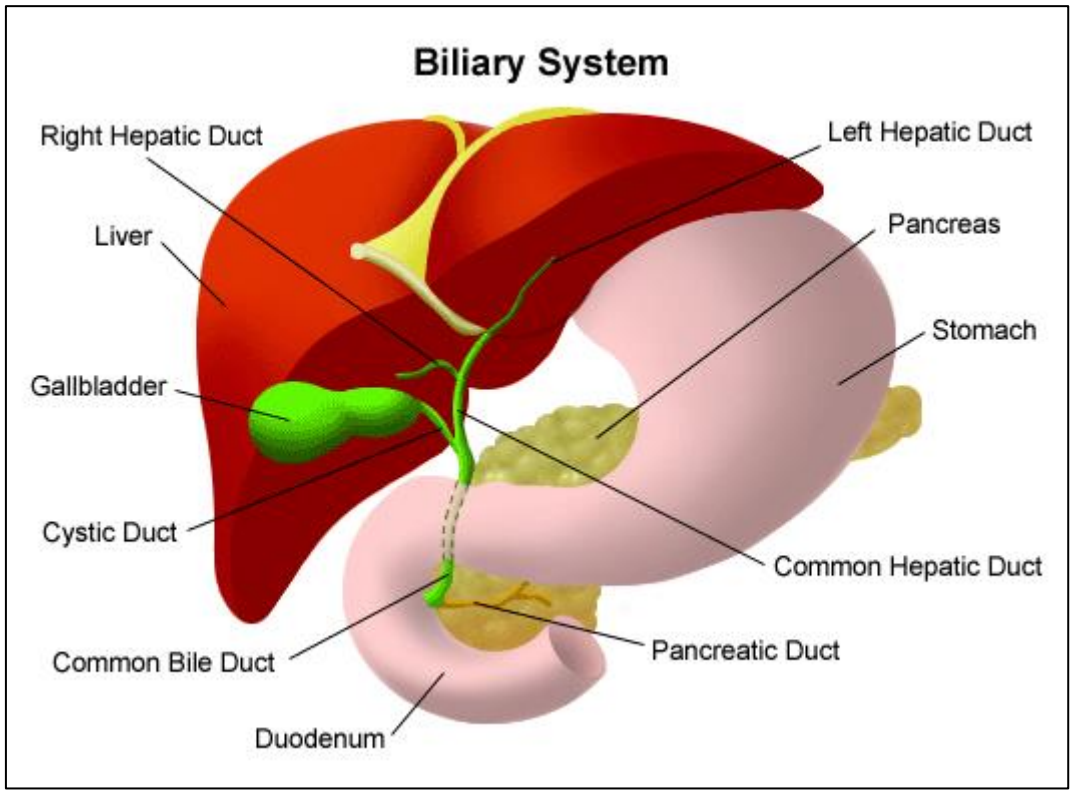


Figure 3: Anatomy of Biliary system[18]

Hepatic encephalopathy: Historical remarks

Association between liver disease and mood disturbances was first recognized Hippocrates (460–371 B.C.), the father of medicine. He described the association between jaundice and acute behavioural disturbances. Based on his humoral theory, he explained the relationship between bile and irritability. [19]

Later GB. Morgagni described a case of hepatic encephalopathy in the 38th letter of the 3rd book of the treatise which is named as '*De sedibus et causis morborum per anatomen indagatis*'. The case he reported was a middle-aged man from Venice who had history of alcohol abuse in the past. This patient had developed ascites and some episodes of agitation followed by prolonged episodes of somnolence and delirium and finally died. His autopsy revealed the existence of liver cirrhosis which named after the scholar "the Morgagni-Laennec cirrhosis."[19,20]

CHRONIC LIVER DISEASE AND HEPATIC ENCEPHALOPATHY

CHRONIC LIVER DISEASE[21,22]

Chronic liver disease (CLD) is a progressive deterioration of liver functions for more than six months. The pathology includes the disorganised synthesis of clotting factors, proteins, detoxification of harmful intermittent products of metabolism and excretion of bile.

This is a continuous process of inflammation, destruction, and regeneration of liver parenchyma, finally leading to fibrosis and cirrhosis.

Etiologies: Exposure to the toxins, chronic alcoholism, infection: of any kind either bacterial, viral or fungal, autoimmune diseases, genetic and metabolic disorders.

Brief Pathophysiology: Cirrhosis is a final stage of chronic liver disease resulting in disruption of architecture of the liver, the formation of widespread nodules, vascular reorganization, neo-angiogenesis and deposition of an extracellular matrix. The underlying

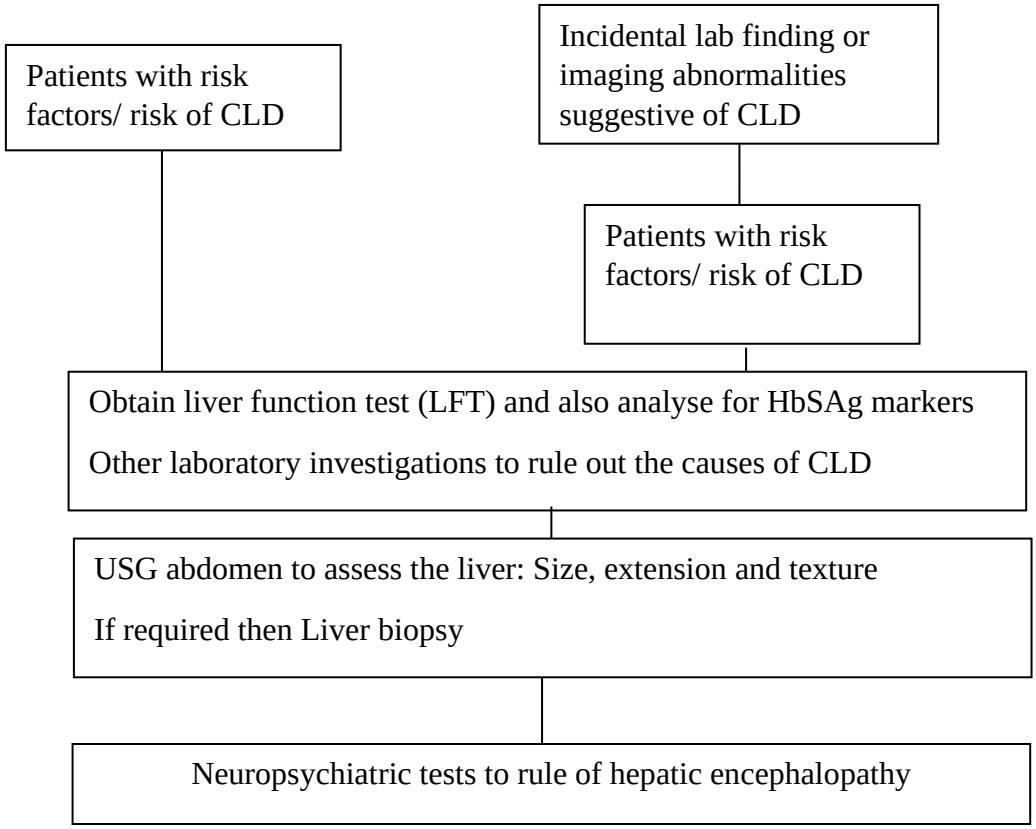
mechanism of fibrosis and cirrhosis at a cellular level is primarily the recruitment of stellate cells and fibroblasts.

Cirrhosis is defined by three main morphologic features.

- Bridging fibrous septa- linking portal tract with one another and portal tract with terminal hepatic veins.
- Parenchymal nodules-hepatocyte encircled by fibrosis.
- Disruption of liver architecture.

Clinical manifestations: Include fatigue, anorexia, weight loss, or depend upon the complication that the patient has developed.

DIAGNOSIS OF CLD



ASSESMENT OF SEVERITY[23]

Severity may be assessed by;

1) **Child Pugh's scoring system:** Based on the clinical and laboratory investigations, each parameter will be graded and the total score will be used for classifying the severity. Which is explained the below table 1

Table 1: Child Pugh's scoring

Clinical and laboratory parameters	**Points**		
	1	**2**	**3**
Ascites	None	Slight	Moderate
Albumin (g/dl)	>3.5	2.8- 3.5	<2.8
Bilirubin (mg/dl)	<2	2-3	>3
Encephalopathy	No	Grade 1 and 2	Grade 3 and 4
INR	<1.7	1.7 – 2.3	>2.3

The severity of cirrhosis:

- Child-Pugh A: 5 to 6 points
- Child-Pugh B: 7 to 9 points
- Child-Pugh C: 10 to 15 points

2) **MELD scoring system:** Model for end stage liver disease (MELD) was initially developed to assess short term prognosis in patients with chronic liver disease who undergo Trans jugular intra-hepatic Porto systemic shunt (TIPS) but its usefulness to assess the prognosis and severity of chronic liver disease has been well validated.

It consists of three variables;

1) Serum bilirubin

2) Serum creatinine

3) Prothrombin time INR (International Normalized Ratio)

3) Liver biopsy

In the present study, we will be using Child Pugh's scoring system for grading the severity of CLD.

Decompensated chronic liver disease can present with one of the following complications;

1. Portal hypertension
2. Hepatocellular insufficiency
3. Spontaneous bacterial peritonitis
4. Coagulopathy
5. Hepatorenal syndrome
6. Hyperestrinism
7. Hepatic encephalopathy

PORTAL HYPERTENSION

Portal hypertension is a detrimental complication resulting from obstruction of portal blood flow, such as cirrhosis or portal vein thrombosis.

Once portal hypertension develops, it influences extrahepatic vascular beds in the splanchnic and systemic circulations, causing collateral vessel formation and arterial vasodilation. This helps to increase the blood flow into the portal vein, which exacerbates portal hypertension and eventually brings the hyperdynamic circulatory syndrome.[24,25]

Consequently, it will lead to esophageal varices or the ascites.

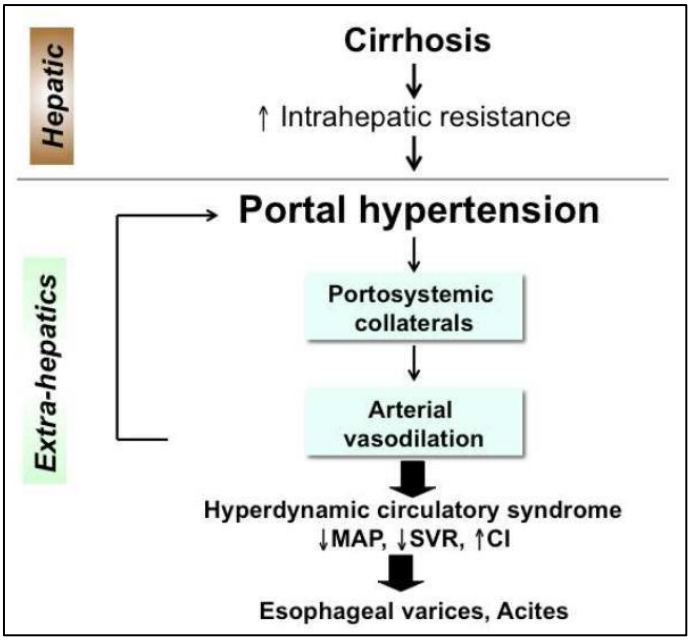


Figure 4: Flow chart illustrating the pathophysiology of portal hypertension

PATHOPHYSIOLOGY OF PORTAL HYPERTENSION

Anatomically, the portal vein is formed by the union of the superior mesenteric vein and the splenic vein. The mesenteric vein collects blood from the splanchnic circulation. Thus, portal venous inflow is determined by the state of constriction or dilatation of splanchnic arterioles.[26]

The initial mechanism in the genesis of portal hypertension is an increase in vascular resistance that can occur at any level within the portal venous system. [24,26,27]

Portal hypertension is therefore classified as;[24,27]

- Prehepatic: In conditions such as Portal or splenic vein thrombosis
- Intrahepatic: As observed in liver Cirrhosis
- Post hepatic: As in Budd-Chiari syndrome.

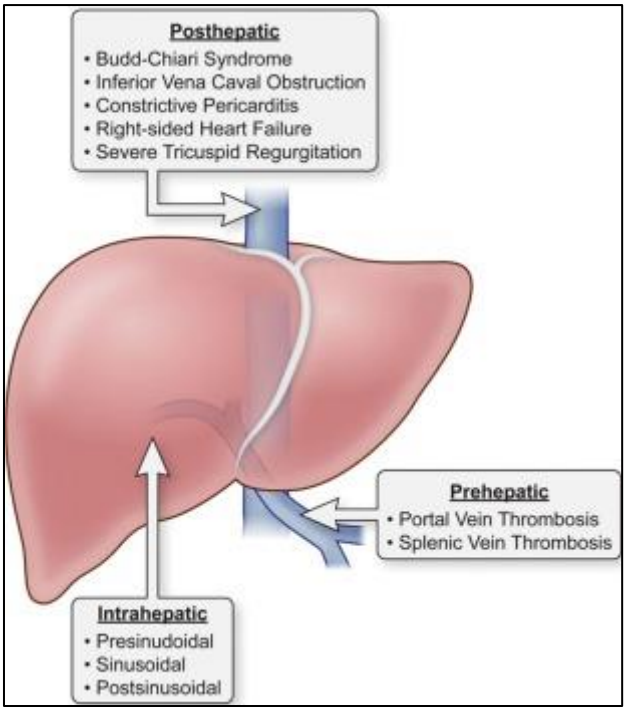


Figure 5: Approach towards portal hypertension[28]

The most common cause of portal hypertension is cirrhosis. In cirrhosis, the increased resistance is mostly caused by hepatic architectural distortion due to fibrosis and regenerative nodules but about a third of the increased resistance is caused by intrahepatic vasoconstriction, amenable to vasodilators.

- This is caused by the activation of stellate cells with active contraction of myofibroblasts and vascular smooth muscle cells in portal venules. Which in turn is caused by increased endogenous vasoconstrictors, such as endothelin, and reduced nitric oxide bioavailability.
- Portosystemic collaterals develop as a consequence of the high pressure in the portal vein and ameliorate the increased resistance. However, even when portal blood flow is entirely diverted through collaterals, portal hypertension persists because of a concomitant increase in portal venous inflow, which in turn is caused by splanchnic vasodilatation. Mostly mediated by an increase in nitric oxide.

- The most important collaterals are those that constitute gastroesophageal varices. Although the formation of collaterals had been assumed to be the result of dilatation of pre-existing vascular channels, recent studies have implicated a process of neo-angiogenesis.
- This process has been shown to contribute not only to portal-systemic collaterals but also to increased splanchnic blood flow via arteriolar-capillary network.[25-27]

CLINICAL PRESENTATION OF PORTAL HYPERTENSION

- Patients usually have no symptoms until complications arise. Hematemesis from bleeding varices is the most common presentation.
- Melena without hematemesis can also be present.
- The major sings include jaundice, gynecomastia, palmar erythema, spider nevi, testicular atrophy, ascites, pedal edema, or asterixis due to hepatic encephalopathy.
- Prominent abdominal wall veins may be visible, which is an attempt to divert the portal blood flow via the paraumbilical veins into the caval system.
- In caput-medusae, the blood flow is away from the umbilicus. However, in inferior vena cava obstruction, the blood flow is toward the umbilicus to reach the superior vena cava system. A venous hum may be audible near the xiphoid process or umbilicus.
- Splenomegaly is another reliable sign in the diagnosis of portal hypertension but not confirmatory.

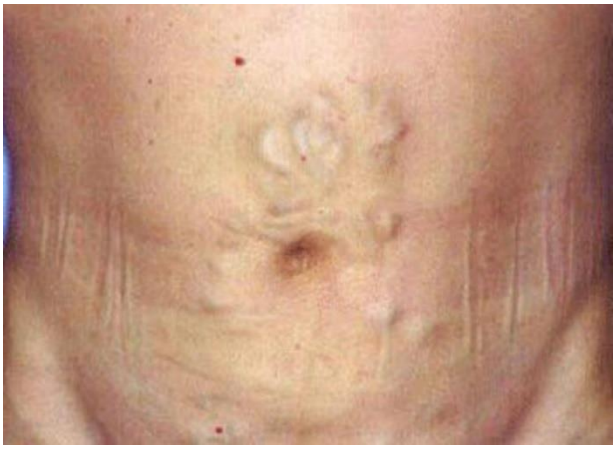


Figure 6: Illustration of Caput medusae

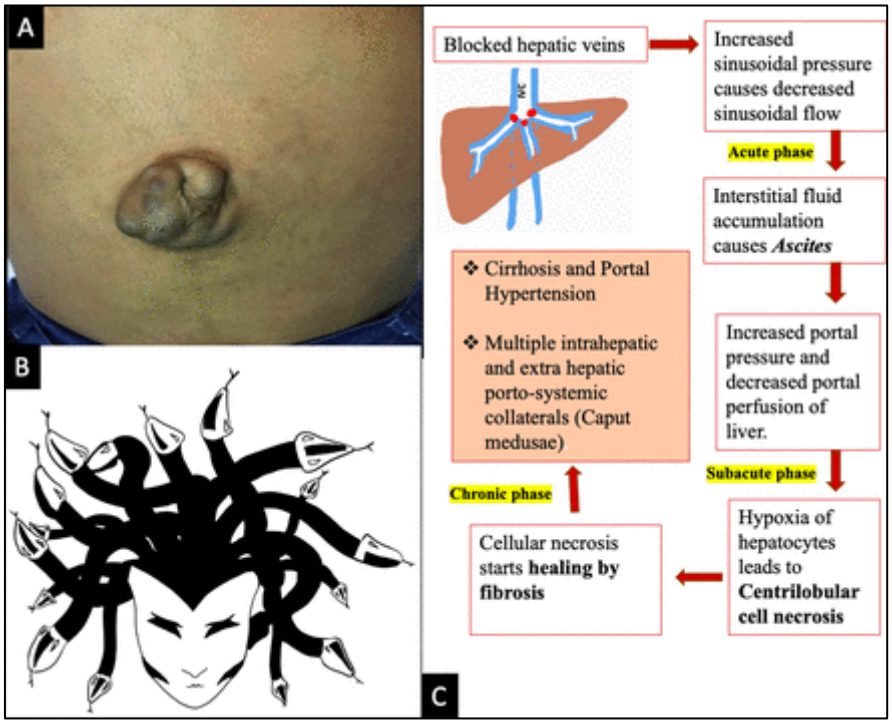


Figure 6: Clinical feature and pathogenesis of portal hypertension[30]

(A) Shows a patient with portal hypertension with tuft of vascular channels at umbilicus and prominent abdominal wall veins

(B) shows a diagrammatic representation of the Greek mythology character ‘medusa’

(C) Schematic representation of pathophysiology of Budd Chiari syndrome.

MEASURING THE PORTAL PRESSURE

Measurement of portal pressure in patients with portal hypertension is important in the evaluation of the efficacy of different portal-hypotensive pharmacologic therapies.

- The most used method to assess portal pressure is the catheterization of the hepatic vein with determination, via a balloon catheter, of the hepatic vein pressure gradient (HVPG), which is the difference between the wedged or occluded hepatic venous pressure and the free hepatic venous pressure.[31]
- Normal HVPG is 3 to 5 mm Hg. In patients with compensated cirrhosis, an HVPG greater than or equal to 10 mm Hg predicts the development, not only of varices, but of complications that mark the transition from compensated to decompensated cirrhosis.[31-33]
- Changes in HVPG during pharmacologic therapy have also been shown to be predictive of clinical outcomes.
- In patients with a history of variceal hemorrhage, a decrease in HVPG to less than 12 mm Hg or a decrease greater than 20% from baseline significantly reduces the risk of recurrent hemorrhage, ascites, encephalopathy and death.
- In patients with compensated cirrhosis, even lower reductions in HVPG (>10% from baseline) have been associated with a reduction in the development of varices, first variceal haemorrhage and ascites. [31-33]

EVALUATION OF THE PORTAL HYPERTENSION

- Ultrasonography (USG)
- Endoscopy
- Doppler ultrasonography
- Computed tomography (CT)
- Magnetic resonance imaging (MRI)

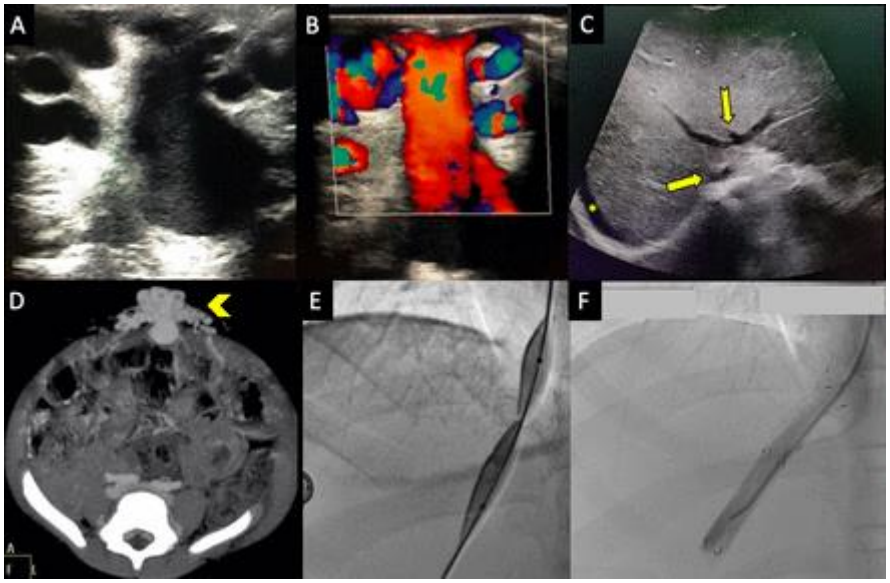


Figure 7: Illustrating Caput medusae in various diagnostic tools[34]

ENDOSCOPY

Esophagogastroduodenoscopy (EGD), also known as endoscopy is a diagnostic endoscopic procedure that includes visualization of the oropharynx, esophagus, stomach, and proximal duodenum. It is one of the most common procedures that a gastroenterologist performs.

The main equipment used;

Gastroscopes

The standard gastroscopes have a diameter of 10 mm with an instrument channel of 2.8 mm. In children weighing less than 10 kg, endoscopes smaller than 6 mm in diameter for routine endoscopy should be used. A gastroscope with a large operating channel measuring 3.8 to 4.2 mm is useful in severe acute upper GI bleeding. High-definition gastroscopes with optical zoom should be available to screen for pre-malignant gastric or duodenal lesions.

Accessories

The biopsy forceps (standard and jumbo) are needed for tissue sampling. For retrieval of a foreign body during esophagogastroduodenoscopy (EGD), rat tooth forceps, alligator

forceps, retrieval net, polypectomy snare, over tubes of esophageal and gastric lengths, and a foreign body protector hood should be available. Additional equipment may be required if therapeutic procedures are anticipated.[35]

PREPARATION OF THE PATIENT[36]

Routine endoscopy in children and adults is usually performed in an outpatient setting using parenteral or general anesthesia. Occasionally, endoscopy is necessary at the hospital bedside or in an operating room.

- **Diet:** Preparation for elective upper endoscopy procedure involves a period of fasting. As per American Society for Anaesthesiologists (ASA) guidelines, patients should fast a minimum of 2 hours after ingestion of clear liquids and 6 hours after ingestion of light meals. In emergency situations or in conditions where gastric emptying is impaired, the potential for pulmonary aspiration of gastric contents must be considered to determine;

(1) level of sedation,

(2) whether endotracheal intubation should be considered to protect the airway or

(3) whether the procedure should be delayed.

- **Medications:** Most medications can be continued and are usually taken with a small sip of water before endoscopy, although diabetes medications need to be adjusted due to the period of fasting before the procedure. American Society for Gastrointestinal Endoscopy (ASGE) guidelines should be followed for decisions regarding the management of anti-thrombotic agents or for the use of antibiotic prophylaxis in at-risk patients before the endoscopy.

- **Sedation and Monitoring**: Sedation is used in most patients not only to minimize discomfort but also to provide amnesia for the procedure. All patients undergoing upper endoscopy require pre-procedural evaluation to assess their risk for sedation and to manage potential problems related to pre-existing health conditions.

  For therapeutic endoscopic procedures such as foreign body removal or in patients in whom cooperation is not anticipated, including very young patients, general anesthesia may be required. ASGE guidelines recommend routine monitoring of vital signs in addition to clinical observation for changes in cardiopulmonary status during all endoscopic procedures performed under sedation.

- One of the major requirements is written informed consent.

INDICATION FOR ENDOSCOPY

Diagnostic

- Persistent upper abdominal pain or pain associated with alarming symptoms such as weight loss or anorexia
- Dysphagia, odynophagia or feeding problems
- Intractable or chronic symptoms of GERD
- Unexplained irritability in a child
- Persistent vomiting of unknown etiology or hematemesis
- Iron deficiency anemia with presumed chronic blood loss when clinically an upper gastrointestinal (GI) source is suspected or when colonoscopy is normal
- Chronic diarrhea or malabsorption
- Assessment of acute injury after caustic ingestion

- Surveillance for malignancy in patients with premalignant conditions such as polyposis syndromes, previous caustic ingestion, or Barrett esophagus

Therapeutic

- Foreign body removal
- Dilation or stenting of strictures
- Esophageal variceal ligation
- Upper GI bleeding control
- Placement of feeding or draining tubes
- Management of achalasia (botulinum toxin or balloon dilation)

CONTRAINDIATION

Absolute Contraindications

- Perforated bowel
- Peritonitis
- Toxic megacolon in an unstable patient

Relative Contraindications

- Severe neutropenia
- Coagulopathy
- Severe thrombocytopenia or impaired platelet function
- Increased risk of perforation including connective tissue disorders, recent bowel surgery or bowel obstruction

- Aneurysm of the abdominal and iliac aorta

ENDOSCOPIC FINDINGS OF PORTAL HYPERTENSION IN LIVER CIRRHOSIS

Chaudhary S et al had included 89 patients with liver cirrhosis were enrolled with mean age of 51.84±12.26 years and male: female ratio of 3.68:1. As per Child Pugh classification (CTP) 45 patients (51%) were in Class C, 33 patients (37%) were in Class B and 11 patients (12%) were in Class A. Esophageal varices were present in 51 (57.3%) patients. According to Westby classification;

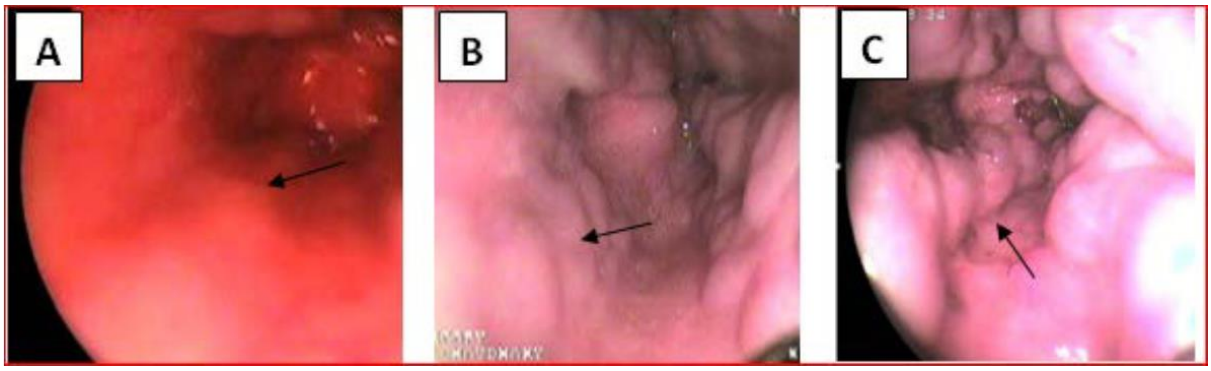
Grade I esophageal varices in 17 (19.1%)

Grade II esophageal varices were seen in 26 (29.2%)

Grade III esophageal varices were seen in 8 (8.9%) patients was observed.

Portal hypertensive gastropathy (PHG) was seen in 64 (71%) patients. The association between esophageal varices and PHG grade was found statistically significant.

Hence, they concluded that liver cirrhosis was more commonly seen in middle age males. Esophageal varices and portal hypertensive gastropathy were common endoscopic findings present in patients with liver cirrhosis. There was statistically significant association between esophageal varices and PHG. Below is the endoscopic image obtained by their study.



Esophageal varices as per Westaby classification A: Grade I, B Grade II and C Grade III.[37]

Mohammad S et al had aimed to assess the endoscopic findings in patients presenting with acute upper gastrointestinal bleed (UGIB). They found that esophageal varices among 65% of their recruited study population was related to portal hypertension secondary to liver cirrhosis. [38]

Another clinical study by **Svoboda P et al** had analysed 151 patients suffering from the cirrhosis of the liver underwent a prospective endoscopic examination of the upper digestive tract.

The most frequent diagnoses in the group with the cirrhosis of the liver included oesophagus varices (64.9%), portal hypertension gastropathy (45.7%) and the peptic ulcer of the gastro-duodenum (25.8%). A normal diagnosis in the endoscopy of the upper digestive tract was found only in 8.6%.

Other diagnoses comprised reflux oesophagitis (13.2%), diaphragm hiatus hernia (12.6 %), duodenogastric reflux (8.6 %), gastric antrum erosion (4.6 %), aphthic gastropathy (3.3 %), rhagades of the cardium (2%), gastric polyp (1.3%), mycotic oesophagitis, gastric carcinoma, oesophagus carcinoma and oesophagus achalasy (0.7% each). Further on the study discusses possible causes of the high incidence of peptic ulcers in the patients with the cirrhosis of the liver. [39]

An observational, cross-sectional study was conducted to study the upper gastrointestinal endoscopic abnormalities in patients with alcoholic liver disease by **Yalamanchi RP et al.** They had recruited 97 patients with ALD. They found that on analysing the outcome of upper gastrointestinal endoscopy, 94.73% cirrhotic patients were found to have esophageal varices 23.68% cirrhotic patients were found to have

esophago-gastro-duodenal PHG (P = 0.04) and 15.78 % cirrhotic patients were found to have Gastric antral vascular ectasia (GAVE). None of the patients with USG findings of fatty liver / acute hepatitis had the above endoscopic abnormalities.[40]

Kumar A et al was another similar study who has assessed the various aetiology of upper GI bleed. They found that the mean age of patients was 48.98 ±14.50 years with male to female ratio of 2.57:1. The most common lesions causing UGI bleed were related to portal hypertension (esophageal and gastric varices) and were seen in 67% of patients. Non portal hypertensive lesions causing UGI bleed (peptic and other lesions) were seen in 46% patients. Twenty six percent patients had combination of lesions while endoscopy was normal in 3% patients.

Rebleeding within 15 days was seen in 11 patients out of whom 3 died during same admission. Out of other 8 patients with rebleed, readmission was seen in 6 patients while 2 patients had minor bleed. We found no correlation of mortality and rebleed with factors like age, history of liver disease, diabetes, NSAIDs use, peptic ulcer disease and presence of cirrhosis. They did not find any correlation between rebleed and death was found to be statistically significant.[41]

MATERIALS AND METHODOLOGY

Present study was a prospective observational hospital based study.

Study setting: The present study was carried out on the patients admitted in wards of SDM college of medical sciences and hospital, sattur, Dharwad.

Study population were selected based on the below mentioned Inclusion and Exclusion criterias.

Inclusion criteria:

- All patients aged >18years age of either gender, diagnosed to have cirrhosis of liver with portal hypertension admitted in Medicine wards and icu. S.D.M College of Medical Sciences and Hospital, Sattur, Dharwad 580009.
- Patients willing to participate in the study

Exclusion criteria:

- Portal hypertension requiring emergency management
- History of trauma
- Non-cirrhotic portal hypertension
- Portal hypertension with mass per abdomen

Sample size

Patients presenting with cirrhosis of liver with portal hypertension admitted to SDM college of medical sciences& hospital. 94 patients(to account for study dropout total number of 100 cases will be taken)

Sample size calculation: Zx^2 PQ/e^2 Zx=1.96 P=57.3% Q=42.7% e=10% sample size population=94 (100) prevalence=57.3 Convenient sampling.

STUDY PROCEDURE

Sampling procedure:

After obtaining the clearance from Institutional Ethics committee, all patients aged 18 years and above diagnosed to have cirrhosis of liver with portal hypertension with upper gastro intestinal bleed admitted in Medicine wards S.D.M College of Medical Sciences and Hospital, Sattur, Dharwad 580009.

Informed consent was taken after explaining in detail about the study procedure and the outcome of it.

Data collection method:

Patients had been screened for the eligibility and those fulfilling the selection criteria was briefed about the nature of the study. In case of comatose patients, the relatives/caretakers were informed about the study.

The patients/caregivers expressing their willingness to participate in the study were enrolled after obtaining a written informed consent.

Demographic data such as age and sex had been recorded. History of other co-morbid conditions such as, Hypertension, diabetes mellitus, personal history such as habits of alcohol consumption were asked on face to face interview.

A thorough physical examination was conducted for vitals such as the pulse rate, blood pressure and respiratory rate followed by systemic examination was conducted.

The diagnosis of cirrhosis of liver with USG findings, blood investigations and correlation with upper gastro intestinal endoscopy was done to look for presence or absence of esophageal varices. These findings were recorded on a predesigned and pretested proforma.

Investigations:

All the patients will be evaluated for the following tests.

a. complete haemogram and blood group: hemoglobin, total count, platelets, mean corpuscular volume
b. Random blood sugar
c. blood urea
d. serum creatinine
e. serum electrolytes(sodium, potassium, chloride)
f. liver function test (total bilirubin, direct bilirubin, serum glutamic oxaloacetic transaminase /serum glutamic pyruvic transaminase, alkaline phosphatase, albumin)
g. PT, INR
h. Viral markers.

Upper gastrointestinal endoscopy.

Child Pugh score was assessed for all the study population.

1. serum bilirubin
2. serum albumin
3. ascites
4. INR
5. hepatic encephalopathy

STATISTICAL METHODS:

Data is analyzed using SPSS software version 21 and Excel. Categorical variables are given in the form of frequency table. Continuous variables are given in Mean ± SD/ Median (Min, Max) form. Chi-square test is used to check the dependency between categorical variables. Two-sample t test is used to compare means of variables over groups. P-value less than or equal to 0.05 indicates statistical significance.

RESULTS

Data contains measurements on 100 subjects whose age ranges from 21 - 93 years with mean age 51.78 ± 14.75 years. The following table gives the distribution of subjects according to different variables.

Table 1: Distribution of demographic details

Variables	Sub Category	Number of Subjects (%)
Age (Years)	20-35	12 (12%)
	36-50	42 (42%)
	51-65	26 (26%)
	66-80	15 (15%)
	81-95	5 (5%)
	Mean ± SD Median (Min, Max)	51.78 ± 14.75 49 (21, 93)
Gender	Female	18 (18%)
	Male	82 (82%)

It can be observed that mean age of patients in the present study was 51.78 ± 14.75 with age range 21-93 years. Most of the subjects were in their third, fourth, fifth and sixth decade of life and together they constituted 68% (n=68) of total study population. Highest number of subjects belong to 36-50 age group. The male were 82 (82%) and female of 18(18%).

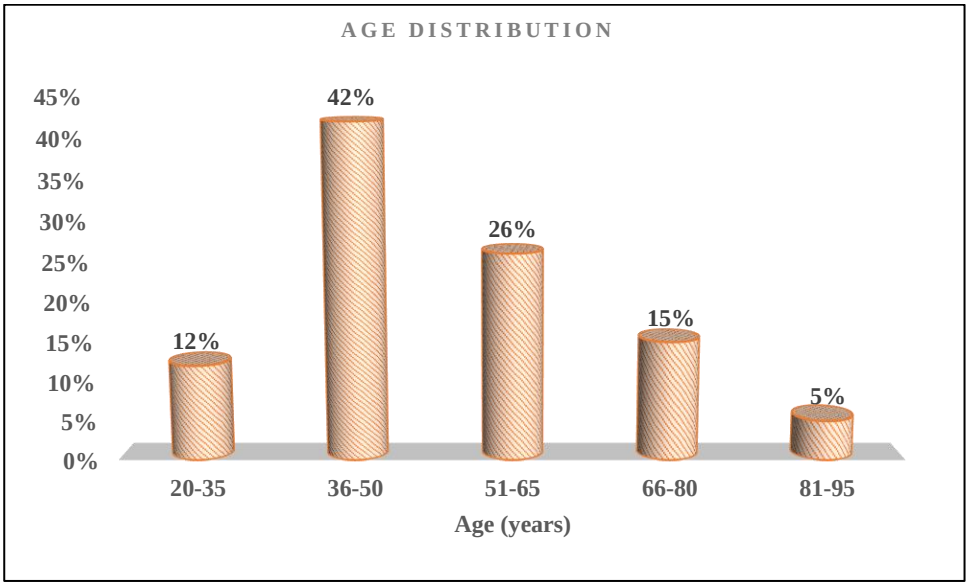


Figure 1: Distribution of subjects based on age

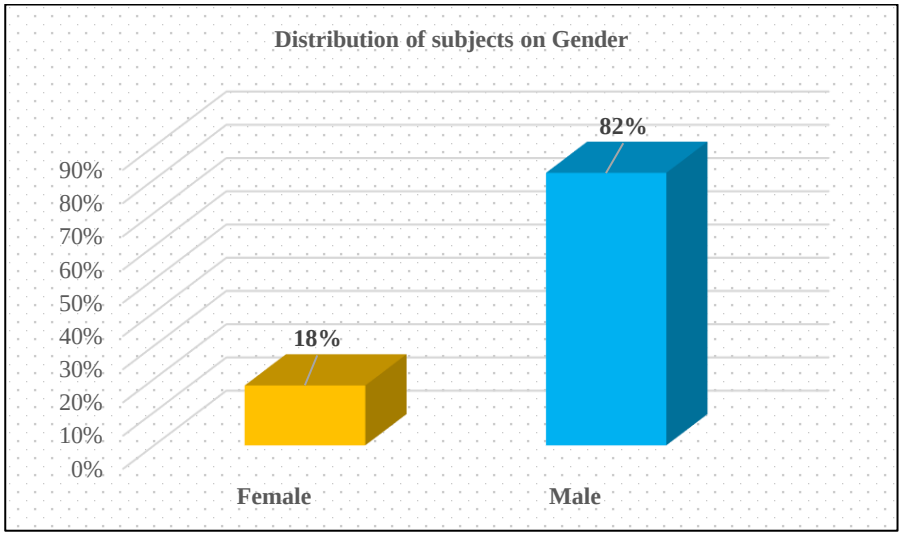


Figure 2: Distribution of subjects based on Gender

Table 2: Distribution of symptoms

Pain Abdomen	No	71 (71%)
	Yes	29 (29%)
Jaundice	No	36 (36%)
	Yes	64 (64%)
Abdominal Distension	No	40 (40%)
	Yes	60 (60%)
Pedal Edema	No	31 (31%)
	Yes	69 (69%)

Fever	No	75 (75%)
	Yes	25 (25%)
Altered Sensorium	No	71 (71%)
	Yes	29 (29%)
Bleeding Manifestations	No	70 (70%)
	Yes	30 (30%)

Pain in abdomen was observed in 29 (29%), Jaundice in 64(64%), Abdominal Distension in 60(60%) and Pedal Edema in 69 (69%) of subjects. Fever was observed in 25(25%), Altered Sensorium in 29(29%) and Bleeding Manifestations in 30(30%) of subjects.

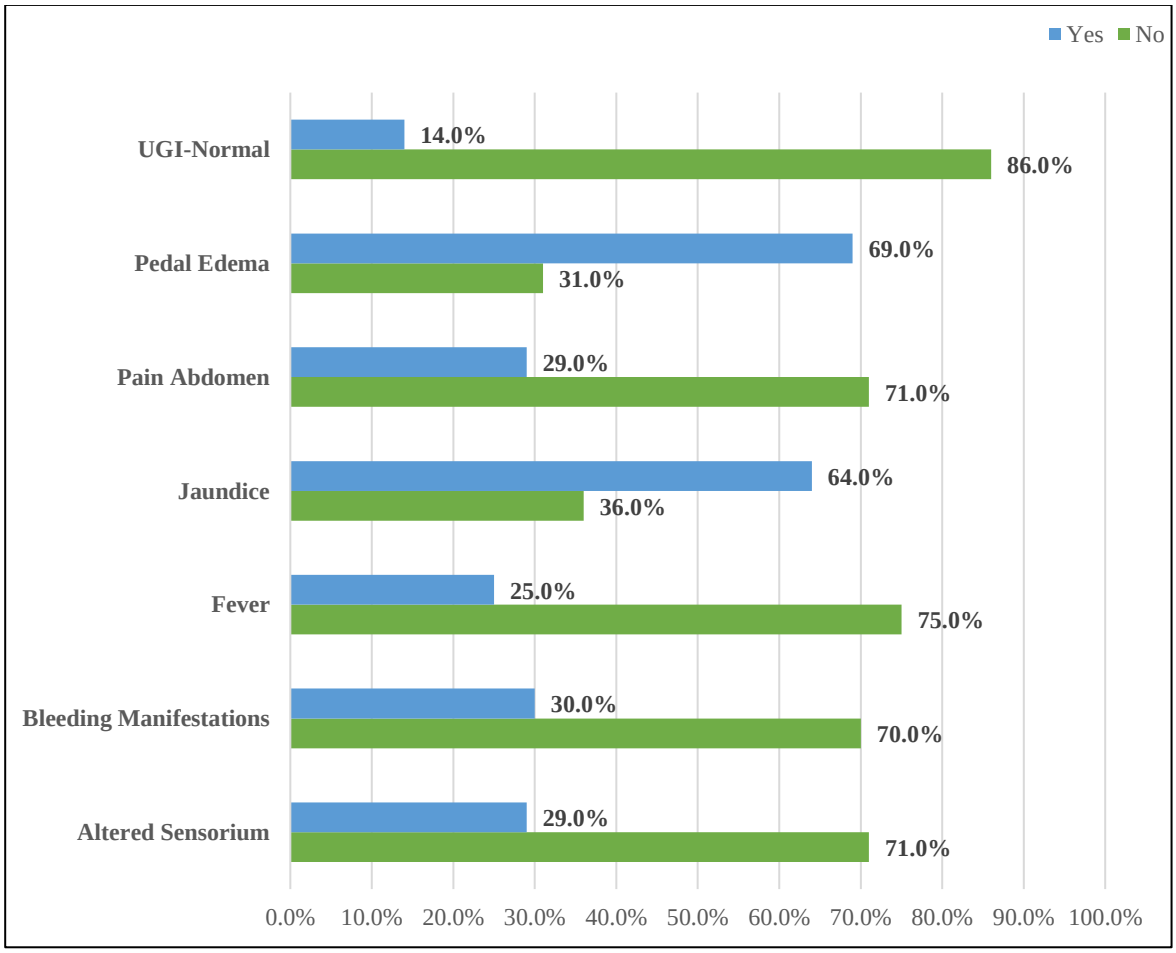


Figure 3: Distribution of subjects based on various variables

Table 3: Distribution of details of UGI

UGI-Normal	No	86 (86%)
	Yes	14 (14%)
GRADE1 V	No	72 (72%)
	Yes	28 (28%)
GRADE2 V	No	67 (67%)
	Yes	33 (33%)
GRADE3 V	No	78 (78%)
	Yes	22 (22%)

14 (14%) of subjects had UGI-Normal. GRADE1 V, GRADE2 V and GRADE3 V were observed in 28%, 33% and 22% of subjects respectively. Upper gastrointestinal (UGI) endoscopy findings in subjects was done and looked for varices, portal hypertensive gastropathy or any other endoscopic findings. UGI Endoscopy findings were normal in 14(14%) subjects. There were 15 (15%) subjects with gastric varices.

Table 4: Distribution of PHG and other findings

Gastric Varices	No	85 (85%)
	Yes	15 (15%)
PHG	No	36 (36%)
	Yes	64 (64%)
Erosive Gastritis	No	79 (79%)
	Yes	21 (21%)
Hiatus Hernia	No	94 (94%)
	Yes	6 (6%)
Duodentis	No	86 (86%)
	Yes	14 (14%)

PHG was observed in 64(64%), Hiatus Hernia was observed in only 6(6%).

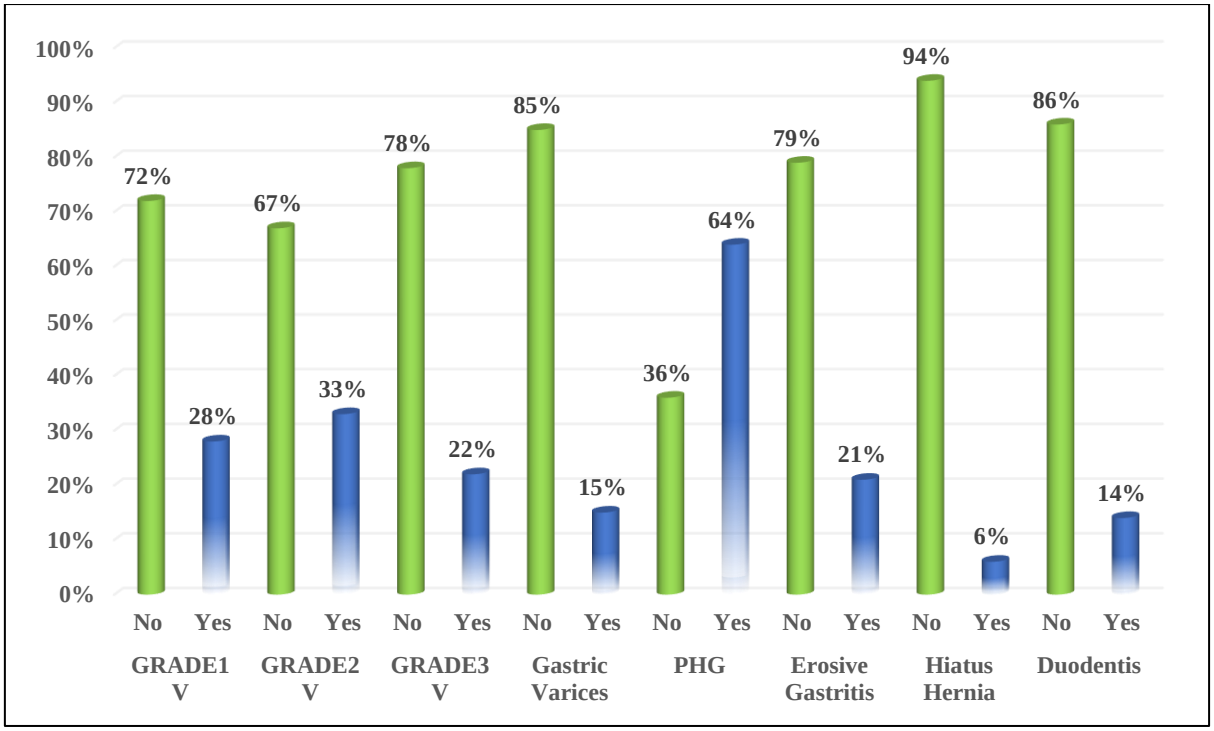


Figure 4: Distribution of subjects based on various variables

Table 5: Distribution of Child Pugh Score

Child Pugh Score	A	10 (10%)
	B	29 (29%)
	C	61 (61%)

The subjects were grouped according to Child Pugh score in which 61 (61%) were in Class C, 29 (29%) were in Class B and remaining 10 (10%) were in Class A. It was observed that majority of the subjects belong to class C which shows that they were in advance stage of liver disease.

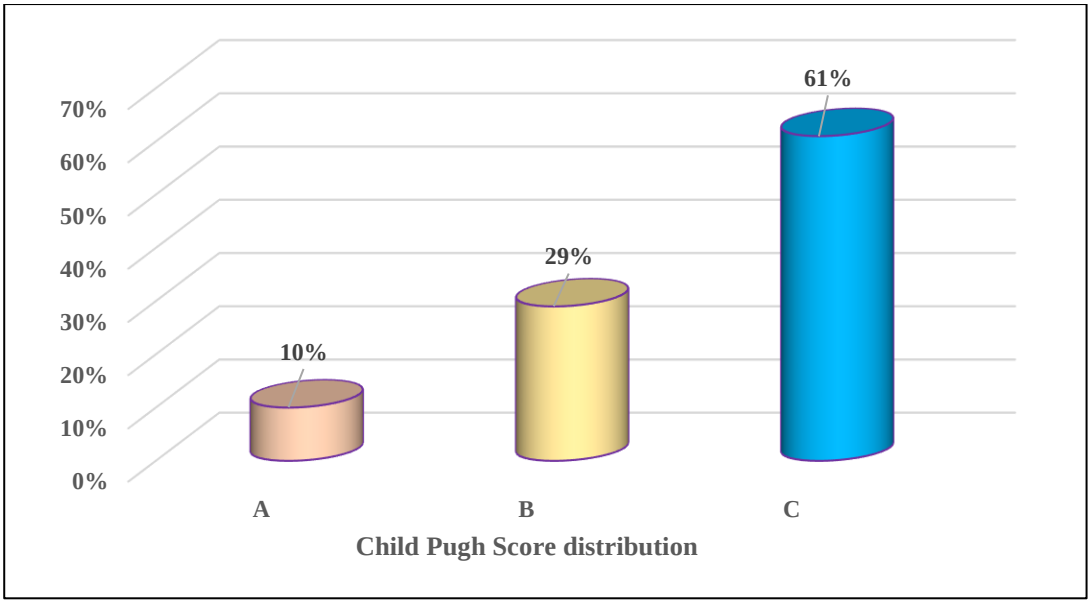


Figure 5: Distribution of subjects based on Child Pugh Score

Table 6: Distribution of etiological factors

Etiology	Autoimmune	1 (1%)
	Cryptogenic	1 (1%)
	Ethanol	76 (76%)
	HBsAG	5 (5%)
	Ethanol+HBsAG	2 (2%)
	HCV	5 (5%)
	NASH	8 (8%)
	Wilsons Disease	2 (2%)

It was observed that most common cause of cirrhosis was ethanol ingestion which was found in 76 (76%) subjects. NASH was the second most common cause which was seen in 8 (8%) subjects followed by HCV in 5 (5%) and HBsAG in 5 (5%) subjects.

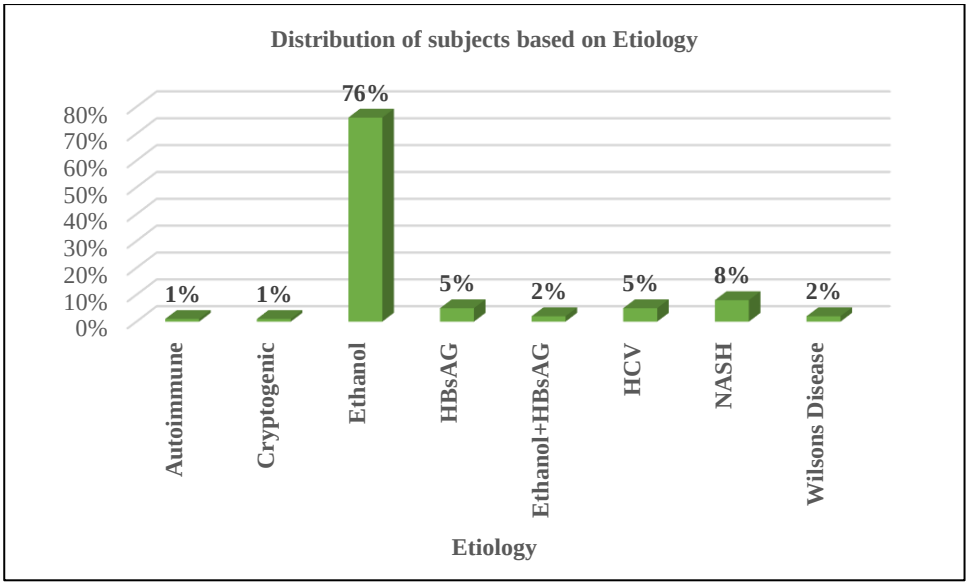


Figure 6: Distribution of subjects based on Etiology

Table 7: Distribution of endoscopy and its details

Endoscopic Procedure	Endotheraphy	1 (1%)
	EVL	23 (23%)
	No	76 (76%)

Majority of subjects endoscopic procedure finding was nil with 76(76%) followed by EVL findings in 23(23%) subjects.

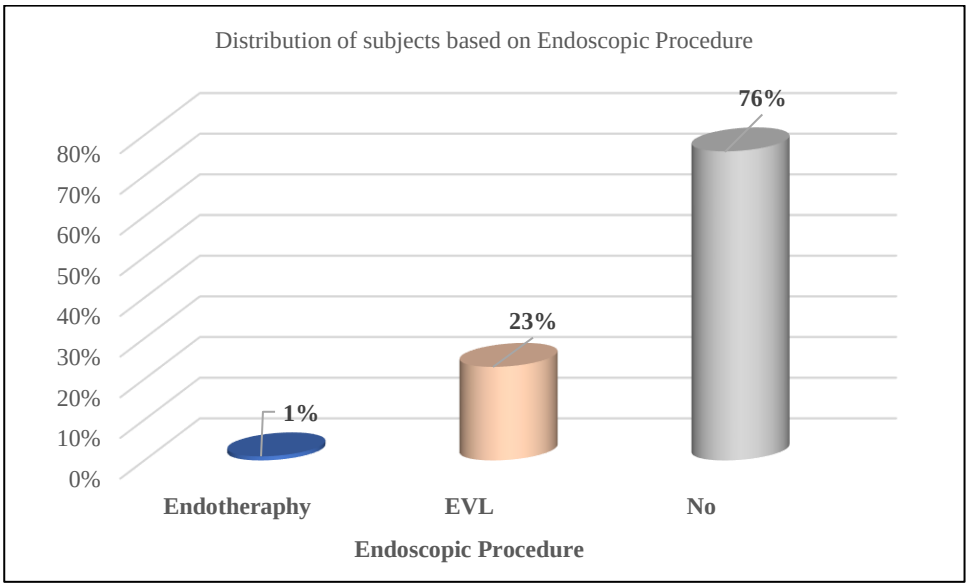


Figure 7: Distribution of subjects based on Endoscopic Procedure

Table 8: Distribution of mortality

Mortality	No	81 (81%)
	Yes	19 (19%)

Mortality was observed in 19(19%) of subjects.

Table 9: Distribution of causes of mortality

Cause of Mortality	Cardiogenic Shock	2 (10.52%)
	Haemorrhagic Shock	1 (5.26%)
	Hypovolemic Shock	3 (15.78%)
	Septic Shock	11 (57.89%)
	Severe Metabolic Acidosis, HRS	1 (5.26%)

Septic shock was the most common cause of death, with the incidence of 11(57.89%) followed by three patients with hypovolemic shock.

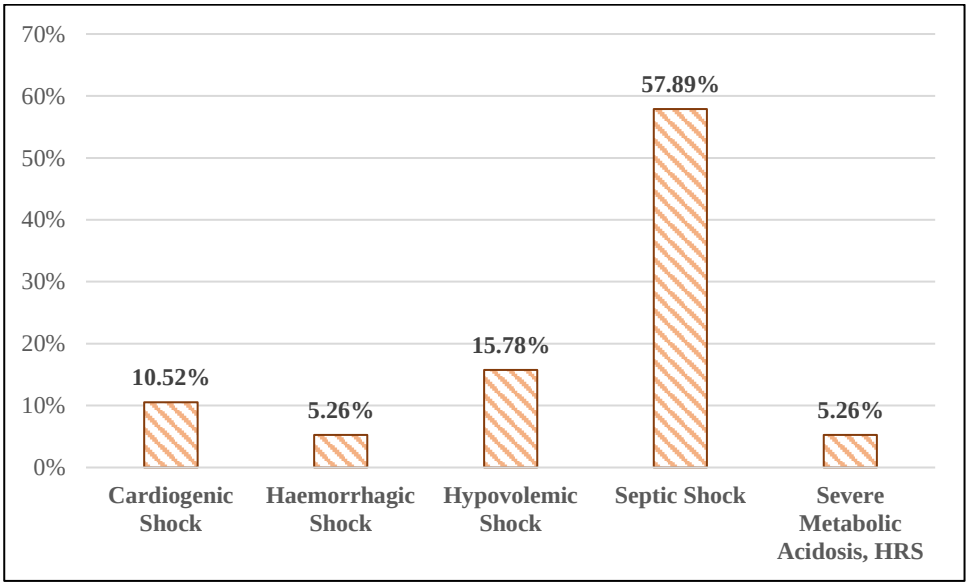


Figure 8: Distribution of subjects based on Cause of Mortality

Table 10: Distribution according to baseline characteristics of the subjects.

Variables	Sub Category	Number of Subjects (%)
SBP (mmHg)	Mean ± SD Median (Min, Max)	109.9 ± 15.99 110 (70, 140)
DBP (mmHg)	Mean ± SD Median (Min, Max)	69.72 ± 11.3 70 (40, 96)
Haemoglobin (gm/dl)	Mean ± SD Median (Min, Max)	8.62 ± 1.95 8.6 (3.8, 13.8)
TLC (/cumm)	Mean ± SD Median (Min, Max)	9335.92 ± 4811.83 8770 (2990, 28080)
Platelets (/cumm)	Mean ± SD Median (Min, Max)	1.31 ± 0.85 1.1 (0.25, 6.5)
PT	Mean ± SD Median (Min, Max)	23.01 ±7.62 21.95 (12.6, 71.5)
INR	Mean ± SD Median (Min, Max)	2.07 ± 1.03 1.85 (0.9, 7.8)
Pulse	Mean ± SD Median (Min, Max)	87.99 ± 16.56 84 (54, 130)
Serum Urea (mg/dl)	Mean ± SD Median (Min, Max)	43.52 ± 25.38 36.5 (9, 132)
Serum Creatine (mg/dl)	Mean ± SD Median (Min, Max)	1.5 ± 1 1.17 (0.38, 5.29)
Total Protein (mg/dl)	Mean ± SD Median (Min, Max)	6.24 ± 0.85 6 (3.2, 8.1)
Albumin (mg/dl)	Mean ± SD Median (Min, Max)	2.08 ± 0.611 2 (1.04, 4)
Total Bilirubin (mg/dl)	Mean ± SD Median (Min, Max)	5.16 ± 6.43 2.7 (0.2, 34.7)
AST (IU/L)	Mean ± SD Median (Min, Max)	86.77 ± 74.14 68.5 (16, 494)
ALT(IU/L)	Mean ± SD	48.58 ± 42.36

	Median (Min, Max)	38 (12, 349)
ALP (IU/L)	Mean ± SD Median (Min, Max)	133.72 ± 89.88 113 (30, 768)
Duration	Mean ± SD Median (Min, Max)	7.45 ± 5.69 6 (1, 30)

The following table gives the mean difference between blood parameters with PHG.

From two sample t test, we observe that, there is significant difference in mean of PHG with SBP, DBP, haemoglobin, TLC. There is no significant difference in mean of PHG with Platelets, PT, INR, Serum Urea, Serum Creatine, Total Protein, Albumin, Total Bilirubin, AST, ALT and ALP.

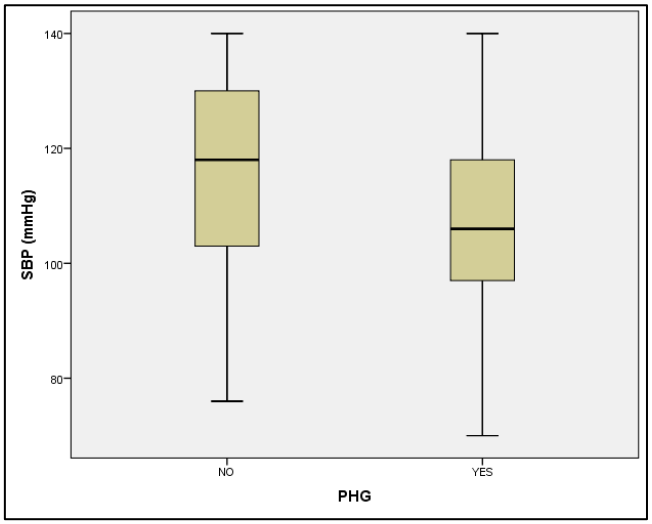


Figure 9: Mean plot of PHG over SBP.

Table 11: Mean difference between blood parameters with PHG.

Variables	**PHG**	**Mean ± SD** **Median (Min, Max)**	**p-value**
SBP (mmHg)	No	115 ± 16 118 (76, 140)	**0.011**[t*]
	Yes	107 ± 15 106 (70, 140)	
DBP(mmHg)	No	73 ± 12 73 (48, 96)	**0.014**[t*]
	Yes	68 ± 10	

		70 (40, 90)	
Haemoglobin (gm/dl)	No	9.4 ± 1.9 9.3 (6.1, 13.8)	**0.004**[t*]
	Yes	8.2 ± 1.9 8.3 (3.8, 13.6)	
TLC (/cumm)	No	7876 ± 3101 7210 (3040, 15740)	**0.022**[t*]
	Yes	10157 ± 5398 9280 (2990, 28080)	
Platelets (/cumm)	No	1.4 ± 1.09 1.08 (0.25, 6.5)	0.485[t]
	Yes	1.27 ± 0.69 1.1 (0.38, 3.6)	
PT	No	22.29 ± 6.52 20.5 (15, 43.4)	0.476[t]
	Yes	23.43 ± 8.20 23 (12.6, 71.5)	
INR	No	2.05 ± 0.71 2.01 (1.12, 4.2)	0.989[t]
	Yes	2.07 ± 1.19 1.8 (0.9, 7.8)	
Serum Urea (mg/dl)	No	38.6 ± 21.3 30 (9, 102)	0.145[t]
	Yes	46.3 ± 27.2 37.5 (13, 132)	
Serum Creatine (mg/dl)	No	1.44 ±1.11 1.05 (0.5, 5.29)	0.627[t]
	Yes	1.54 ± 0.95 1.2 (0.38, 4.8)	
Total Protein (mg/dl)	No	6.45 ± 0.78 1.05 (4.8, 8)	0.073[t]
	Yes	6.13 ± 0.88 6 (3.2, 8.1)	

Albumin (mg/dl)	No	2.23 ± 0.63 2.11 (1.04, 3.7)	0.081[t]
	Yes	2.01 ± 0.59 1.87 (1.1, 4)	
Total Bilirubin (mg/dl)	No	4.28 ± 4.89 1.9 (0.2, 19)	0.303[t]
	Yes	5.66 ± 7.14 2.8 (0.31, 34.7)	
AST (IU/L)	No	82 ± 58 74 (17, 337)	0.620[t]
	Yes	90 ± 82 68 (16, 494)	
ALT(IU/L)	No	44 ± 21 37 (17, 113)	0.390[t]
	Yes	51 ± 50 39 (12, 349)	
ALP (IU/L)	No	138 ± 126 107 (46, 768)	0.707[t]
	Yes	131 ± 62 116 (30, 291)	

*Abbreviation: t – Two sample t test, * indicates statistical significance.*

DISCUSSION

As we had discussed in the previous sections, the Portal hypertension is 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". Liver cirrhosis is one of the most common problems faced by physicians worldwide. Also, the incidence of associated portal hypertension has been increased due to the increased pressure in portal vessels. [1,7]

These will be opening the collaterals and leads to oesophageal varices, even at higher portal pressure, these might rupture and lead to further complications. Many clinical studies we came across had difference in the associated risk factors and the further complications but the incidences and the conditions had varied widely. Hence, the present study was taken to assess the associated complications and factors influencing the hospital morbidity and mortality which will help in a better management of the patients with cirrhosis with portal hypertension.[7]

This was a prospective observational study conducted by including 100 patients diagnosed with liver cirrhosis. The mean age of our patients in the present study was 51.78 ± 14.75 with age range 21-93 years. Most of the subjects were in their third, fourth, fifth and sixth decade of lifeand together they constituted 68% (n=68) of total study population. Highest number of subjectsbelong to 36-50 age group. Similar to our observations, a review of epidemiological survey on the presentation of liver cirrhosis by **Sajja KC et al** had reported that the age of presentation in patients with certain causes of cirrhosis differed among the different ethnic groups.[52] African American patients with alcoholic cirrhosis had an older average age of 54 ± 10 years than both Hispanics, whose average age was 50 ± 11 and Whites it was found to be 51 ± 9 years. Whereas in Indian patients, it was 56 ±13 years.[43,44]

Chaudhary et al who had similar objectives as the present study also reported that 51.84±12.6 years is the average age of their study population.[37] This was even in consistent with the clinical observation of **Maskey R et al**.[46]

In the present study, the males were 82 (82%) and female of 18(18%). Similar to our observation, **Guy J et al** also had observed that men are 2-fold more likely to die from chronic liver disease and cirrhosis than are women.[47] **Becker U et al** explains that as the incidence of chronic alcoholism is comparatively higher among men, one of the related complications cirrhosis is also found to be common among them.[48] Even Indian clinical studies such as **Mishra D et al** and others also had observed significantly higher number of male patients being affected with cirrhosis than females. Similarly, **Chaudhary et al** also found that the male: female population in their study being 3.68: 1.[37] **Bhattarai S et al** and **Pathak OK et al** also reported the similar findings with respect to the distribution of gender in their trial.[50]

In our study, of all the clinical complaints patient came with, pain abdomenwas observed in 29 (29%), Jaundice in 64(64%), Abdominal Distension in 60(60%) and PedalEdema in 69 (69%) of subjects. Fever was observed in 25(25%), Altered Sensorium in 29(29%) and Bleeding Manifestations in 30(30%) of subjects. Jaundice, Ascites and features of coagulopathy were the most common findings observed even in the clinical study conducted by **Ray G et al.**[52]

Unlike our observation, the commonest presenting complain was disorientation in 35 patients (39.3%) and abdominal distension in 28 (31.5%) among the patients with liver cirrhosis as reported by **Chaudhary et al**. Their study population were also presented with altered sleep like major complaints but we did not find such complaints indicating the involvement of central nervous system.[37] Whereas in **Bhattacharyya et al,** the most common symptoms were pedal odema, reported among 80.5% of the population, abdominal

distention in 74.3%, Gastro intestinal bleed among 43.4%, jaundice (36.3%), low urine output (31%) and altered sensorium was the least but not negligible with the incidence of 23%.[51] We could observe that the distribution of clinical symptoms also varies with demographical area.

Among our study subjects, who were grouped according to ChildPugh score in which 61 (61%) were in Class C, 29 (29%) were in Class B and remaining 10 (10%) were in Class A. It was observed that majority of the subjects belong to class C which shows that they were in advance stage of liver disease. This observation was in consistent with the outcome reported by **Mansour A et al** and **Garrison RN et al,** the oldest clinical studies who had reported that as the ChildPugh score increases, the severity of liver involvement also increases.[53,54] Another Indian recent clinical study by **Kumar AS et al** also had described that among the patients with the Child-Pugh score of 10 and above, which is Class C, indicated a grave prognosis and if aggressive intervention is not undertaken soon the mortality is certain in such patients.[41] Even **Chaudhary et al** found that 45/89 patients (51%) were in Class C, 33 patients (37%) were in Class B and 11 patients (12%) were in Class A.[37] Another Indian research by **Tiwari et al** found that 12.4% patients were in class A, 35.1% in CTP class B and 52.5% patients were in CTP class C. The common reason behind the Indian patients presenting with higher degree of liver cirrhosis than other developed countries might be due to poor socio-economical status which is leading to lack of seeking medical care at earliest and the lack of awareness of the disease and its progression. [55]

In the present study, 14 (14%) of subjects had UGI-Normal. GRADE1 V, GRADE2 V and GRADE3 varices were observed in 28%, 33% and 22% of subjects respectively. PHG was observed in 64(64%), Hiatus Hernia was observed in only 6(6%). Mortality was observed in 19(19%) of subjects. Like our study, Esophageal varices were classified according to

Westaby classification. Grade I esophageal varices were seen in 17 (19.1%) patients, grade II esophageal varices were seen in 26 (29.2%), grade III esophageal varices were in 8 (8.9%) patients in **Chaudhary et al.**[37] In **Pathak OK et al** grade I, grade II and grade III varices were diagnosed among 33%, 17% and 8% of their study samples respectively. Whereas 42% of the patients had no varices in their clinical study. [37]

Among 51 patients with esophageal varices in their study, one column of varix was present in 9 (17.65%) patients, two columns of varices were present in 15 (29.41%) patients and 3 columns of varies were present 20 (39.22%) patients and four columns were present in 7 (13.73%) patients.

In this study, Upper gastrointestinal (UGI) endoscopy findings in subjects were done and looked for varices,portal hypertensive gastropathy or any other endoscopic findings. UGI Endoscopy findings were normal in 14(14%) subjects. There were 15 (15%) subjects with gastric varices. Similar to our study, UGI was done in all the patients and looked for varices, portal hypertensive gastropathy or any other endoscopic findings. UGI Endoscopy findings were normal in 24 (27%) patients. Esophageal Varies were present in 51 (57.3%) patients in **Chaudhary et al.** which had comparatively higher patients with variceal bleed than our study population.[37] **Battarai et al** had found gastro-oesophageal varices among 57.5% of their patients.[50]

It was observed that most common cause of cirrhosis was ethanol ingestion which was found in 76 (76%) subjects. NASH was the second most common cause which was seen in 8 (8%) subjects followed by HCV in 5 (5%) and HBsAg in 5 (5%) subjects. Majority of subjects endoscopic procedure finding was nil with 76(76%) followed by EVL findings in 23(23%) subjects. Similar to this, even **Mishra D et al** reported that 63.3% had alcohol-related cirrhosis followed by 19.8% had viral hepatitis-related cirrhosis in their study.[49] Even other

clinical studies such as **Sharma et al, Pati et al** and others also have been found that chronic alcohol consumption being the commonest cause for cirrhosis. [6]

Also, the present study observed significant difference in mean of PHG withSBP, DBP, haemoglobin, TLC. There is no significant difference in mean of PHG with Platelets, PT, INR, Serum Urea, Serum Creatine, Total Protein, Albumin, Total Bilirubin, AST,ALT and ALP. This outcome was contrary to our observation in **Chaudhary et al.** who did observe the significant association with INR and prothrombin time.[37]

There was significant positive association between PHG and Grade 1 and 3 of Esophageal varices. Also, significance can be observed for Gastric Varices and Erosive Gastritis. However, there is no significant association between PHG and Hiatus Hernia, Duodenitis. Unlike our study, PHG was seen in 64 (71%) patients. PHG was mild in 54 (84.38%) patients and severe in 10 (15.62%) patients in **Chaudhary et al** and there was significant positive association between esophageal varices with PHG in their study population.[37] 67.1% patients were found with PHG in the study done by **Tiwari et al**.[55]

From the obtained data of our clinical study, we observed that Jaundice had significantly higher positive association with endoscopic finding of GRADE3 V, Abdominal Distension is associated significantly with Hiatus Hernia, Pedal Edema with GRADE 1 V and Hiatus Hernia. Bleeding Manifestations isassociated significantly with UGI-Normal, GRADE3 V and Hiatus Hernia.

CONCLUSION

Liver cirrhosis is common among the age group of 36-50 years with male predominance, and in 76% it was ethanol related. The most common clinical presentation is with jaundice and pedal oedema compared with the other clinical features. patients with proven cirrhosis of liver and who underwent upper gastrointestinal endoscopy, the common findings were portal hypertensive gastropathy followed by grade 2 oesophageal varices, and 24% of them underwent the endoscopic procedure (23% EVL and 1% endotherapy). The in-hospital mortality of patients with cirrhosis was 18% with septic shock being the most common cause (spontaneous bacterial peritonitis and bronchopneumonia).

the portal hypertensive gastropathy correlated well with the presence of oesophageal varices, erosive gastritis and the gastric varices suggesting a common pathophysiology in the formation.

Portal hypertensive gastropathy has a statistical association with anemia as it can cause acute and chronic bleeding leading to iron deficiency anemia, it had no statistical significance with biochemical parameters.

SUMMARY

- We had taken the present clinical trial to study the common clinical presentation of patients with cirrhosis. to study the upper gastrointestinal endoscopy findings in patients with cirrhosis and portal hypertension and to study in hospital mortality in patients with cirrhosis and factors affecting the outcome.
- 100 subjects whose age ranges from 21 - 93 years with mean age 51.78 ± 14.75 years.
- 82% Males and 18% females were present in our study.
- Pain in abdomen was observed in 29 (29%), Jaundice in 64(64%), Abdominal Distension in 60(60%) and Pedal Edema in 69 (69%) of subjects. Fever was observed in 25(25%), Altered Sensorium in 29(29%) and Bleeding Manifestations in 30(30%) of subjects.
- 14 (14%) of subjects had UGI-Normal. GRADE1 V, GRADE2 V and GRADE3 V were observed in 28%, 33% and 22% of subjects respectively.
- UGI Endoscopy findings were normal in 14(14%) subjects. There were 15 (15%) subjects with gastric varices.
- Child Pugh score C among 61 (61%) was the most common.
- Ethanol ingestion which was found in 76 (76%) subjects was the most common etiology.
- On endoscopic procedure EVL findings in 23(23%) subjects.
- Mortality was observed in 19(19%) of subjects and the major cause of which was Septic Shock among 11 (57.89%).
- There is no significant difference in mean of PHG with Platelets, PT, INR, Serum Urea, Serum Creatine, Total Protein, Albumin, Total Bilirubin, AST, ALT and ALP.
- Hence, we concluded that Liver cirrhosis most common among the patients aged more than 50 years with male predominance. Esophageal varices and portal

hypertensive gastropathy have been the most common endoscopic findings present in patients with liver cirrhosis. Also, there is strong positive correlation between the severity of cirrhosis and the incidence of PHG.

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ANNEXURES

CONSENT FORM

I, the undersigned _____have been explained in my own vernacular language, about the study and that my participation in study is voluntary. I have been explained about the risks involved in the study and have been given enough time to clear my doubts and rights as study participant. In case I have questions related to the study, I have been asked to contact Dr. MADHU S PATIL.

(Mobile No: 6360929001)

Signature or the left thumb print of participant or legally authorized representative.

Participants name _____ Signature_____

Witness name _____ Signature_____

Investigator's name_____ Signature_____

Date_____ Place_____

PROFORMA FORMAT

Name:

Address:

Age:

Gender:

Occupation:

D.O.A:

Education:

D.O.D:

Marital status:

HOSPITAL NO:

Unit:

Ward:

PRESENTING COMPLAINTS: SYMPTOMS PRESENT / ABSENT DURATION

1. Jaundice 2. Abdominal distension 3. Pedal edema 4. Facial puffiness 5. Altered sensorium 6. Bleeding manifestations

PAST HISTORY Jaundice / Type-2 Diabetes Mellitus / Tuberculosis / Bronchial Asthma / IHD / Seizure Disorder.

FAMILY HISTORY Jaundice / Any Liver Disease / Type-2 Diabetes Mellitus / Ischemic Heart Disease DRUG HISTORY:

PERSONAL HISTORY Diet: Appetite: Sleep: Bladder: Bowel habits: Habits: ¬ Smoking : ¬ Alcohol : 1. Duration 2. Quantity 3. Type ¬ Tobacco chewing :

GENERAL P HYSICAL EXAMINATION:

Height:

Weight:

BMI :

Abdominal girth :

Pallor :

Icterus :

Clubbing :

Cyanosis :

Lymphadenopathy:

Edema :

Signs of liver cell failure: Spider naevi / Flapping tremor / Gynaecomastia (in Males) / Parotid enlargement / Sparse pubic and axillary Hair / Testicular atrophy / Palmar erythema.

Pulse rate : bpm Blood Pressure : mmhg Respiratory Rate : cpm Temperature : CVS: RS: CNS: PER ABDOMEN: Inspection: Palpation: 1. Organomegaly: 2. Fluid thrill: Percussion: Shifting dullness: present / absent Auscultation:

INVESTIGATIONS: Hemoglobin g/dl ESR mm/1st hr end Total count cells/cumm Platelets lakhs/cumm Blood ureamg/dl Serum creatinine mg/dl Total bilirubin mg/dl Direct bilirubin mg/dl Total Protein g/dl Serum Albumin g/dl Serum glutamic oxaloacetic transaminase U/l SGPT U/l ALP U/l RBS mg/dl GGT U/l

VIRAL MARKERS: HIV,HBsAg,HCV ULTRASOUND ABDOMEN: ENDOSCOPY: 1. ESOPHAGEAL VARICES 2. GASTRIC VARICES 3. PORTAL GASTROPATHY 4. OTHERS

ETHICAL CLEARANCE LETTER

SDM
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SHRI DHARMASTHALA MANJUNATHESHWARA UNIVERSITY

A Constituent Unit of **Shri Dharmasthala Manjunatheshwara University**
Recognised by Medical Council of India, New Delhi

Ref: SDMCMS&H/ IEC : 21:2021

Date : 19-02-2021

SUB: - Institutional Ethics Committee permission.

Permission is granted to the following postgraduate student to carry out the dissertation study :

Name of Postgraduate student:	Dr. MADHU S PATIL
Title of the dissertation study:	ETIOLOGY, CLINICAL PROFILE, UPPER GASTROINTESTINAL ENDOSCOPIC FINDINGS AND OUTCOME OF PATIENTS PRESENTING WITH LIVER CIRRHOSIS WITH PORTAL HYPERTENSION.

Dr.Deepak Kanabur
Member Secretary-SDMIEC

Dr.Sujata Giriyan
Chairperson-SDMIEC

Dr. Sujata S. Giriyan
Chairman
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Certificate of Plagiarism Check

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This is to certify that **Dr Madhu S Patil**, postgraduate student in the subject of **General Medicine** under the guidance of **Dr Hemamalini G**, **Professor,** Department of **General Medicine** has submitted dissertation entitled **Etiology, clinical profile, upper gastrointestinal endoscopic findings and outcome of patients presenting with liver cirrhosis with portal hypertension** for plagiarism check as per the requirement of Shri Dharmasthala Manjunatheshwara University, Dharwad. The UGC service of OURIGINAL plagiarism detection software licensed to Shri Dharmasthala Manjunatheshwara University, Dharwad has returned a similarity of **3%.**

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1.	**Signature of the candidate**	Dr. MADHU. S. PATIL
2.	**Remarks of the guide**	Good feasable study.
3.	**12.1 Guide: Name & Designation**	DR HEMAMALINI GURURAJ PROFESSOR, DEPARTMENT OF GENERAL MEDICINE, SDM COLLEGE OF MEDICAL SCIENCES AND HOSPITAL, DHARWAD.
	Signature	
	12.2 Co-Guide: Name & Designation	DR PREETHAM HURKADLI PROFESSOR, DEPARTMENT OF GASTROENTEROLOGY, SDM COLLEGE OF MEDICAL SCIENCES AND HOSPITAL, DHARWAD
	Signature	
	12.3 Head of the Department	DR. KIRAN AITHAL. PROFESSOR AND HEAD OF DEPARTMENT. MEDICINE. SDM COLLEGE OF MEDICAL SCIENCES AND HOSPITAL. SATTUR, DHARWAD
	12.6 Signature	Dr. KIRAN AITHAL Professor & Head of the Department Medicine DM College of Medical Sciences & Hospital Sattur, Dharwad-580 009
4.	**13.1 Remarks of the Principal**	Recommended
	13.2 Signature	Dr RATHNAMALA DESAI KMC. Reg. No. 19027 SDMCMS & H, Dharwad

Masterchart

NAME	AGE (years)	UHID	SEX	PULSE (beats/min)	SBP (mmHg)	DBP(mmHg)	HEMOGLOBIN(gm/dl)	TLC(/cumm)	platelets(/cumm)	PT	INR	Sr. UREA(mg/dl)	Sr CREAT (mg/dl)	T PROTEIN (mg/dl)	ALBUMIN (mg/dl)	T Bilirubin (mg/dl)	AST (IU/L) ALT(IU/L)		ALP (IU/L)	PAIN ABDOMEN	DURATION	JAUNDICE	ABDOMINAL DISTENSION	PEDAL EDEMA	FEVER	ATERED SENSORIUM	BLEEDING MANIFESTATIONS	UGI-NORMAL	GRADE 1 V	GRADE2 V
RAMANNA H BAGANAL	60	1336818	MALE	90	110	80	11.1	15740	6.5	17.9	1.48	21	0.84	6.8	1.97	1.91	81	59	289	NO	4	NO	YES	YES	NO	NO	NO	NO	NO	NO
YALLAPPA J GADDADAVAR	81	1328657	MALE	78	130	80	8.3	15950	1.37	18.7	1.57	25	0.62	5.63	1.55	0.83	17	15	40	NO	3	NO	YES	YES	YES	NO	YES	NO	NO	NO
MOHIT J KYALAKONDA	21	989326	MALE	74	140	70	12.4	5310	0.25	21	1.8	24	0.72	7	2	1.2	36	28	90	YES	6	NO	NO	NO	YES	NO	YES	NO	NO	YES
GEETA MENEDAL	34	1016967	FEMALE	80	100	70	8.8	4350	1.12	14.1	1.13	30	1.2	6	3.02	0.87	21	25	44	YES	3	NO	NO	YES	NO	NO	NO	NO	YES	NO
MANJAPPA SHAGOTI	45	1383396	MALE	110	90	60	10.2	15990	1.76	16.3	1.15	60	1.61	5.2	1.41	6.55	125	58	157	NO	11	YES	YES	YES	NO	YES	YES	NO	NO	YES
NABISAHEB TASHILDAR	74	1377938	FEMALE	62	110	70	7.9	4890	1.65	31.6	2.79	82	2.64	6	2.1	1.04	42	22	109	NO	6	NO	YES	YES	NO	NO	NO	NO	NO	YES
VIJAY LAKKASAKOPPA	35	1379905	MALE	96	86	60	7.1	3760	0.82	29.9	2.63	38	1.23	5.26	1.6	2.82	47	30	176	NO	7	YES	NO	YES	YES	NO	NO	NO	NLO	YES
VINAYKUMAR S AKKASALIGAR	33	1376193	MALE	100	90	60	6.7	19,900	0.9	25.4	2.2	25	1.02	5.9	2.53	24.47	127	62	107	NO	25	YES	YES	YES	YES	YES	NO	NO	YES	NO
DEVAKKA	58	1117284	FEMALE	110	130	60	8.2	14320	1.38	16.7	1.34	52	1.06	5.8	2.5	1.27	23	18	63	NO	8	YES	NO	NO	YES	NO	YES	NO	NO	NO
USHA GUNJIKAR	66	1080459	FEMALE	100	110	70	7.2	3880	1.11	18	1.9	90	2	7	3.2	0.8	40	37	108	NO	4	NO	NO	NO	NO	NO	YES	NO	NO	NO
BELLAD SOMASHEKAR	66	1307465	MALE	104	120	80	8.2	9020	1.43	18	2.6	58	2.08	8	2.5	1.67	30	18	151	NO	9	NO	NO	NO	YES	YES	NO	NO	NO	NO
UDAYKUMAR BALAKATTI	56	1080071	MALE	70	96	50	8.6	5500	0.98	18	1.8	29	1.13	7	1.9	1.2	40	36	80	NO	5	NO	YES	YES	NO	NO	YES	NO	YES	YES
SUNIL MUTALIKDESAI	46	1306547	MALE	68	112	76	7.5	4120	0.58	22.7	1.9	55	2.48	6.7	1.7	2.3	89	25	74	NO	7	YES	YES	YES	NO	NO	NO	NO	YES	NO
GIRIJA RAMU	70	1145187	FEMALE	110	100	50	6.6	4830	0.88	15.5	1.26	90	3.05	5.4	1.7	1	26	30	80	NO	8	NO	YES	YES	NO	NO	NO	NO	NO	YES
RAMESH IRAPPA S	39	1331278	MALE	88	110	80	9.4	8370	1.2	18	1.6	40	0.38	5.8	2	7.6	116	48	208	NO	3	YES	YES	YES	NO	NO	NO	NO	YES	NO
BALAPPA	39	627771	MALE	120	130	88	6.5	10870	0.69	20	1.8	34	1.03	5.6	2.5	9.5	117	35	69	NO	3	YES	YES	YES	NO	YES	NO	NO	NO	NO
RAMESH H K	47	631722	MALE	84	100	60	10.4	3040	0.67	21	2.1	18	0.6	5.4	2.09	2.46	56	28	228	NO	6	YES	YES	NO	NO	NO	NO	NO	NO	YES
BASAVARAJ BELAGALI	43	720128	MALE	76	120	80	13.8	6820	1.8	19.1	1.57	22	0.68	6.7	3.56	1.84	43	31	63	NO	8	YES	YES	YES	NO	YES	NO	YES	NO	NO
HUSAINSAB H	48	945482	MALE	98	90	60	8.6	4490	0.63	25	2.14	9	0.8	6.3	2.04	1.9	88	40	80	NO	8	YES	NO	YES	YES	NO	NO	YES	NO	NO
SUMANGALA PATIL	70	971141	FEMALE	92	102	70	10	8680	2	23	1.8	28	0.8	6.2	2.2	2.8	42	40	120	NO	10	YES	YES	YES	NO	NO	NO	NO	NO	NO
SHIVAPUTHRAPPA S	55	993423	MALE	110	90	60	9	10800	0.78	24	2.04	26	1.2	6	1.8	2	90	26	135	YES	5	YES	YES	YES	NO	NO	NO	NO	YES	NO
RANGARAO KUCHIPUDI	58	1003684	MALE	88	80	60	5.2	9430	0.38	24	2.5	80	2.3	5.5	1.86	2.7	106	40	108	NO	7	YES	YES	YES	YES	YES	NO	NO	YES	YES
GANGAMMA H	83	1017991	FEMALE	70	116	78	8.8	10360	0.97	28	2	40	0.63	7	2.9	22	450	246	97	YES	6	YES	NO	NO	NO	NO	YES	NO	NO	YES
SAVANNA H	65	1147329	MALE	88	130	80	9	3220	0.85	16	1.3	60	1.8	7	2.2	2.5	66	68	134	NO	3	YES	YES	YES	NO	YES	NO	NO	NO	YES
LAKSHMAVVA	44	1167286	FEMALE	101	90	60	4.1	20050	1.5	26.7	2.3	66	2.7	5.7	1.3	2.8	165	90	90	NO	10	YES	NO	NO	YES	NO	YES	NO	NO	NO
DEEPAK	30	1216124	MALE	116	108	60	9	9920	0.57	22.1	1.7	20	0.9	6	1.3	4.5	65	42	77	YES	30	YES	YES	YES	NO	YES	YES	NO	YES	YES
SADANANDH	40	725589	MALE	54	96	50	8.4	13630	0.87	31.9	2.8	30	1.2	6	1.4	6.1	51	21	139	NO	28	YES	YES	YES	NO	YES	YES	NO	YES	NO
SUBHASH	46	411392	MALE	105	76	48	9.3	5050	0.48	28	1.2	40	4.4	5.9	1.7	5.5	130	78	55	NO	3	YES	NO	NO	NO	NO	NO	YES	NO	NO
CHOWDAPPA	41	399577	MALE	104	106	76	11.3	10590	0.93	14	1.3	87	0.9	5.8	2	7.2	84	32	243	NO	8	YES	YES	YES	NO	NO	NO	NO	NO	YES
SHRISHAIL	30	441593	MALE	70	120	70	8	12620	1.4	17	2.5	48	2.8	6	2.2	3	112	88	84	NO	5	YES	NO	NO	NO	NO	NO	YES	NO	NO
PRAKASH	41	451429	MALE	130	100	60	7	18270	1.5	28	1.7	49	1.8	6	1.7	10	110	90	210	YES	8	YES	NO	YES	NO	NO	YES	NO	YES	NO
NAGARAJ	51	389416	MALE	70	110	70	11	10480	0.72	32	2.3	30	0.72	6.6	1.8	19	157	37	137	YES	15	YES	YES	YES	YES	NO	NO	NO	YES	NO
VEERANGOWDA	43	239932	MALE	106	100	80	5.2	2990	0.5	16	2.6	36	0.9	7	2.4	4	80	46	70	NO	5	YES	YES	YES	NO	NO	NO	NO	YES	YES
GURUPADAPPA	74	1195029	MALE	76	110	70	8.9	8860	1.1	19	1.1	60	2.51	7	2	1.9	54	38	272	YES	6	YES	YES	YES	NO	NO	NO	NO	NO	NO
SHARANAMM	78	1330023	FEMALE	90	140	90	11.1	8630	2	22	1.5	30	1.56	6	2.3	0.8	28	20	88	NO	3	NO	NO	YES	YES	NO	NO	YES	NO	NO
PARAPPA	60	1330179	MALE	80	100	70	5.4	8030	1.7	19.7	1.67	20	0.8	7	2.3	2.09	120	45	126	NO	10	YES	NO	YES	NO	NO	NO	NO	NO	NO
NINGAPPA	40	1330192	MALE	68	90	60	8.3	8480	0.7	25.2	2.16	26	0.69	7	2.2	16.6	98	60	146	YES	8	YES	YES	YES	NO	YES	YES	NO	NO	NO

YALAMMA	55	1327536	FEMALE	110	110	70	9.5	8060	0.41	15	1.3	37	0.6	6.3	2.1	1.6	58	30	30	NO	2	NO	NO	NO	NO	NO	YES	NO	NO	YES
RAMESH SHINDE	46	1330654	MALE	82	140	90	9.8	9250	0.84	18	1.5	79	2	4.8	1.7	0.9	110	38	126	NO	11	NO	YES	YES	NO	NO	NO	NO	NO	NO
VOILET PETERS	81	1332526	FEMALE	100	120	80	10.2	7740	2.1	25	2.1	40	0.9	5.1	1.16	0.8	88	70	106	YES	2	NO	NO	NO	NO	NO	NO	NO	NO	NO
FAKIRAPPA	67	1334480	MALE	76	98	70	7.7	8940	1.02	27.3	7.8	80	1.9	6	1.7	0.54	26	29	96	NO	7	NO	NO	YES	YES	NO	NO	NO	YES	YES
SHIVAKKA	75	1334568	FEMALE	90	130	80	7.2	9840	0.9	16	2	60	0.5	7.8	3	0.8	32	37	188	NO	3	NO	NO	NO	YES	NO	NO	YES	NO	NO
SHIVANAND	47	1308037	MALE	70	116	70	7.8	12350	1.4	19	1.7	72	0.8	6	1.1	3	50	28	182	NO	7	YES	NO	YES	NO	NO	NO	NO	NO	NO
BALAPPA O	55	1296215	MALE	80	106	60	8.8	14000	1.02	23	1.8	79	2.02	4.3	1.5	2.2	113	102	77	YES	5	YES	YES	NO	NO	YES	YES	NO	NO	YES
ABDULGANI	58	1216625	MALE	74	90	60	9.8	11070	0.78	21	1.5	56	1.9	6	1.4	9.4	177	68	114	NO	3	YES	YES	YES	YES	YES	NO	NO	NO	NO
NOORJANBI	59	410742	FEMALE	80	140	90	11.6	6100	2.07	18.4	2.2	22	0.97	7.5	3.5	0.7	46	43	160	NO	6	NO	NO	YES	NO	NO	YES	NO	YES	YES
SUNBHASH	46	411392	MALE	74	70	48	9.3	15050	0.48	15.6	1	39	3.3	5.9	1.6	5	130	73	55	YES	3	YES	NO	NO	YES	YES	NO	NO	NO	NO
UMESH	49	1280351	MALE	90	130	80	7.5	7500	2.1	17.9	1.1	14	0.6	7.2	3	2.09	42	38	174	YES	5	YES	YES	YES	NO	NO	YES	NO	YES	YES
DASHARATHAPPA	74	1336130	MALE	98	126	60	9.1	5010	1.1	20.1	2.1	41	1.3	6	2.4	5.8	107	74	234	NO	10	YES	YES	YES	NO	YES	YES	NO	NO	NO
DASTAGIR B	54	1292000	MALE	70	120	70	8	8090	2.04	18	1.3	71	5.29	6.3	2.03	0.8	66	62	194	NO	17	NO	YES	YES	NO	NO	NO	YES	NO	NO
ASHOK K	66	397067	MALE	82	138	80	8.5	13870	1	23	2.1	30	1.76	7	1.88	1.09	35	22	94	YES	8	NO	NO	NO	NO	NO	NO	NO	NO	YES
SHARANABASAPPA	52	1339442	MALE	80	112	60	8.3	12220	0.8	17.4	2.3	60	1.4	6.5	1.3	11	86	21	269	NO	6	YES	YES	YES	NO	YES	NO	NO	NO	NO
RAMESH IRAPPA S	38	1331278	MALE	95	90	50	5.8	5340	1.3	19	1.2	30	0.53	5.8	2.29	3.04	118	43	70	NO	6	YES	YES	YES	NO	NO	NO	NO	YES	NO
SUVARNA	93	1061546	FEMALE	80	102	70	7.2	14360	1.5	23	2.2	26	1.2	5.6	1.7	0.6	51	29	153	NO	5	NO	YES	YES	NO	NO	NO	NO	YES	NO
NANDISH KUMAR	50	839056	MALE	60	120	50	6.1	6360	1.1	21.3	2.1	60	1.83	6.8	1.5	7.15	137	44	127	NO	7	YES	YES	YES	YES	YES	NO	NO	NO	YES
SOUMYA	21	496335	FEMALE	70	112	60	8	7430	3.4	26	1.8	13	0.5	7	4	1.3	17	18	58	YES	4	YES	NO	NO	NO	NO	NO	NO	NO	NO
MOULA	81	1335635	MALE	80	86	40	8.7	13130	1.5	23.4	1.7	48	2.1	5.7	1.8	2.9	83	44	235	YES	4	NO	YES	YES	NO	YES	NO	NO	NO	NO
RAVIKUMAR	48	1089863	MALE	88	110	70	8.9	9540	0.81	22	0.9	31	1.5	4.7	1.7	8.1	173	78	181	NO	11	YES	YES	YES	YES	NO	NO	NO	NO	YES
SAMBAJI	61	1221030	MALE	70	100	70	10	7240	0.9	15	1.1	33	1.2	5.9	2.1	2.7	49	27	117	NO	3	YES	YES	NO	NO	NO	NO	NO	YES	NO
SHIVANAND	47	1308037	MALE	70	106	70	6.9	6060	0.9	19.2	1.5	36	1	5.4	2.5	8.7	33	20	144	YES	8	YES	NO	NO	NO	NO	NO	NO	NO	YES
SANJEEV V	44	1311764	MALE	116	130	90	8.6	9940	0.61	28	1.7	35	0.8	7.3	1.8	7.7	115	48	247	NO	5	YES	YES	YES	NO	NO	NO	NO	NO	YES
NAGESH V B	58	1314140	MALE	80	130	80	12.4	8970	2.72	18.1	1.5	23	0.73	7.3	2.3	1.8	337	113	768	YES	6	YES	NO	NO	NO	NO	NO	NO	NO	NO
GONIBASASAPPA	48	1314578	MALE	70	110	70	9.2	5720	0.92	23	2.01	30	0.8	5.9	1.65	2.9	56	25	50	NO	6	YES	YES	YES	NO	NO	NO	YES	NO	NO
SATISH TUKARAM	24	1325116	MALE	70	110	70	3.8	5630	1.8	23.1	2.1	33	0.85	5.7	2.3	0.31	30	50	39	NO	1	NO	YES	YES	NO	YES	YES	NO	NO	NO
UMESH S	61	1291968	MALE	76	120	70	8.7	6520	1.27	25.7	2.4	60	1.32	6.9	2.9	1.2	98	68	112	YES	4	NO	YES	YES	NO	NO	NO	YES	NO	NO
PAVITRA B	30	1338841	FEMALE	80	120	70	12	7950	2.35	18	2.3	20	0.75	7	3	0.5	16	21	97	NO	3	NO	NO	NO	NO	NO	YES	NO	NO	YES
RAGHUNATH	61	1214111	MALE	71	122	74	9.9	7340	1.78	19	1.4	25	1.2	7.6	3.7	0.2	33	37	53	YES	3	NO	NO	NO	NO	NO	NO	NO	NO	NO
RAMESH K	43	1334157	MALE	86	120	80	9.2	3230	0.96	23.2	5.3	30	0.7	7.3	2.7	0.69	69	51	86	NO	8	NO	YES	YES	NO	YES	NO	NO	YES	YES
IRAPPA	62	840638	MALE	80	100	70	13	3860	1.3	21.9	4.2	37	0.87	6.5	2.9	0.9	77	42	49	NO	11	NO	NO	YES	NO	NO	NO	NO	NO	NO
RAVINDRA	69	1288239	MALE	80	130	80	10.9	6090	1.2	21.09	2.8	22	1.06	5.9	2.1	1.9	77	55	100	NO	6	YES	YES	NO	NO	NO	NO	YES	NO	NO
HULAGESH	46	1234471	MALE	90	90	60	11	11360	0.8	22.5	1.9	41	1.8	8.1	1.8	2.7	50	37	153	NO	4	YES	YES	YES	YES	NO	NO	NO	NO	YES
SUBHASH	56	1245218	MALE	100	126	90	8	4090	0.72	19	1.61	55	2.2	5.1	2.1	2.6	66	51	190	NO	7	YES	YES	YES	NO	YES	YES	NO	NO	NO
MALLIKARJUN	50	1249662	MALE	80	120	80	6.5	6880	2	24	1.7	30.8	1.6	6.3	3.3	5.4	68	44	105	NO	3	YES	YES	YES	YES	YES	NO	NO	NO	NO
KALAPPA	54	1285780	MALE	70	116	72	8.2	6100	0.96	22.8	1.9	38	0.83	6	1.8	3.8	42	28	119	YES	3	YES	YES	YES	NO	NO	NO	NO	NO	NO
UMESH K	49	1280351	MALE	90	96	60	7.5	9840	2.1	17	1.2	42	0.97	7.2	3.01	2.09	42	38	170	NO	4	YES	NO	NO	NO	NO	YES	NO	NO	YES
TIPPANNA F H	50	451429	MALE	84	110	70	8.7	7510	1.22	19.7	1.3	15	0.74	6.9	1.45	3.01	57	12	163	NO	23	YES	YES	YES	NO	YES	NO	NO	YES	NO
HARESH V JOSHI	36	511479	MALE	98	100	60	11.1	27230	3.6	19.7	1.4	96	4.2	3.2	1.5	26.8	131	23	131	NO	15	YES	YES	YES	YES	YES	YES	NO	YES	NO
MARUTI H	56	1373735	MALE	80	116	78	8.6	4700	1.35	28	1.5	101	1.86	6.8	2.08	2.7	37	26	86	YES	5	YES	YES	NO	NO	NO	NO	NO	NO	YES
VINAYKUMAR P V	30	1373474	MALE	114	102	56	7.4	28080	3.05	27.4	1.6	58	2.39	4.9	1.92	24.8	52	13	127	NO	12	YES	NO	NO	NO	NO	NO	NO	NO	NO
K SRINIVAS BABU	45	1315093	MALE	68	112	60	10.8	9400	0.95	15.7	1.12	24	1.06	6.8	2.2	5.01	150	68	46	NO	12	YES	YES	NO	YES	YES	NO	YES	NO	NO
SUDHENDRA S J	65	1328581	MALE	105	140	84	10	18360	1.9	12.6	1	28	0.8	7.2	2.4	0.5	79	36	212	NO	6	NO	NO	YES	YES	NO	NO	NO	NO	YES
AMBAKKA K	71	1032902	FEMALE	72	98	68	8	6810	2.5	23.1	2.7	132	3.7	5	2.3	1.7	46	30	53	NO	3	NO	NO	NO	NO	NO	YES	NO	NO	NO
FAKIRAPPA N	41	1077727	MALE	96	104	80	7.4	12068	1.3	25.2	2.5	40	1.02	5.9	2.58	10	46	36	73	YES	5	YES	NO	NO	NO	NO	NO	NO	YES	NO
VASANTH CHANDRAYYA	37	1323549	MALE	80	80	58	6.8	9860	2	43.4	3.4	22	0.65	7.3	1.6	15.24	130	28	186	NO	20	YES	YES	YES	NO	NO	YES	NO	NO	YES
RAVEENDRA V	57	1320340	MALE	77	130	80	10.7	11030	0.76	21.4	1.8	64	4.8	5	1.6	34.7	68	56	152	NO	17	YES	YES	YES	NO	YES	NO	NO	YES	NO
BASAVARAJ SALI	41	1376083	MALE	105	90	60	7.9	10090	0.56	38.6	3.7	37	3.1	6.3	1.16	5.09	80	35	104	YES	2	YES	NO	NO	YES	NO	NO	NO	YES	NO
LAKSHMIKATH M	44	944644	MALE	130	100	80	6.4	7080	0.98	38	3.4	30	0.78	5.6	2.75	0.93	25	24	58	NO	4	NO	NO	NO	NO	NO	YES	NO	YES	NO

G PRAKASH	49	1327601	MALE	102	130	80	13.6	9130	1.5	29.2	2.5	30	1.4	8	1.7	8.3	494	349	263	NO	5	YES	YES	YES	YES	YES	YES	NO	YES	NO
NINGAPPA METI	36	1325615	MALE	76	100	66	9	6624	1.08	36	3.2	13	1.15	6.8	1.55	2.4	144	62	147	NO	2	YES	YES	YES	NO	NO	NO	NO	NO	YES
BISTARA B	65	1326610	MALE	62	110	70	10.1	3860	1.06	20	1.67	9	0.6	7.2	2.8	15.5	74	57	240	NO	20	NO	YES	NO	NO	NO	NO	NO	NO	NO
SAGAR.M.P	36	1374068	MALE	110	90	60	5.8	11080	0.9	28	2.4	28	1.2	5.8	1.14	16	154	77	107	YES	5	YES	NO	NO	NO	NO	NO	NO	NO	NO
CHANDRU S K	32	1329420	MALE	96	100	70	7.9	11570	2.18	32.4	2.85	9	0.68	5.7	1.04	10.8	95	30	147	YES	18	YES	YES	YES	NO	NO	NO	YES	NO	NO
SHIVAPPA B S	68	1199868	MALE	120	130	88	9.4	9120	1.2	26	2.67	102	4.2	6.9	2.7	0.82	17	24	54	NO	3	NO	NO	NO	NO	NO	NO	YES	YES	NO	NO
BALAPPA YAMUNAPPA	55	1296215	MALE	112	110	78	7.3	6820	0.43	18	1.3	53	1.32	5.1	2	0.89	74	52	53	NO	4	NO	NO	NO	NO	NO	NO	YES	NO	NO	YES
RAMESH T G	32	1304061	MALE	102	112	78	6.9	20360	1.29	71.5	6.8	132	2.8	6.8	1.78	11.5	95	91	291	NO	4	YES	NO	NO	NO	NO	NO	YES	NO	YES	YES
HANUMAGOUGA C K	40	1304269	MALE	82	130	80	10.1	12660	3.19	33.1	2.91	60	1.8	6.4	2.12	7.04	98	17	155	YES	4	YES	YES	NO	NO	YES	NO	NO	NO	NO
YALLAPPA N SULLAD	49	1304605	MALE	102	126	74	6.6	3050	0.39	26.5	2.28	41	1.06	7	2.2	1.4	58	40	86	YES	19	NO	YES	NO	NO	NO	NO	NO	YES	NO
RAJAN K	48	1307009	MALE	112	120	60	4.3	10190	1.4	29	2.52	15	0.93	6.1	1.7	3.51	34	26	165	NO	7	NO	YES	YES	NO	YES	YES	NO	NO	NO
BASAVARAJ S HOSAMANI	41	1244764	MALE	76	98	58	7.8	9760	0.6	31	2.1	27	1.07	5.7	1.5	4.9	208	90	102	NO	5	NO	YES	NO	NO	YES	NO	NO	NO	NO
INDIRAMMA MIRJI	66	1332124	FEMALE	94	102	96	10.2	3840	0.71	15	1.2	30	1.7	5.2	1.4	1.2	56	58	102	YES	4	NO	NO	NO	YES	NO	NO	YES	NO	NO
NAME	AGE (	UHID	SEX	(beat	SBP (	DBP (	HEMO	TLC(/cur	platele	PT	INR	Sr. UR	Sr CR	T PRO	ALBUN	T Bilirul	AST (IU/L) A		ALP (IN	ABDON	DURA	JAUNI	ABDO	PEDAL	FEVER	ATERE	BLEED	UGI-N	GRADE	GRADI

GRADE3 V	GASTRIC VARICES	PHG	EROSIVE GASTRITIS	HIATUS HERNIA	DUODENITIS	MORTALITY CPS		ETIOLOGY	ENDOSCOPIC PROCEDURE	CAUSE FORMORTALITY
NO	NO	NO	YES	NO	NO	NO	C	HBsAG	NO	NO
YES	NO	YES	NO	NO	YES	NO	B	ETHANOL	NO	NO
YES	NO	NO	YES	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	NO	C	WILSONS DISEASE	EVL	NO
NO	NO	YES	NO	NO	NO	YES	C	ETHANOL	NO	SEPTIC SHOCK -SBP
YES	GOV TYPE2	YES	NO	NO	NO	NO	B	ETHANOL	EVL	NO
NO	NO	YES	YES	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	YES	B	ETHANOL	NO	SEPSIS WITH SEPTIC SHOCK-HEPATIC ENCEPHALOPATHY.
YES	NO	YES	NO	NO	NO	NO	C	NASH	EVL	NO
YES	NO	YES	NO	YES	NO	NO	B	HBsAG	EVL	NO
NO	NO	NO	YES	YES	YES	YES	B	ETHANOL	NO	SEPTIC SHOCK, COAGULOPATHY
NO	NO	NO	NO	NO	NO	NO	C	ETHANOL	EVL	NO
NO	YES	YES	NO	NO	YES	NO	C	ETHANOL	NO	NO
YES	NO	YES	NO	NO	NO	NO	C	NASH	EVL	NO
NO	NO	YES	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	NO	YES	NO	YES	YES	B	ETHANOL	NO	HYPOVOLEMIC SHOCK,DCLD,AKI
NO	NO	YES	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	NO	YES	NO	YES	YES	B	ETHANOL	NO	HYPOVOLEMIC SHOCK,DCLD,AKI
NO	NO	NO	NO	NO	NO	NO	A	ETHANOL	NO	NO
NO	NO	NO	NO	NO	NO	NO	C	HBsAG	NO	NO
NO	NO	NO	NO	NO	NO	NO	B	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	YES	C	HBsAG	NO	CARDIOGENIC SHOCK,SEPSIS,PNEUMONIA,DCLD
NO	NO	YES	NO	NO	NO	NO	C	ETHANOL	NO	DAMA
NO	NO	YES	NO	NO	YES	NO	C	ETHANOL	NO	NO
YES	NO	YES	NO	NO	YES	NO	C	NASH	EVL	NO
YES	YES	NO	NO	NO	NO	NO	B	HCV	NO	DAMA
YES	NO	YES	NO	NO	NO	NO	B	ETHANOL	EVL	NO
NO	NO	YES	NO	NO	NO	YES	C	ETHANOL	EVL	MASSIVE HEMOPTYSIS, CARDIOGENIC SHOCK
NO	YES	YES	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	NO	NO	NO	NO	NO	B	ETHANOL	NO	NO
NO	YES	YES	NO	NO	NO	NO	C	ETHANOL	NO	DAMA
NO	NO	NO	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	YES	NO	YES	NO	NO	C	ETHANOL	NO	NO
NO	NO	NO	YES	NO	NO	YES	C	ETHANOL	NO	SEPSIS WITH SEPTIC SHOCK-BRONCHOPNEUMONIA.
NO	NO	YES	NO	NO	NO	NO	B	ETHANOL	NO	NO
NO	NO	YES	YES	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	NO	NO	NO	YES	NO	A	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	NO	B	ETHANOL	NO	NO
YES	YES	YES	NO	NO	NO	NO	C	ETHANOL	NO	NO

YES	NO	YES	YES	NO	NO	NO	A	HCV	EVL	DAMA
NO	NO	NO	YES	NO	NO	NO	B	ETHANOL	NO	NO
NO	NO	YES	NO	YES	NO	NO	C	ETHANOL	NO	NO
YES	NO	YES	NO	NO	NO	NO	B	ETHANOL	NO	NO
NO	NO	NO	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	YES	C	ETHANOL	NO	SEPSIS WITH SEPTIC SHOCK-MODS,HEPATIC ENCEPHALOPATHY
NO	NO	YES	YES	NO	NO	NO	C	ETHANOL	EVL	NO
NO	NO	YES	NO	NO	NO	YES	C	ETHANOL	NO	REFRACTORY SHOCK,UPPER GI BLEED,DCLD
NO	NO	NO	YES	NO	NO	NO	C	NASH	NO	NO
NO	NO	YES	NO	NO	NO	NO	C	NASH	NO	NO
NO	YES	YES	YES	NO	NO	NO	C	ETHANOL	EVL	NO
YES	YES	YES	NO	NO	NO	NO	C	ETHANOL	EVL	NO
NO	NO	NO	NO	NO	NO	NO	C	NASH	NO	NO
YES	NO	YES	NO	NO	NO	YES	B	ETHANOL	EVL	SEPSIS-BIBASAL PNEUMONIA,DCLD,LEFT LOWERLIMB CELLULITIS
NO	NO	NO	YES	NO	YES	NO	C	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	NO	A	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	YES	C	ETHANOL	NO	UPPER GI BLEED, AKI,DCLD
NO	NO	NO	NO	NO	NO	NO	C	ETHANOL	EVL	NO
NO	NO	YES	NO	NO	NO	NO	B	ETHANOL	NO	DAMA
NO	NO	YES	YES	NO	YES	NO	C	ETHANOL	NO	NO
YES	NO	YES	NO	NO	NO	NO	B	ETHANOL	EVL	NO
NO	NO	YES	NO	NO	NO	NO	B	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	NO	A	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	YES	C	ETHANOL	NO	SEPTIC SHOCK,SEVERE METABOLIC ACIDOSIS,AKI,COAGULOPATHY,DCLD
NO	NO	NO	YES	NO	NO	NO	B	ETHANOL + HBsAG	NO	NO
NO	NO	NO	NO	NO	NO	NO	C	ETHANOL	NO	NO
YES	NO	YES	NO	NO	NO	NO	B	ETHANOL	EVL	NO
NO	NO	NO	NO	NO	NO	NO	C	ETHANOL	NO	NO
YES	NO	YES	NO	NO	NO	NO	B	ETHANOL	EVL	NO
NO	NO	NO	NO	NO	NO	NO	A	ETHANOL	NO	DAMA
NO	NO	YES	YES	NO	YES	NO	B	ETHANOL	NO	NO
NO	NO	NO	YES	NO	NO	NO	C	HCV	NO	NO
NO	NO	YES	NO	NO	NO	NO	A	ETHANOL	NO	NO
NO	NO	NO	YES	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	NO	NO	NO	NO	YES	B	ETHANOL	NO	SEPSIS WITH MODS, DCLD
NO	NO	YES	NO	NO	NO	YES	C	HBsAG+ETHANOL	NO	SEPSIS WITH SEPTIC SHOCK, UGI BLEED, PNEUMONIA, METABOLIC WITH HEPATIC ENCEPHALOPATHY
YES	NO	YES	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	YES	YES	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	NO	NO	NO	YES	NO	C	ETHANOL	NO	NO
NO	NO	YES	NO	YES	NO	NO	C	ETHANOL	EVL	NO
NO	YES	YES	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	YES	NO	NO	YES	NO	B	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	NO	C	ETHANOL	NO	DAMA
NO	YES	YES	YES	NO	NO	YES	A	HCV	NO	DCLD, SEVERE METABOLIC ACIDOSIS,HRS
NO	NO	NO	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	NO	B	NASH	NO	NO
YES	IGV-1	YES	NO	NO	NO	YES	B	CRYPTOGENIC	NO	SEPSIS WITH MODS-DCLD,ACUTE ON CKD, T2DM,HTN
NO	NO	NO	YES	NO	NO	NO	B	ETHANOL	NO	NO
NO	NO	NO	NO	NO	YES	NO	C	ETHANOL	EVL	DAMA
NO	NO	YES	NO	NO	YES	NO	B	ETHANOL	NO	NO
NO	NO	YES	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	GOV2	NO	NO	NO	NO	YES	C	ETHANOL	ENDC	HEMORRHAGIC SHOCK-GASTRIV VARICEAL BLEEDING,DCLD

NO	NO	YES	NO	NO	NO	NO	A	AUTOIMMUNE	NO	NO
NO	NO	YES	NO	NO	NO	NO	C	ETHANOL	NO	NO
YES	NO	NO	NO	NO	NO	NO	C	ETHANOL	EVL	NO
NO	GOV2	YES	NO	NO	NO	YES	C	ETHANOL	NO	SEPSIS WITH SEPTIC SHOCK-SBP,GRADE 4 HEPATIC ENCEPHALOPATHY,GRADE 4 ESOPHAGEAL VARICES
NO	NO	NO	NO	NO	NO	NO	C	ETHANOL	NO	NO
NO	NO	NO	NO	NO	NO	NO	C	NASH	NO	NO
NO	NO	NO	NO	YES	NO	NO	B	HCV	NO	NO
NO	IGV1	YES	NO	NO	NO	NO	C	ETHANOL	EVL	NO
NO	NO	NO	YES	NO	NO	NO	B	ETHANOL	NO	NO
YES	NO	YES	NO	NO	NO	YES	C	ETHANOL	EVL	HEMORRHAGIC SHOK,SEVEREVE LACTIC AND METABOLIC ACIDOSIS,DCLD.
YES	NO	YES	NO	NO	NO	NO	C	ETHANOL	EVL	NO
YES	NO	YES	NO	NO	NO	NO	C	WILSONS DISEASE	NO	NO
NO	NO	NO	NO	NO	NO	NO	A	HBsAG	NO	DAMA
GRADE	GASTRIC VAR	PHG	EROSI	HIATU	DUOD	MORTALI		ETIOLOGY		