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RATIO OF PLATELET COUNT TO SPLENIC DIAMETER AS A PREDICTOR OF ESOPHAGEAL VARICES IN CIRRHOSIS OF LIVER - A CROSS SECTIONAL STUDY AT KLE'S DR. PRABHAKAR KORE HOSPITAL AND MRC, BELAGAVI.

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JAWAHARLAL NEHRU MEDICAL COLLEGE, BELAGAVI, KARNATAKA.

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LIST OF ABBREVIATIONS

- UGI- Upper gastrointestinal
- PC/SD- Platelet count/ splenic diameter
- CHD- Common hepatic duct
- IVC- Inferior vena cava
- NK- Natural killer
- LSECs- Liver sinusoidal endothelial cells
- KCs- Kupffer cells
- HSCs- Hematopoetic stem cells
- CNS- Central nervous system
- TLC- Total leucocyte count
- PT/INR- Prothrombin time/ International normalized ratio
- GGT- Gamma glutamyl transferase
- ANA- Anti-nuclear antibody
- LKM- Liver-kidney microsomal

- ANCA- Anti-neutrophil cytoplasmic antibody
- HBV- Hepatitis B virus
- HCV- Hepatitis C virus
- CO- Cardiac output
- RBF- Renal blood flow
- ALP- Alkaline phosphatase
- PPG- Portal pressure gradient
- VEGF- Vascular endothelial growth factor
- PPV- Positive predictive value
- NPV- Negative predictive value
- LR- Likelihood ratio
- ROC- Receiver operating curve
- HCC- Hepatocellular carcinoma
- CTP- Child Turcotte Pugh
- MELD- Model for end stage liver disease
- AUC- Area under the curve
- EGD- Esophago-gastro-duodenoscopy
- PSR- Platelet count to splenic diameter ratio
- USG- Ultrasonography
- SD- Splenic diameter
- NAFLD- Non alcoholic fatty liver disease
- IQR- Interquartile range

ABSTRACT

BACKGROUND: Cirrhosis of the liver is a chronic and progressive disease characterized by extensive fibrosis and the formation of regenerative nodules throughout the liver tissue, which can result in complications such as portal hypertension and esophageal varices[1]. The prevalence of esophageal varices in cirrhotic patients varies widely, from 24% to 80%[2], and they carry a high risk of bleeding. Detecting these varices is essential for diagnosing cirrhosis and serves as a prognostic indicator. Currently, endoscopy is the standard diagnostic tool[3], but not all regions have easy access to this procedure. Recent research has focused on noninvasive methods to identify patients and potentially avoid the need for invasive procedures like endoscopy in low-risk populations. One such noninvasive tool is the platelet count to spleen diameter ratio (PC/SD), which can predict the presence of varices in cirrhotic patients[5].

AIM: To validate whether the platelet count/spleen diameter ratio can be used to predict the presence of esophageal varices in patients with hepatic cirrhosis.

METHODS: This analytical cross-sectional study aimed to validate a diagnostic test for hepatic cirrhosis and was conducted from January 2023 to December 2023. Patients diagnosed with hepatic cirrhosis were included and categorized based on their Child-Pugh score. Biochemical parameters were assessed, and spleen diameter was measured using ultrasound. The ratio of platelet count to spleen diameter was calculated and analyzed to evaluate its ability to predict the

presence of esophageal varices. Upper gastrointestinal endoscopy served as the gold standard. Sensitivity, specificity, positive and negative predictive values, and positive and negative likelihood ratios were determined, with cutoff points established using receiver-operating characteristic curves.

RESULTS: A total of 110 patients were included. The mean age was 50.88 ± 11.8years. The etiology of cirrhosis included alcohol in 96 (87.2%), NAFLD in 4 (3.6%), hepatitis B in 8 (7.2%) and Wilson's disease in 2 (1.8%). Esophageal varices were present in 80 (72.7%) patients. Child-Pugh classification, 9 (8.2%) patients were classified as class A, 61 (55.5%) as class B, and 40 (36.4%) as class C. The platelet count/spleen diameter ratio to detect esophageal varices independent of the grade showed using a cutoff value of ≤ 896, had 91.3% sensitivity, 80% specificity, and positive and negative predictive values of 92% and 77.4%, respectively.

CONCLUSION: Our findings indicate that the platelet count to spleen diameter ratio could be an effective tool for detecting esophageal varices in patients with hepatic cirrhosis.

KEYWORDS: Platelet count/spleen diameter ratio, Esophageal varices, Hepatic cirrhosis, PC/SD ratio, UGI scopy.

INTRODUCTION

Cirrhosis of liver is a chronic progressive disease, characterized by widespread fibrosis(scarring) and formation of regenerative nodules throughout the liver parenchyma[1]. In other words, the final outcome of liver parenchyma fibrosis due to any cause is cirrhosis. It is 14th leading cause of death worldwide[1].

A major complication of hepatic cirrhosis is portal hypertension[1]. Esophageal varices can be seen in about 80% of the people with cirrhosis of liver and around 28% among this population will experience a minimum of one bleeding episode due to varices.[2] Most of the first bleeding episodes occur within the first year after varices are detected, with an initial hemorrhage mortality rate of 5%-10%.[2] The prevalence of esophageal varices varies, reported to be between 24%-80%.[2]

The emergence of esophageal varices due to elevated portal pressures in individuals with liver cirrhosis significantly influences the determination of prognostic scores and the formulation of management strategies for these patients.[3] Therefore, recognising the presence, of these esophageal varices is an important part of diagnosis in cirrhosis patients and serves as a marker for prognosis of the disease[3].

The first step which is most critical in preventing this bleed involves identifying

patients at risk of bleeding and selecting them for prophylactic treatment[3]. Numerous noninvasive factors have supplanted the necessity for UGI scopy in assessing the presence and extent of esophageal varices in cirrhosis of liver[3]. Among multiple factors available, platelet counts and measurements of diameter of the spleen have been identified as positively associated with histopathological alterations seen in liver cirrhosis[3].

Further assessment of their predictive capacity for esophageal varices is warranted. Current guidelines state that there are no markers that can substitute in determining whether varices are present and grades of esophageal varices[3]. Endoscopy remains the one and only standard, accepted technique for diagnosis[3]. However, not all the countries have free, easiliy available access for all the patients to undergo endoscopy and many of the resources are quite limited in several countries.

Non-invasive methods that can predict esophageal varices could help us to limit use of endoscopy to those patients with higher probability of having varices. Not much information was available on this topic until recent times. Only recently, a general agreement has been reached on several aspects of portal hypertension, such as it's definition, methods of diagnosis and strategies for therapy, which recommends endoscopy for all cirrhosis patients to verify varices[4].

Recent studies have highlighted noninvasive methods to identify patients, aiming to avoid invasive procedure such as endoscopy in population which might be at low risk for having or developing esophageal varices.

A lot of variability has been observed in predictors of varices being present, across several studies which likely reflects differences in study populations and disease spectrum, complicating the development of a universally applicable predictive model.

A very simple tool that consists of ratio of platelet count and splenic diameter i.e., (PC/SD) can act as a non-invasive, marker to predict whether varices are present in patients of liver cirrhosis[5]. This ratio meets strict methodological criteria and is pathophysiologically based[5]. Its diagnostic accuracy is validated through use of endoscopy in patients with no esophageal varices who follow up later[5].

Precursory results indicate that the diagnostic precision of the platelet count/splenic diameter ratio is consistent across patient subgroups with various liver diseases and different testing methods, implying its broad applicability. However, no studies have confirmed these initial findings. Most of the past studies had been conducted in Caucasian populations and hence, ratio may vary among different populations and across different regions[5].

Therefore, different non invasive tools or markers are needed that may act as predictors for presence or absence of esophageal varices. This study aims to substantiate the PLATELET COUNT/SPLENIC DIAMETER ratio as a predictor of esophageal varices being present or absent in patients of liver cirrhosis.

OBJECTIVES

To validate whether the platelet count/spleen diameter ratio can be used to predict the presence of esophageal varices.

REVIEW OF LITERATURE

ANATOMY

The liver, a large organ in humans, weighs between 1200 and 1500 grams[15]. it lies under the protection of ribs in the upper quadrant on the right side. Anatomically, it is divided into two lobes: the right lobe, which is six times larger than the left lobe[15]. Another small lobe called the caudate lobe is located posteriorly, and the quadrate lobe is situated inferiorly within the right lobe[15]. The lobes of right and left are separated by the falciform ligament in the front, the

ligamentum venosum fissure in the back, and the ligamentum teres fissure below[15]. (Figure A, B, C)

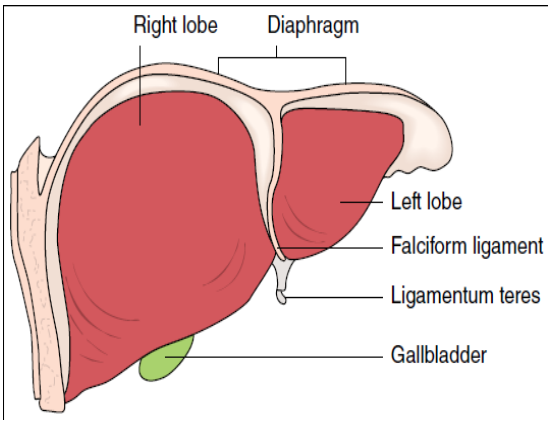


Fig A- Anterior surface of liver

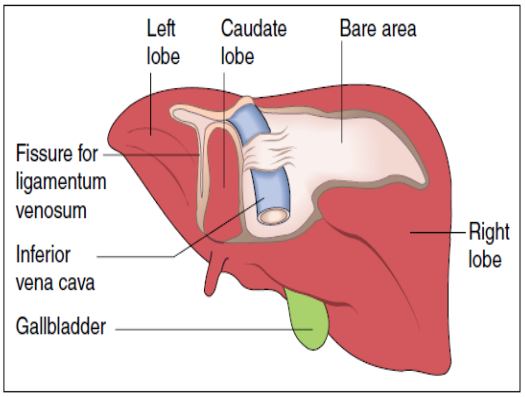
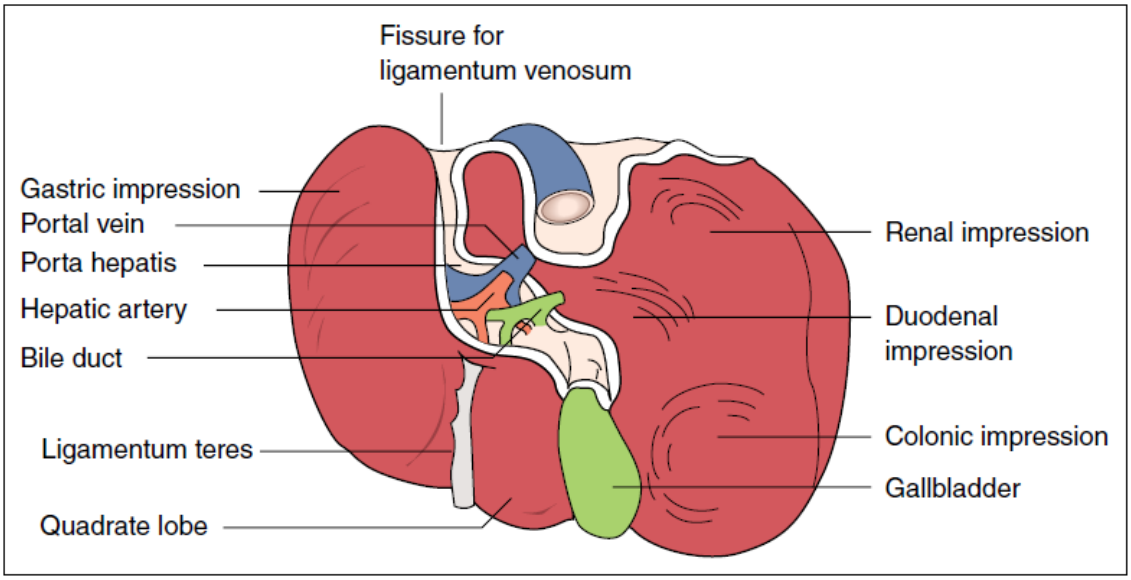


Fig B- Posterior surface of liver

Fig C- Inferior surface of liver



The liver has a dual blood supply from the portal vein and the hepatic artery, both entering through the porta hepatis. Within the porta hepatis, these two major blood vessels divide to supply different liver quadrants. The right and left hepatic biliary ducts merge to form the common hepatic duct(CHD)[15].

The liver's nerve supply includes the nerve plexus of the hepatic system, which consists of fibers from T7–T10 sympathetic ganglia, synapsing in celiac plexus, and also fibers from the vagus nerves of both the sides and right phrenic nerve[15].

Ligamentum venosum is an embryologic remnant of the fetal ductus venosus. It emerges from the left side of portal vein and connects with the IVC where the left hepatic vein enters. Ligamentum teres, which is a leftover portion of the umbilical vein from fetal development, extends along the unfettered border of the falciform ligament from the navel to lower margin of the liver, where it fastens to the left branch of portal vein. Tiny veins accompanying it establish connections between the portal vein and the veins surrounding the umbilicus. These veins become more noticeable when there's obstruction in the portal venous system within the liver[15].

Veins from the liver empty into the right and left hepatic veins, that stem from dorsal aspect of the liver, ultimately discharging into the IVC close to its junction with the right atrium.[15] The lymphatic vessels of the liver culminate into small gland clusters and surround the porta hepatis. From there,

efferent blood vessels drain into the glands encircling the celiac axis. Additionally, superficial hepatic lymphatics traverse the diaphragm through falciform ligament and conclude into mediastinal glands[15].

One more cluster of lymphatics go along the IVC thorax glands within the thoracic portion of IVC[15].

The normal diameter of the spleen radiologically is less than 13 cm.[15]

LIVER HISTOLOGY

Hepatocytes make uptwo-thirds of the structure of liver. Hepatocytes are multiangular and approximately 32 micronmeters in diameter. Each hepatocyte contains a single nucleus, though occasionally they may have multiple nuclei, and they divide through mitosis. Average lifespan of a hepatocyte is approximately 160 days[19].

Hepatocytes have three surfaces:

1. Sinusoid and space of Disse
2. Canaliculus
3. Neighbouring hepatocytes[19]

They lack a basement membrane. According to Wiley, sinusoids are basically lined by endothelial cells featuring small pores, facilitating the

diffusion of macro molecules from the bloodstream to hepatocytes. The vascular side of the sinusoids reside the phagocytic cells of the reticuloendothelial system known as Kupffer cells, along with pit cells, also referred to as natural killer (NK) cells, which are cytotoxic lymphocytes[19].

The normal human liver contains approximately 202,000 cells per milligram, with 171,000 being parenchymal cells and 31,000 being sinusoidal cells, which include Kupffer cells. Between hepatocytes and sinusoidal endothelial cells, is the space of disse, that houses fat storing cells known as Ito cells and lipocytes. Most important function of these cells is storage of vitamin A, and when activated during disease states, they transform into collagen-synthesizing myofibroblasts. Lymphatics of the liver are present in periportal connective tissue and are lined with endothelium throughout their course. Tissue fluid exudes through these endothelial cells into lymph vessels[19]. (Figure D, E)

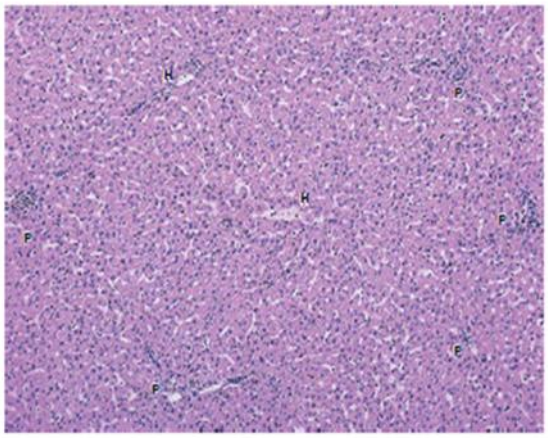


Fig D:Normal Liver Histology[19]

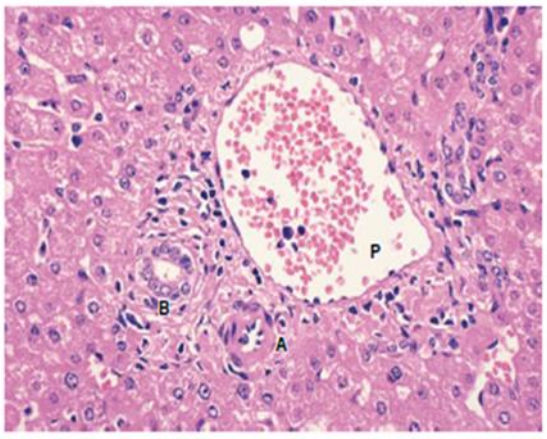


Fig E: Normal Portal Tract[19]

CIRRHOSIS OF LIVER

ETIOLOGY OF CIRRHOSIS[18]

1. Viral Hepatitis i.e., B, C and D

2. Alcohol

3. Non alcoholic steatohepatitis

4. Metabolic- Iron overload (hemochromatosis)

    Wilson's disease

    Alpha1-antitrypsin deficiency

    Tyrosinemia

    Galactosemia

5. Primary sclerosing cholangitis

6. Primary biliary cirrhosis

7. Autoimmune hepatitis

8. Hepatic venous outflow obstruction

9. Toxins and drugs- Methotrexate, Amiodarone

PATHOGENESIS OF CIRRHOSIS

The liver cells involved in the pathogenesis of cirrhosis include both cells of liver parenchyma and cells of other functions as well. The hepatic sinusoids are lined by three types of non-parenchymal cells:

1. Liver sinusoidal endothelial cells (LSECs),
2. Kupffer cells (KCs),
3. Hepatic stellate cells (HSCs).

Among these, stellate cells are most commonly implicated in the formation of cirrhosis. When activated by inflammatory cytokines, stellate cells transform into myofibroblasts. This activation leads to collagen deposition, which is the primary cause of cirrhosis in the liver[19].

APPROACH TO A PATIENT WITH CIRRHOSIS-[16]

1. Ask for the patient's demographic profile.
2. Take an appropriate clinical history and inquire about complaints of:

- Fatigue

- Loss of weight
- Loss of appetite
- Abdominal pain
- Yellowish discoloration of the sclera and skin
- Pruritus
- Changes in the color of urine and feces
- Leg edema
- Abdominal distension
- Bleeding from gums, nosebleeds, bruises, purpura, hematemesis, and melena
- Sexual abnormalities and menstrual history

3. Enquire about the history of past illnesses such as:

- Jaundice
- Hepatitis
- Drug ingestion
- Blood transfusion

4. History of alcohol consumption.

5. Any family history of liver disease or autoimmune disorders[16].

6. On examination, look for:[16]

● Nutritional status, hyperthermia, foul-smelling breath, jaundice, hypo- or hyperpigmentation, purpuric rash, finger clubbing, leukonychia, spider angioma, palmar erythema, gynecomastia, testicular atrophy, alopecia, hand contracture deformities, and blood pressure.

● On abdominal examination, check for fluid accumulation, prominent or engorged veins in the epigastrium and flanks, and palpation of abdominal organs.

● In CNS examination, look for asterixis, higher mental functions, stupor, and tremors.[11]

INVESTIGATIONS:[17]

1. In Hematology:
    - Hemoglobin
    - Total leukocyte count (TLC)
    - Platelet count
    - Prothrombin time/International Normalized Ratio (PT/INR)
2. In Biochemistry:
    - Serum bilirubin
    - Transaminases
    - Albumin
    - Globulin

- Alkaline phosphatase
- Gamma-glutamyl transferase (GGT)
- Serum iron, ferritin, transferrin saturation
- Serum ceruloplasmin, serum copper
- Alpha-1 antitrypsin
- Renal function tests

3. If ascites is present:

- Diagnostic paracentesis
- Daily weight monitoring
- 24-hour urine volume and sodium excretion

4. Autoimmune profile including:

- Anti-Smith (Sm) antibody
- Anti-mitochondrial antibody
- ANA antibodies
- Anti-liver kidney microsomal (LKM) antibody
- Antineutrophil cytoplasmic antibody (ANCA)

5. Viral markers:

- HBV+
- HCV+

6. Tumor marker:

- Alpha-fetoprotein

7. Upper GI endoscopy.

8. Imaging:

   - Ultrasonography of the hepatic system
   - CT scan or MRI of the abdomen

9. Liver biopsy if not contraindicated.

10. Electroencephalography for grading hepatic encephalopathy.

CLINICAL CIRRHOSIS[16]

COMPENSATED CIRRHOSIS-

Cirrhosis is often asymptomatic and may be discovered through routine biochemical assays or a thorough clinical examination.[16]

- On examination, if the liver is palpable in the epigastrium, it is an important sign for considering a diagnosis of cirrhosis.
- Confirmation of cirrhosis is achieved through liver imaging and FibroScan.
- If signs and symptoms suggestive of portal hypertension are present, it indicates a worse prognosis.[16]

DECOMPENSATED CIRRHOSIS-[18]

Patients usually seek advice due to symptoms such as ascites, jaundice, gastrointestinal bleeding, or altered sensorium (hepatic encephalopathy).

• Decompensated cirrhosis can be precipitated by bacterial infection,surgery, trauma, or medications.

• Weight loss in the form of muscle wasting is common.

• Most common cause of hepatic encephalopathy is cirrhosis.

• The presence of jaundice indicates that the liver's destructive capacity exceeds its regenerative capacity.

• Purpura over the arms and forearms, chest, shoulders, and lower limb shins indicates thrombocytopenia.

• Overactive circulation is indicated by warm peripheries, tachycardia, hypotension, and easily palpable pulses.

• Ascites, hepatomegaly, and splenomegaly are present, along with peripheral edema.[17]

• Other features of decompensation include:[17]

a) Hepatorenal syndrome

b) Spontaneous bacterial peritonitis

c) Hyponatremia[17]

MODIFIED CHILD PUGH SCORE USED IN OUR STUDY-[18]

Parameter	Points assigned		
	1	2	3
Ascites	Absent	Slight	Moderate
Bilirrubin, mg/dL	<= 2	2-3	>3
Albumin, g/dL	>3.5	2.8-3.5	<2.8
Prothrombin time * Seconds over control	>4	4-6	>6
Encephalopathy	None	Grade 1-2	Grade 3-4

Figure F: CHILD PUGH SCORE

Class A- score 5-6

Class B- score 7-9

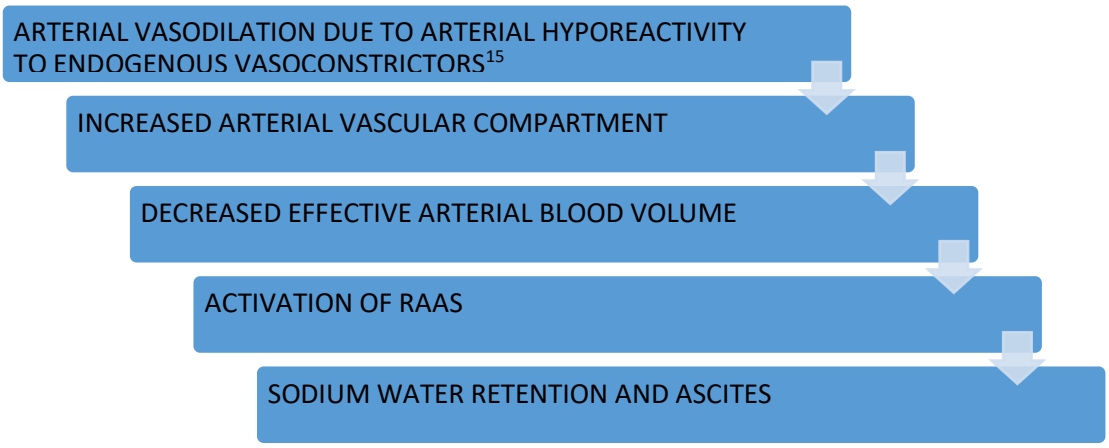
Class C- score 10-15

PATHOPHYSIOLOGY OF CIRRHOSIS

Complications in decompensated cirrhosis arise from vasodilation and hyperdynamic circulation.[15]

- Increased blood flow in peripheral arteries and portal vein blood flow.
- Increased cardiac output (CO).
- Reduced renal blood flow (RBF), particularly perfusion of the renal cortex.

a) Vasodilation



b) Hyperdynamic circulation:[17]

- Nitric oxide is the most significant molecule for hyperdynamic circulation.
- Increased splanchnic and systemic nitric oxide production leads to vasodilation.
- Decreased nitric oxide in the intra-hepatic circulation results in:
- Increased intra-hepatic vascular resistance
- Increased portal venous pressure

Extrahepatic manifestations of cirrhosis:[18]

a) Hepato-pulmonary syndrome

b) Hepato-renal syndrome

c) Cirrhotic cardiomyopathy

d) Gastrointestinal manifestations of cirrhosis:

- Splenomegaly
- Peptic ulcer
- Small bowel bacterial overgrowth
- Gallstones
- Chronic calcific pancreatitis

e) Foetor hepaticus

f) Spider angioma

g) Palmar erythema

h) Leukonychia

i) Madelung disease

j) Clubbing

k) Dupuytren's contracture

l) Sarcopenia

m) Gynecomastia

SIGNS SUGGESTIVE OF POOR PROGNOSIS-[15]

a) Prolonged PT

b) Marked ascites

c) Advanced age

d) GI bleeding

e) High daily alcohol consumption

f) Hyperbilirubinemia

g) Increased ALP

h) Hypoalbuminemia

i) Poor nutrition

FORMATION OF VARICES AND COLLATERALS[15]

In response to elevation in hepatic portal pressure, there is development of collateral circulation between portal vein and systemic circulation. This rise in portal pressure is usually much larger than systemic venous pressure, and hence there occurs reversal of blood flow i.e., from portal system towards systemic venous circulation. Common sites where these collaterals form are distal esophagus and proximal part of the stomach, around the umbilicus, also known as caput medusae, the retroperitoneum, formed by communication between the

ovarian and iliac veins and last is the rectum, where rectal varices develop due to communication between the inferior mesenteric vein and the pudendal vein.[15]

An area called the palisade zone is the predominant watershed zone between portal circulation and systemic circulation. Veins in this region are most prone to bleeding because there are no perforating veins at this level that connect veins in the submucosa to periesophageal veins. The main theory of pathogenesis behind the development of collaterals and varices is the collective effect of new onset angiogenesis and dilatation of vessels which is under the influence of VEGF and nitric oxide.[15]

For the development of esophageal varices,,a portal pressure gradient(PPG) of at least 10 mmHg is required. Moreover, a ppg of at least 12 mmHg is necessary for varices to bleed. Local factors such as increased wall tension in the varices and also sometimes required for bleeding to occur.[15]

DETECTION OF VARICES[15]

Most common and widely accepted method to detect esophageal varices in endoscopy. There is a general agreement that all patients with cirrhosis should undergo screening for esophageal varices by UGI scopy. It is repeat at intervals of every 2-3 years in patients of cirrhosis who have no evidence of varices on initial endoscopy. So far, none of the non invasive methods of detecting varices have been deemed useful. Therefore, large multi-centre studies are required for the same.[15]

Figure G: Endoscopic appearance of small esophageal varices in the distal esophagus.[15]

Figure H: Large esophageal varices with a site of recent bleed indicated by the fibrin plug.[15]

MANAGEMENT OF CIRRHOSIS[16]

COMPENSATED CIRRHOSIS	DECOMPENSATED CIRRHOSIS
a) Adequate balanced diet- 35-40kcal/kg/day 1.2-1.5g/kg/day Avoid fatty foods, salt restriction.	Treatment of precipitating factors such as hepatic encephalopathy, ascites and variceal haemorrhage.
b) Avoidance of alcohol and obesity	
c) Avoid fluid retention	
d) Avoid encephalopathy	
e) Functioning of the renal system to be maintained	
f) Haemorrhage from the varices should be prevented	

RELEVANT STUDIES

An analytical study that was cross sectional, aimed to substantiate a simple diagnostic investigation for liver cirrhosis by **González-Ojeda A et al.** (2019)[1]. Patients with liver cirrhosis were included for the study and subsequently

classified based on their Child Turcotte Pugh scores. Biochemical lab investigation were assessed, and the longest bipolar diameter of the spleen was measured via ultrasound. The platelet count/spleen diameter(PC/SD) ratio was calculated and further analysis was done to evaluate its predictive value for detecting esophageal varices. Upper GI scopy was used as the gold standard for diagnosis. Sensitivity, specificity, PPV, NPV and, positive and negative LR ratios were calculated, with cut-off points established using ROC curves

Results -:

Study Patients: 90

Mean Age: 53.75 ± 12 years

Gender Distribution: 50 men, 41 women

Etiology of Cirrhosis:

Alcohol: 48 patients

Viral (Hepatitis B/C): 24 patients

Alcohol plus Hepatitis C: 3 patients

Cryptogenic: 9 patients

Primary Biliary Cirrhosis: 7 patients

Esophageal Varices present: 73 patients

Child Turcotte Pugh Classification:

Class A: 17 patients

Class B: 37 patients

Class C: 37 patients

Using a cut-off value of <884 for the PC/SD ratio to detect esophageal varices, irrespective of,,grade, the study found:

Sensitivity-: 84%

Specificity-: 70%

PPV-: 94%

NPV-: 40%

These results suggest that the ratioPC/SD may be a useful tool for detecting esophageal varices in patients with hepatic cirrhosis.

A cross-sectional observational study was conducted in a tertiary care hospital in Raichur, by **Payal B et al** (2022)[2] on 114 diagnosed cases of liver cirrhosis. Portal hypertension and the development. of esophageal varices are significant complications in cirrhosis, with a high risk of, bleeding, from, the, varices. Consequently, endoscopic screening every few months is strongly recommended for liver cirrhosis patients to detect esophageal varices. Several studies have noted that certain-parameters, such as enlargement of the spleen and drop in platelet count, are linked to the presence of varices and also portal-hypertension.

This study aimed to evaluate the ratio, of PC/SD in patients with-liver-cirrhosis and its relationship with presence-of-esophageal varices. The inclusion criteria were diagnosed cases of liver cirrhosis in patients with age 13 years and more. The exclusion criteria were patients with HCC, thrombosis of portal vein, and patients with a past history of perabdominal surgery.

By applying a ROC curve, a platelet count to spleen diameter ratio cutoff value of 1050 was obtained which provided positive and negative predictive values with 97.11% sensitivity and 100% specificity.

This study concluded that the presence of esophageal varices, is a common complication in liver cirrhosis, with a prevalence of 91.23%. This ratio may be recommended as a predictive marker for the presence of esophageal varices and as a surrogate marker in setting where endoscopy facilities are not available.[2]

A study conducted by **Mattos A Z et al (2017)[3]** sought to assess the effectiveness of the PC/SD ratio in noninvasively predicting esophageal varices in a group of cirrhotic patients from a separate center, distinct from the original one where the ratio was first established. The research, carried out at the ambulatory clinic for cirrhosis in a Brazilian hospital, analyzed platelet count, splenic diameter, and the presence of esophageal varices, along with CTP and M.E.L.D scores.

Using previously published cutoff of 909 for the PC/SD ratio, the study required sample size of 139 patients to ensure results with 95%CI. Ultimately, 163 cirrhotic patients were included, with 56.7% being male and an average age of 56.6 ± 11.6 years.

Key Findings:

Patient Demographics:

Total Patients: 164

Male: 56.7%

Mean Age: 56.5 years

Significant Factors in this analysis :

Platelet Count

Spleen-Diameter

Ascites

CTP Score

M.E.L.D Score

PC/SD ratio

Each of these factors was significantly related to the presence of esophageal varices ($P<0.05$).

The study concluded that the platelet count/spleen diameter ratio is not adequate for predicting esophageal varices in cirrhotic patients in this independent cohort. This suggests that while the ratio might show promise in some settings, its general applicability as a non-invasive predictive tool for esophageal varices in cirrhosis requires further validation across different populations and clinical settings.

The following study was conducted by Giannini et al in two parts. In first part of the study, they retrospectively assessed presence of esophageal varices in 144 patients of cirrhosis. In the second part, they assessed the reliability of these results in a following group that consisted of 122 patients. Ultimately, they evaluated these parameters in another subgroup of 144 patients of compensated liver disease. Each of the 264 patients underwent a comprehensive biochemical

workup, UGI scopy, and USG measurement of spleen bipolar diameter. The PC/SD ratio was derived for each of the patients.[3]

Prevalence of varices in the first group was 61% and in the second group was 58%. In multivariate-analysis, it was found to be the only parameter independently associated with the presence of esophageal varices. A cut-off value of 909 for the PC/SD ratio had 100% NPV for diagnosis of esophageal varices. This result was consistent in the other group of patients as well and in those who had compensated liver disease.

Cost-benefit analysis-:

Screening cirrhotic patients using the "platelet count/spleen diameter ratio strategy" was more cost-effective compared to "scope all strategy."

Another study conducted by **Giannini et al (2019)[4]**, noninvasive evaluation of esophageal varices can enhance the management in liver cirrhosis patients, potentially reducing both medical as well as financial burden associated with routine endoscopy. This international study, carried out across multi-centres aimed to successfully substantiate the use of PC/SD ratio for the non-invasive diagnosis of varices.

Study Details:

Participants: 216 patients

Procedure: Endoscopy for screening of esophageal varices and assessment of the platelet count/spleen diameter ratio $(N/mm^3)/mm$ for all patients.

Analysis: Diagnostic accuracy of the platelet count/spleen diameter ratio was calculated with a previously established cutoff of 908. The diagnostic performance was evaluated across different severity and etiology subgroups.

Key Findings:

Prevalence of EV: 54.1%

Diagnostic accuracy of PC/SD : 86.0%, significantly higher than that of either platelet count-or spleen diameter alone.

Performance Metrics for the 909 Cutoff:

Sensitivity: 91.5%

Specificity: 67.0%

Positive Predictive Value: 76.6%

Negative Predictive Value: 87.0%

Positive Likelihood Ratio: 2.77

Negative Likelihood Ratio: 0.13

The diagnostic accuracy of the PC/SD ratio was consistent across different stages of the disease and etiology subgroups of the same.

The platelet count/spleen diameter ratio demonstrates high diagnostic accuracy for the noninvasive detection of EV in cirrhotic patients. With a cutoff value of 909, this ratio shows high sensitivity and acceptable specificity, making it a valuable tool for predicting the presence of EV. This approach can be deemed as safe and easily available method to improve the prognosis and management of liver cirrhosis, potentially reducing the need for routine endoscopic screening.[4]

A retrospective analysis was performed by **Schwarzenberger E et al (2016)**[5] on 137 cirrhotic patients over 18 years old who were screened by endoscopy to look for presence of varices for a period of one year. Collected information included age of the patients, sex, etiology of cirrhosis, diameter of the spleen, PT/INR, total bilirubin, albumin, platelet count, child pugh score, and endoscopy features.

Total Patients: 136

Gender Distribution: 86 males

Average age: 56 years

Findings:

Esophageal Varices: 76 patients had esophageal varices.

Group Comparisons: There were no significant differences in mean age, sex or cirrhosis etiology between patients with and without evidence of varices.

Diagnostic Accuracy of PC/SD:

Cutoff : 909

Negative Predictive Value: 73%

Positive Predictive Value: 74%

Conclusion:

PC/SD ratio with cutoff value of 908 was not adequately accurate in predicting varices in this group of patients. Therefore, upper endoscopy remains the preferred method for screening for varices in cirrhotic patients.[5]

In the entire cohort, of a study carried out by **Agha A et al (2019)[6]**, platelet count/spleen diameter cutoff of 908 was assessed in diagnosing esophageal varices in the patient pool of 311 subjects and specifically in those who have compensated disease (n=112).

Findings:

Overall Population (EV prevalence 49.5%):

Positive Predictive Value: 96.9%

Negative Predictive Value: 100%

Diagnostic Efficiency: 98.2%

Compensated Cirrhotics (esophageal varices prevalence 26.3%):

NPV: 100%

PPV: 93.8%

In patients with cirrhosis secondary to hepatitis c, the platelet count-spleen diameter ratio can be suggested as noninvasive test for diagnosing esophageal varices, particularly useful in financially backward and developing countries.[6]

A study conducted by **Harit Goverdhan Kothari et al (2019)[7]** included 204 patients who were male with a mean age of 42.77 ± 8.9Dyears. Among them,

188 (93%) patients exhibited esophageal varices, and 61 (30.19%) experienced variceal bleeding. Univariate analysis revealed significant associations of varices with platelet count, spleen diameter, and ratio of the two. For predicting esophageal varices, only the PC/SD ratio demonstrated significance, with an AUC of 65.6% with respect to a cutoff value less than 997. Regarding variceal bleeding prediction, CTP score and the PC/SD ratio were important factors. A platelet and splenic diameter ratio cutoff of <985 showed 82% sensitivity and 63% specificity, with an area under curve of 78% for predicting bleeding through varices.

It was concluded that:

Among noninvasive markers, PC-SD ratio shows promise in predicting esophageal varices and the probability of bleeding through varices in cirrhosis of liver, offering valuable additions to the diagnostic toolkit.[7]

A study conducted by **Zubia Jamil et al (2017)[8]** evaluates the effectiveness of noninvasive markers: platelet count, portal vein diameter, splenic diameter, and the ratio of platelet count to splenic diameter (PC/SD ratio) in detecting esophageal varices.

Study Details:

Participants: 150 cirrhotic patients

Method: Endoscopy was used to detect esophageal varices; diagnostic accuracy of the markers was assessed via ROC curves and Youden index.

Results:

PC/SD Ratio:

AUC: 0.9

Sensitivity: 88.75%

Specificity: 81.43%

Cutoff value: ≤ 1077.42

Platelet Count:

AUC: 0.85

Splenic Diameter:

AUC: 0.77

Portal Vein Diameter:

AUC: 0.59 (not a good predictor)

Conclusion:

The PC/SD ratio is the most reliable marker for predicting esophageal varices, offering a noninvasive, cost-effective alternative to endoscopy, and can help identify high-risk patients, reducing the need for endoscopic screening.[8]

Another study done by **Haile Tesfaye Gebregziabiher et al (2023)[9]**, was to evaluate the diagnostic accuracy of noninvasive tests (platelet count, spleen diameter, and platelet count/spleen diameter ratio) in predicting esophageal varices among cirrhotic patients at University of Gondar Comprehensive Hospital.

Method: A cross-sectional study was conducted from March to October 2022 with 206 cirrhotic patients. Participants were screened for esophageal varices, spleen diameter,

platelet count, and the PC/SD ratio. Data analysis was done using ROC curves to determine diagnostic performance.

Results:

Participants: 206 patients (mean age 41.84 years; 79.4% male)

Esophageal Varices: Present in 85.4% of cases (176 patients)

Cirrhosis: 67% had decompensated cirrhosis (Child-Pugh class B&C)

Diagnostic Performance:

PC/SD Ratio (<818):

PPV: 94.7%

AUROC: 0.835

Spleen Diameter (>145 mm):

PPV: 93.7%

AUROC: 0.783

Platelet Count (<121,000/mm^3):

PPV: 95.1%

AUROC: 0.818

Conclusion: The PC/SD ratio, platelet count, and spleen diameter are effective noninvasive markers for diagnosing esophageal varices, with the PC/SD ratio being the most reliable. These markers can be used in healthcare settings without access to gastrointestinal endoscopy to identify high-risk patients and guide prophylactic treatment.[9]

Another similar study was done by **Sihao Yu et al (2021)[10]** to evaluate the clinical value of the platelet count/spleen volume ratio (PSVR) and spleen volume (SV) in predicting esophageal varices (EV) and high-risk esophageal varices (HRV) in patients with hepatitis B cirrhosis.

Methods:

Study Design: Diagnostic accuracy experiment, retrospective

Participants: 199 patients with hepatitis B cirrhosis

Procedure:

Blood samples collected within 2 days

Electronic gastroscopy and abdominal magnetic resonance examination within 10 days

Patients categorized by Child-Pugh score into groups with or without EV and HRV

Statistical analysis conducted on these groups

Results:

Diagnostic Performance:

AUC for predicting EV/HRV: 85.5% - 92.6%

Higher AUC than PSDR, SV, spleen diameter, and platelet count

PSDR: Higher AUC for diagnosing HRV than SV

SV: Higher AUC for diagnosing EV than PSDR, but difference not significant ($P>0.05$)

Child-Pugh A Patients:

PSVR: Significant predictor of HRV ($P<0.05$)

SV: Reliable predictor of EV ($P<0.05$)

Conclusion:

PSVR is more effective than PSDR, spleen diameter, and platelet count in predicting EV.

In the absence of serological results, SV can replace PSDR for predicting EV. Both PSVR and SV are reliable for predicting EV or HRV in patients with hepatitis B cirrhosis.[10]

A study conducted by **Manuela Mangone (2013)[11]**, was to evaluate the diagnostic accuracy of the platelet count/spleen diameter ratio (PC/SD ratio) for identifying esophageal varices and/or hypertensive gastropathy in patients with compensated cirrhosis.

Methods:

Participants: 87 patients with compensated cirrhosis

Procedure: Calculated the PC/SD ratio and identified an optimal cut-off value. Tested the performance of previously reported cut-off values.

Results:

Optimal Cut-off: PC/SD ratio <936.4

Diagnostic Performance:

Identified 64.5% of patients with esophageal varices

Identified 66.7% of patients with any sign of portal hypertension

Excluded esophageal varices in 64.3% of patients

Excluded any sign of portal hypertension in 68.6% of patients

Conclusion: The PC/SD ratio is not a reliable parameter to avoid unnecessary upper endoscopy in patients with compensated cirrhosis, irrespective of the cut-off value used.[11]

A study conducted by **WW Baig et al (2014)[12]**, assessed the diagnostic utility of the platelet count to spleen diameter ratio (PC/SD ratio) as a noninvasive parameter for diagnosing esophageal varices (EVs) in patients with liver cirrhosis.

Methods:

Participants: 150 stable patients with liver cirrhosis, excluding those with active gastrointestinal bleeding.

Procedure: Prospective evaluation of laboratory and ultrasonographic variables, followed by upper gastrointestinal endoscopy for all patients.

Key Findings:

Significant Differences: Patients with EVs showed significant differences in platelet count, spleen diameter, and PC/SD ratio compared to those without EVs.

Diagnostic Accuracy:

Optimal Cut-off: PC/SD ratio <1014

Positive Predictive Value: 95.4%

Negative Predictive Value: 95.1%

Accuracy (AUROC): 0.942 (95% CI: 0.890 to 0.995)

Conclusion: The PC/SD ratio demonstrates high accuracy for diagnosing EVs noninvasively in patients with liver cirrhosis. While it shows promise, it cannot entirely replace upper gastrointestinal endoscopy as the standard diagnostic tool for EVs in cirrhotic patients, but it can be useful when endoscopy is unavailable.[12]

A study conducted by **Augustino Colli et al (2017)[13]**, aimed to compare the diagnostic accuracy of platelet count, spleen length, and platelet count-to-spleen length ratio against esophago-gastro-duodenoscopy (EGD) in detecting varices in individuals with chronic liver disease or portal vein thrombosis.

Key Results:

Participants: The review included 25 studies with a total of 5096 participants.

Diagnostic Tools Evaluated:

Platelet Count: Assessed in 13 studies (1489 participants).

Spleen Length: Assessed in 38 studies (5235 participants).

Platelet Count-to-Spleen Length Ratio: Found to be the most accurate in identifying high-risk varices.

Threshold identified: <909 (platelet count/mm^3)/(spleen length mm).

High sensitivity in detecting varices, missing only 7% of cases where varices were present.

Ratio <909 can effectively exclude the presence of varices.

Advantages: Useful for reducing the number of unnecessary EGDs, particularly beneficial in populations with a high prevalence of cirrhosis and varices.

Limitations: Not sufficient to replace EGD entirely, as there were concerns about bias in how test outcomes were defined and the blinding of test results to endoscopists.

Conclusion:

The platelet count-to-spleen length ratio is highly accurate in identifying individuals at high risk of varices but should be used alongside EGD rather than replacing it. Bias in study methodologies needs addressing for more robust accuracy estimates.[13]

A study conducted by **Yang- Chun Du (2023)[14],** aimed to evaluate noninvasive predictors (PSR, PSVR, SZ) and their combinations for predicting esophageal varices (EV) severity in liver cirrhosis patients. Conducted at Guangxi Medical University, it included 82 cirrhotic inpatients who underwent endoscopy, ultrasound, CT scans, and lab tests. Results showed PSR and PSVR significantly predicted severe and moderate/severe EV, with PSR+PSVR demonstrating the highest diagnostic accuracy (AUC 0.735-0.765). This suggests these parameters can effectively assess EV severity, with PSR+PSVR potentially offering superior diagnostic capability.[14]

METHODOLOGY

Study design

A cross sectional study in a tertiary care Hospital.

Period of study

1st January 2023 to 31st December 2023

Source of the data

Patients consulting Medicine OPD and in-patients admitted in department of medicine at KLES Dr. Prabhakar Kore Hospital and Medical Research Centre, Belagavi, a tertiary care hospital in Karnataka, India, fulfilling the inclusion criteria during the study.

The inclusion and exclusion criteria considered are briefed below:

Inclusion criteria

● Patients above 18 years of age, diagnosed with hepatic cirrhosis.

Exclusion criteria

● Age less than 18 years

● Patient with HCC.

● Patients who are already on medications for the preventive prophylaxis of bleeding through varices.

● Patients who underwent ligation or sclerotherapy and/or portal hypertension surgery.

Ethical consideration

The study received approval from the institutional human ethics committee. Informed written consent was obtained from all participants, and only those who signed the consent form were included in the study.

Sample size: 110

Our institute gets approximately 173 patients with cirrhosis of liver each year. This data has been obtained from the Medical Records Department. The study duration is of 12 months; hence we expect that around 173 patients with cirrhosis of liver will be coming to the institution during the study period. Assuming that, of the study population visiting our institution, nearly 20% of these patients satisfy the exclusion criteria, then around 173-35 = 138 eligible patients may be available for the study.

In a study done by Giannini was entitled, "Platelet count/spleen diameter ratio: proposal and validation of a non-invasive parameter to predict the presence of esophageal varices in patients with liver cirrhosis", they had reported that, the incidence of esophageal varices among the patients with cirrhosis of liver varies from 60-80%[1].

So, considering a study population of 138 and incidence rate of 61%, we used the following incidence formula for calculating the sample size.

The sample size (n) and margin of error are given by:

$x = Z(c/100)2r(100-r)$

$n = Nx/((N-1)E2 + x)$

$E = Sqrt[(N - n)x/n(N-1)]$

where N is the population size (N=138), r is the fraction of responses that you are interested in (r=61%), and Z(c/100) is the critical value for the confidence level c(Z=1.96).

Putting the above values in the above formula, the sample size obtained is 100 patients with cirrhosis of liver, at a confidence interval of 95% and 80% power of the study.

Considering an attrition rate of 10%, we intend to include 110 patients with cirrhosis of liver in our study.

Data collection tool

All the relevant parameters were documented in a structured study proforma.

Material and methods

● All patients fulfilling inclusion criteria are subjected to thorough clinical examination.

● Complete blood counts including platelet count, coagulation profile, liver function tests

including serum proteins to be done.

● USG abdomen to measure the maximum bipolar splenic diameter(splenic size).

● The Platelet count/ Splenic diameter ratio was derived by dividing the platelet count (in

cells/microL)of the patients by the maximum bipolar diameter of the spleen measured in millimeters.

- The diagnostic accuracy of this formula is to be validated using endoscopic diagnosis.

Statistical analysis

All consecutive patients meeting the inclusion criteria were enrolled, and their data was coded and entered into a Microsoft Excel spreadsheet. The analysis was conducted using SPSS version 21 and Statistica 21. Quantitative data were scrutinized using mean, standard deviation (SD), and ranges, while frequencies were derived for qualitative variables. A contingency coefficient was then utilised to evaluate the association between PC/SD ratio and varices. The diagnostic precision of PC/SD ratio was estimated after calculating the area under ROC (receiver operating characteristic) curve. The cutoff point for each variable was determined along with its sensitivity, specificity, positive likelihood ratio (+LR), negative likelihood ratio (-LR), positive predictive value (+PV), and negative predictive value (-PV).

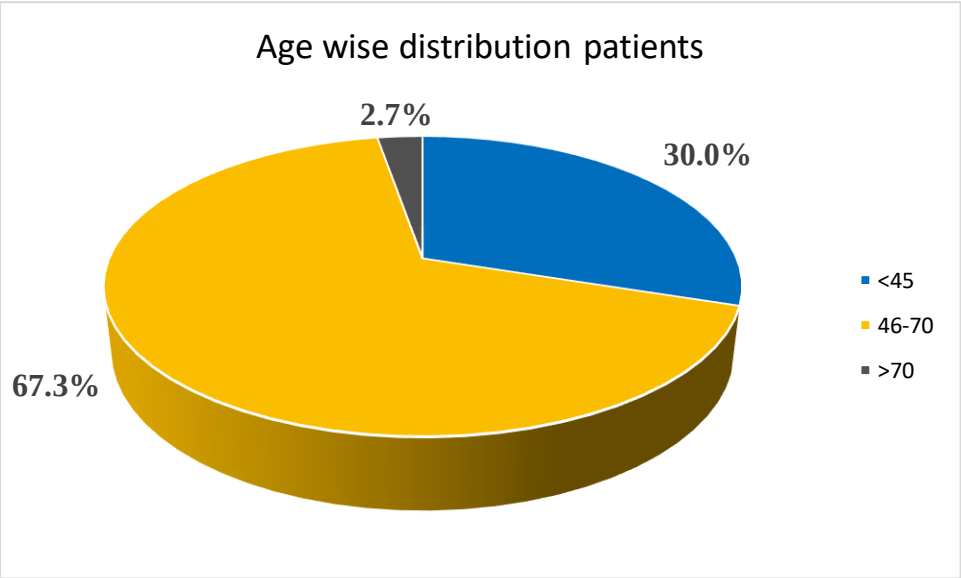
To determine the most definitive screening tool among these variables, a pairwise comparison was conducted by analyzing the differences between the areas under the curve.

RESULTS

Table 1: Age wise distribution of patients

Age group	n (%)
≤45	33 (30)
46-70	74 (67.2)
≥70	3 (2.7)
Total	110 (100)
Mean±SD	50.88±11.810

Figure 1: Age wise distribution of the patients

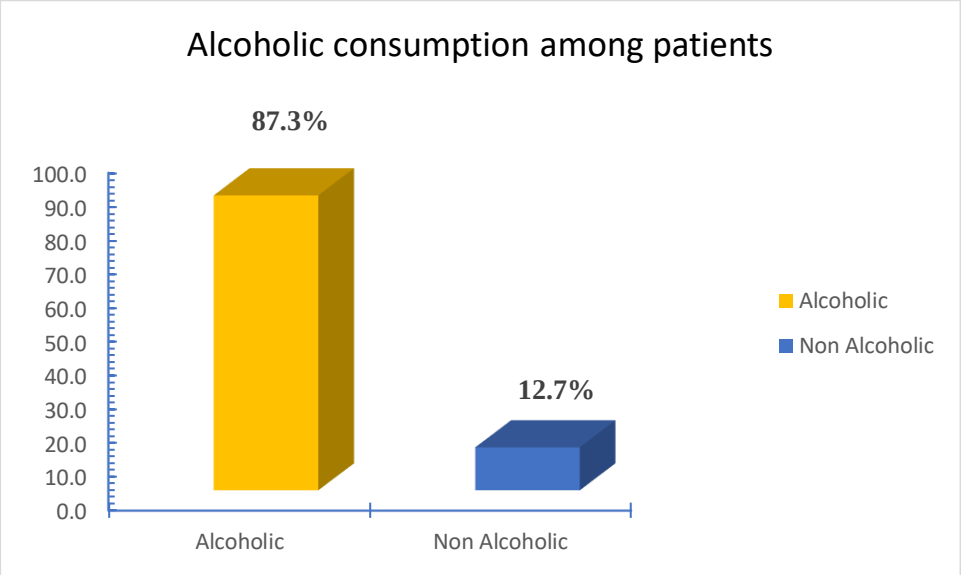


Among 110 patients, 74(67.2%) of the patients were between the age group of 46-70 years, 33(30%) of the patients were less than 45 years old and 3(2.7%) patients were more than 70 years old. Mean age was 50.88 years with standard deviation of 11.810.

Table 2: Distribution of patients based on history of alcohol intake

Alcohol intake	**n (%)**
Alcoholic	96 (87.2)
Non-Alcoholic	14 (12.7)
Total	110 (100)

Fig 2: Distribution of patients based on history of alcohol intake

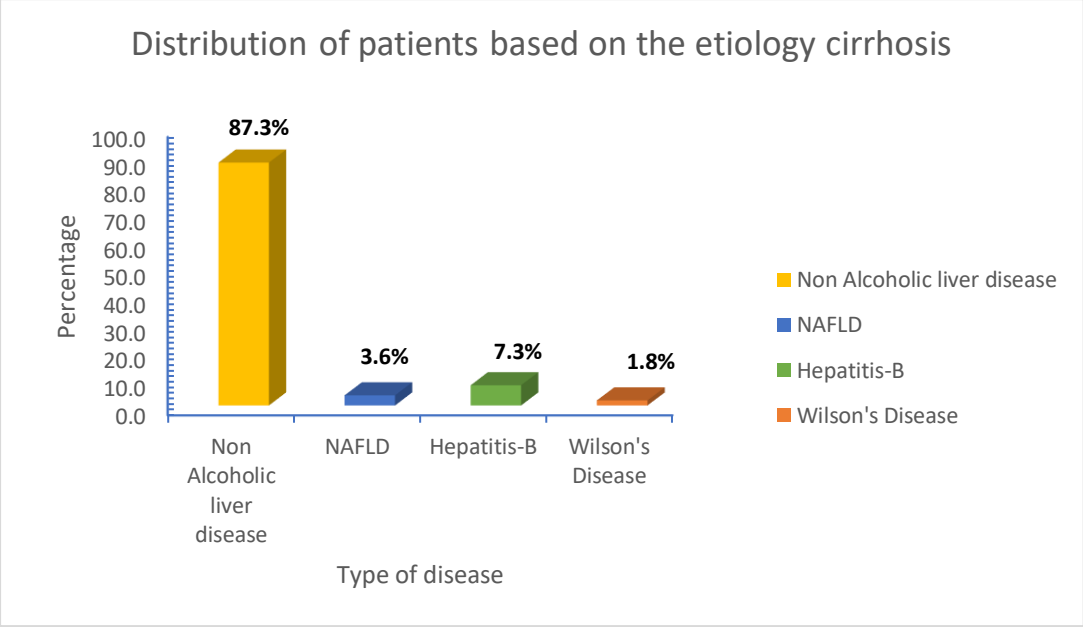


Among the study subjects, 96(87.2%) of the patients were alcoholic and 14(12.7%) of the patients were non alcoholic.

Table 3: Distribution of subjects according to the etiology of cirrhosis

Etiology of cirrhosis	n (%)
Alcoholic liver disease	96 (87.2)
NAFLD	4 (3.6)
Hepatitis-B	8 (7.2)
Wilson's Disease	2 (1.8)
Total	110 (100)

Figure 3: Distribution of subjects according to the etiology of cirrhosis

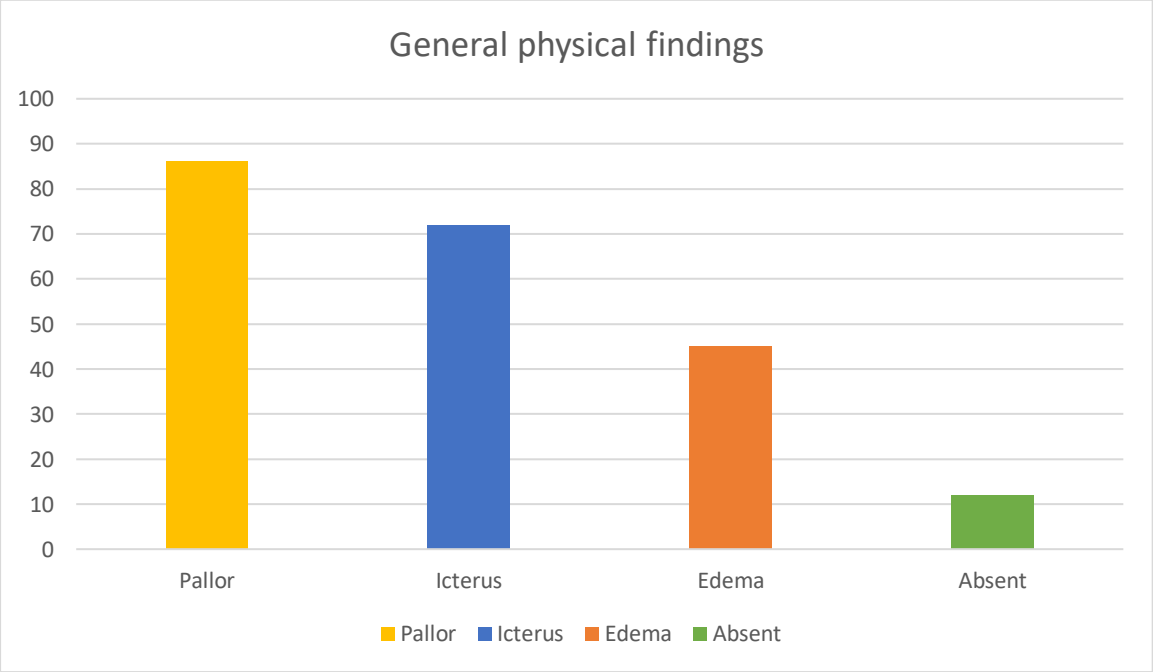


In the study, etiology of cirrhosis of liver was found to be alcohol for 96(87.2%) of the patients, Hepatitis B for 8(7.2%) of the patients, NAFLD for 4(3.6%) of the patients and Wilson's disease for 2(1.8%) of the patients.

Table 4: Distribution of patients according to general physical findings

General physical findings	n(%)
Pallor	86 (78.1%)
Icterus	72 (65.4%)
Edema	45 (40.9%)
Absent	12 (10.9%)

Figure 4: Distribution of patients according to general physical findings

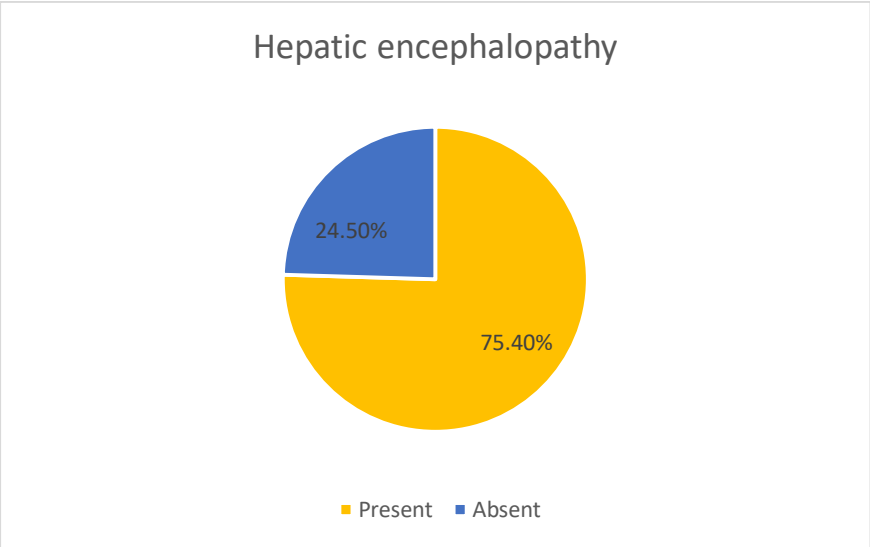


In general physical findings, 86 (78.1%) of the patients had pallor, 72(65.4%) of the patients had icterus, 45 (40.9%) of the patients had edema, whereas any of these were absent in 12 (10.9%) of the patients.

Table 5: Distribution of patients according to Hepatic Encephalopathy

Hepatic encephalopathy	n(%)
Present	83 (75.4%)
Absent	27 (24.5%)
Total	110 (100%)

Figure 5: Distribution of patients according to Hepatic Encephalopathy

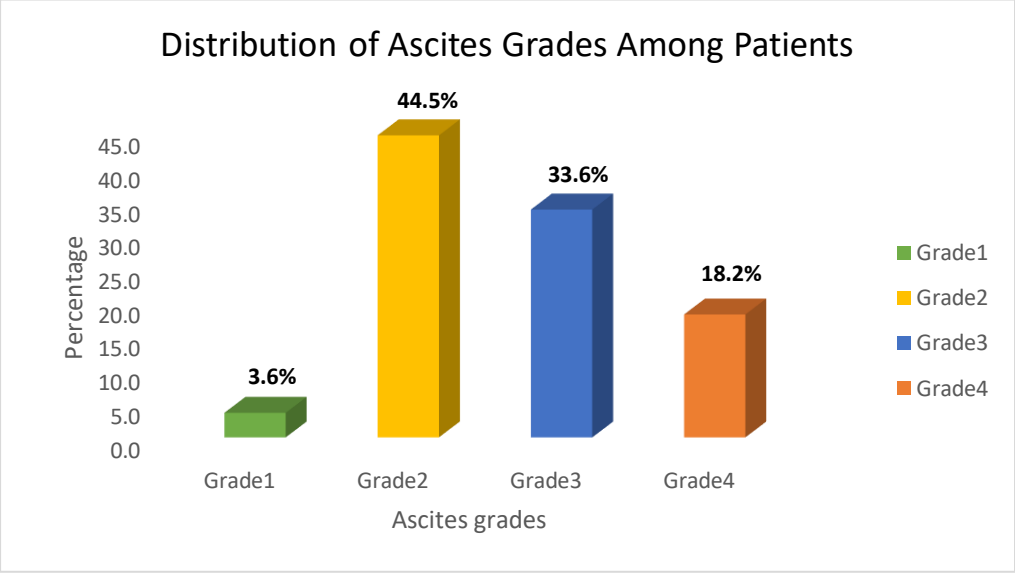


In our study, hepatic encephalopathy was present in 83 (75.4%) of the patients and it was absent in 27 (24.5%) of the patients.

Table 6: Distribution of Ascites Grades Among Patient

Ascites	n (%)
Grade1	4 (3.6)
Grade2	49 (44.5)
Grade3	37 (33.6)
Grade4	20 (18.2)
Total	110 (100)

Figure 6: Distribution of Ascites Grades Among Patient



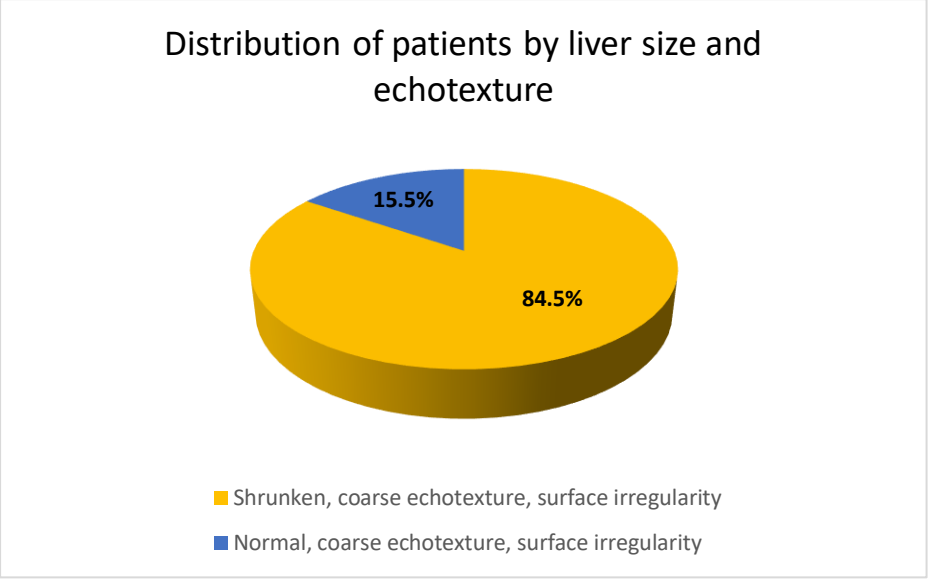
Among the study population and analysis of grades of ascites, grade 2 ascites was most common, found in 49(44.5%) of the subjects, followed by grade 3 ascites found in 37(33.6%)

of the subjects, Grade 4 ascites in 20(18.2%) of the patients and least was grade 1 ascites seen in 4(3.6%) of the patients.

Table 7: Distribution of patients by liver size and echo structure

Liver size, echotexture and surface	**n (%)**
Shrunken, coarse echotexture, surface irregularity	93 (84.5)
Normal, coarse echotexture, surface irregularity	17 (15.5)
Total	110 (100)

Figure 7: Distribution of patients by liver size and echo structure

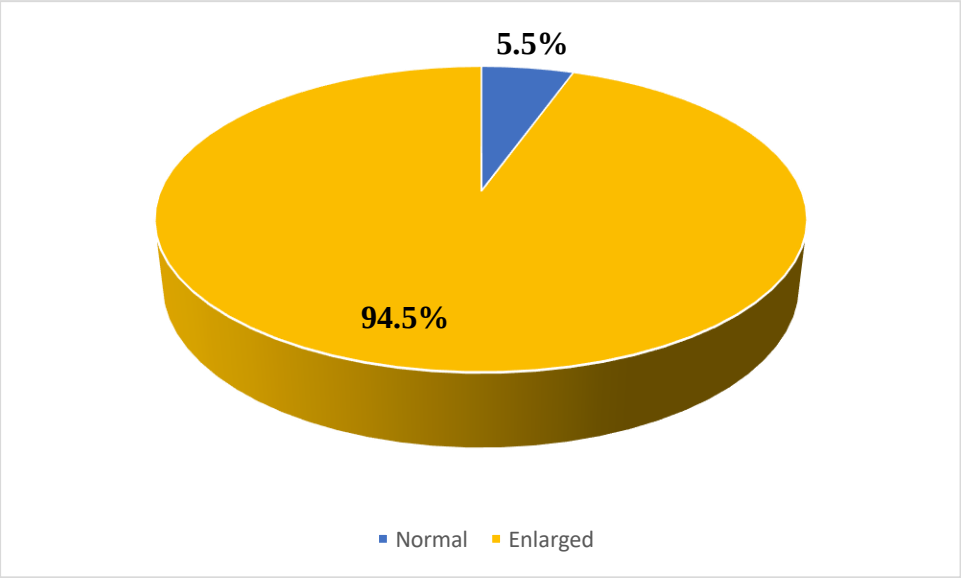


In the analysis, it was found that, 93(84.5%) of the patients had shrunken liver with coarse echotexture and surface irregularity while 17(15.5%) of the patients had normal sized liver with coarse echotexture and surface irregularity.

Table 8: Distribution of patients according to spleen diameter

Spleen diameter	n (%)
Normal	6 (5.5)
Enlarged	104 (94.5)
Total	110 (100)

Figure 8: Distribution of patients according to spleen diameter



Among the study subjects, 104(94.5%) of the patients had enlarged spleen and 6(5.5%) of the patients had normal spleen.

Figure 9: Distribution of patients according to spleen diameter

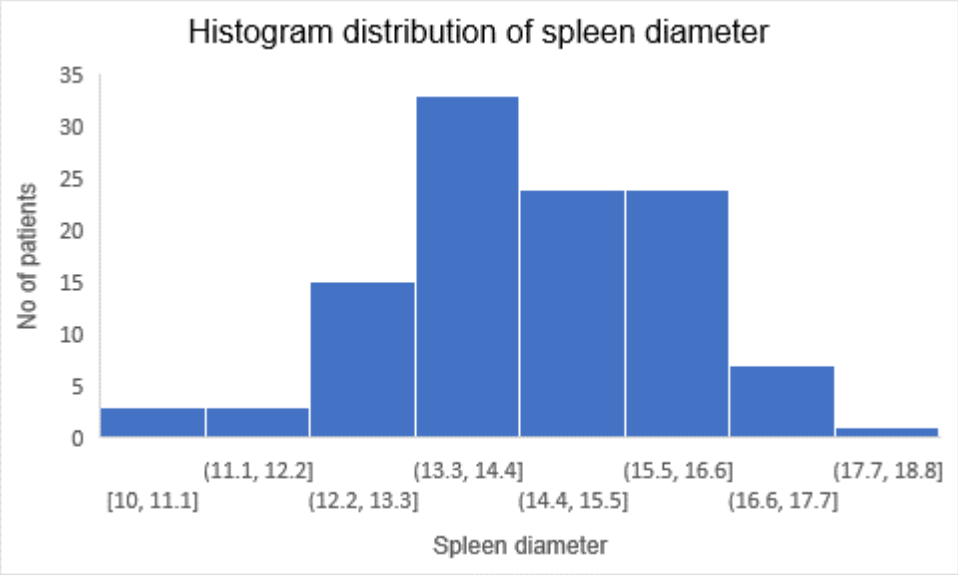


Figure 10: Distribution of individual patients according to spleen diameter

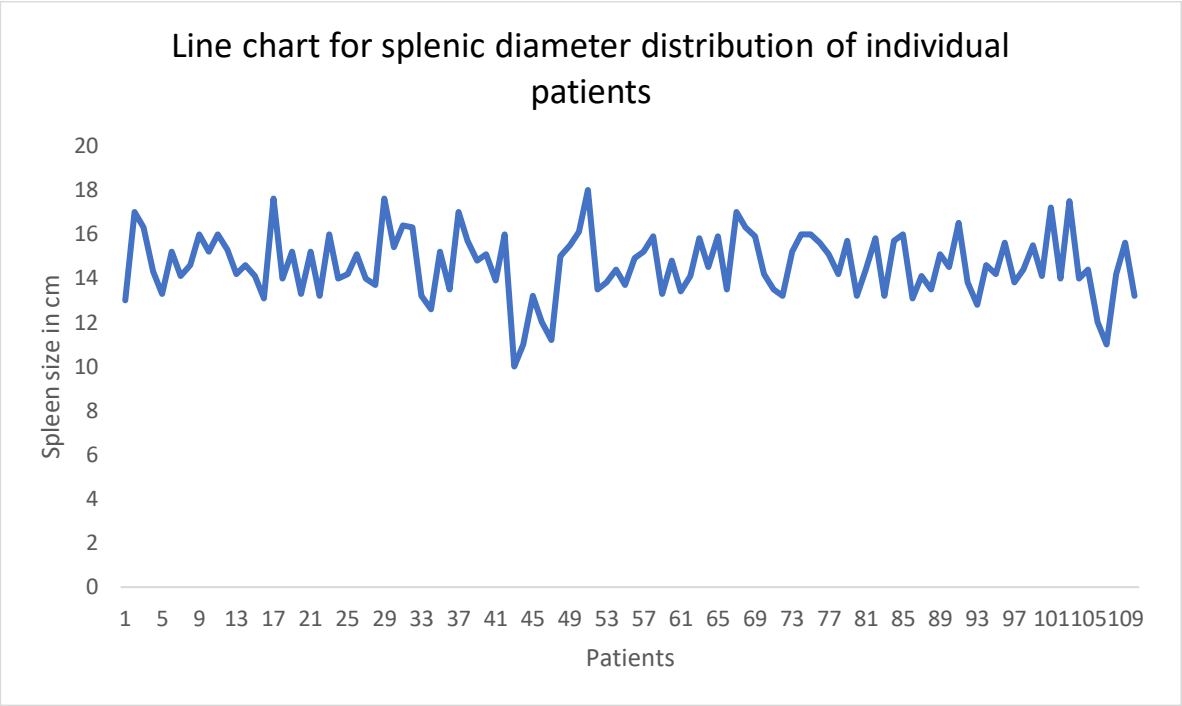


Figure 8 and 9 depicts distribution of the patients according to splenic diameter seen over a wide range. Figure 10 depicts distribution of individual patients according to spleen diameter

Table 9: CTP score wise distribution of patients

CTP class	n (%)
A	9 (8.2)
B	61 (55.5)
C	40 (36.4)
Total	110 (100)

Figure 11: CTP score wise distribution of patients

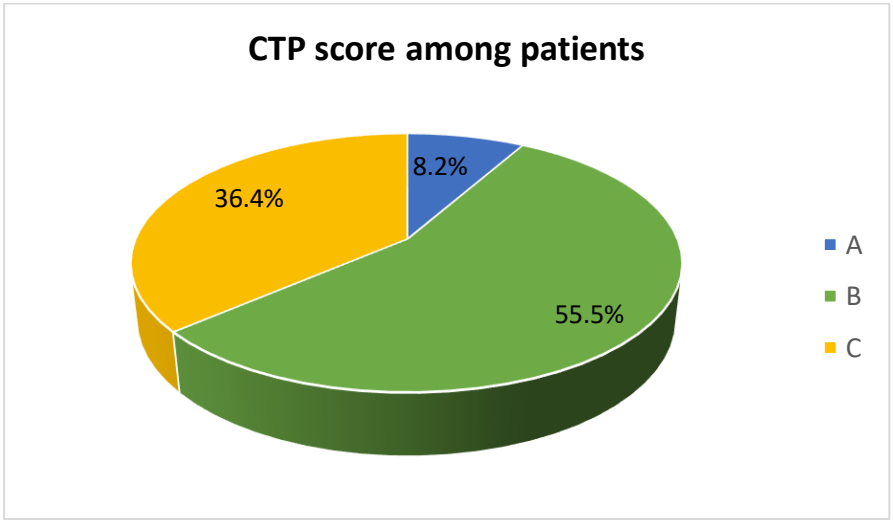


Table 9 and figure 11 reveal distribution of CTP score among patients. It was found that Child Turcotte Pugh class B is the most common, seen in 61(55.5%) of the patients,

followed by class C, seen in 40(36.4%) of the patients, and then class A seen in 9(8.2%) of the patients.

Table 10: Distribution of patients according to the platelet count

Platelets	**n (%)**
$<100\times10^3$	61 (55.5)
100×10^3-200×10^3	32 (29.1)
$>200\times10^3$	17 (15.5)
Total	110 (100)

Figure12: Distribution of patients according to the platelet count

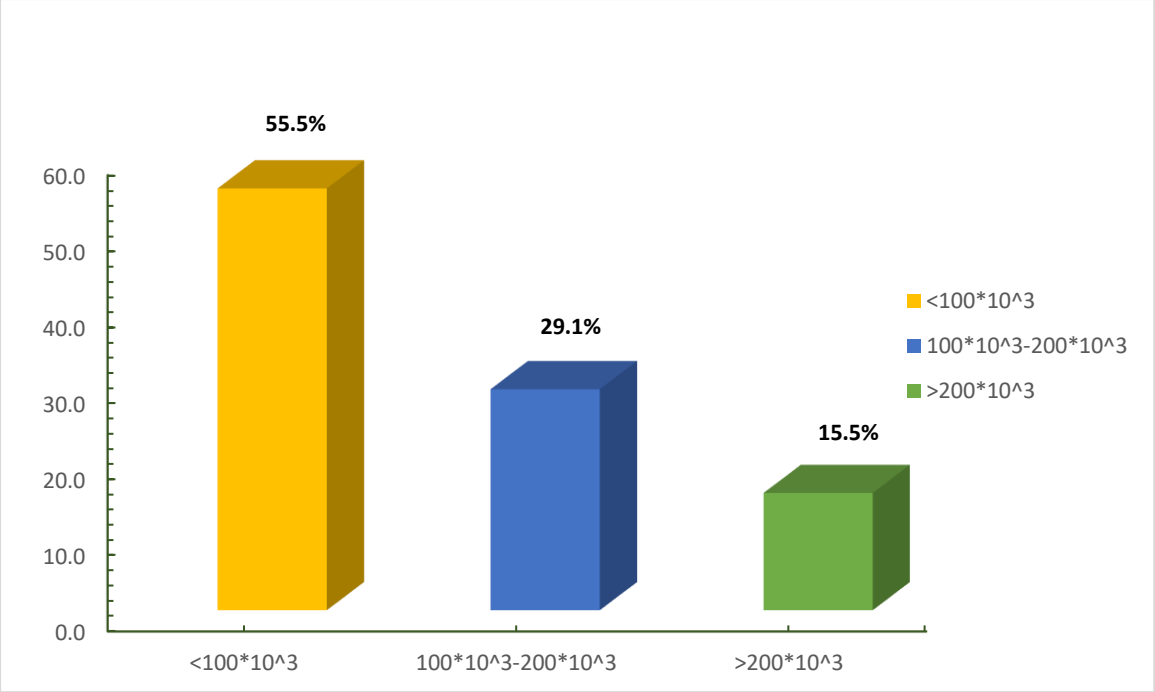


Figure 13: Distribution of platelet count in individual patients.

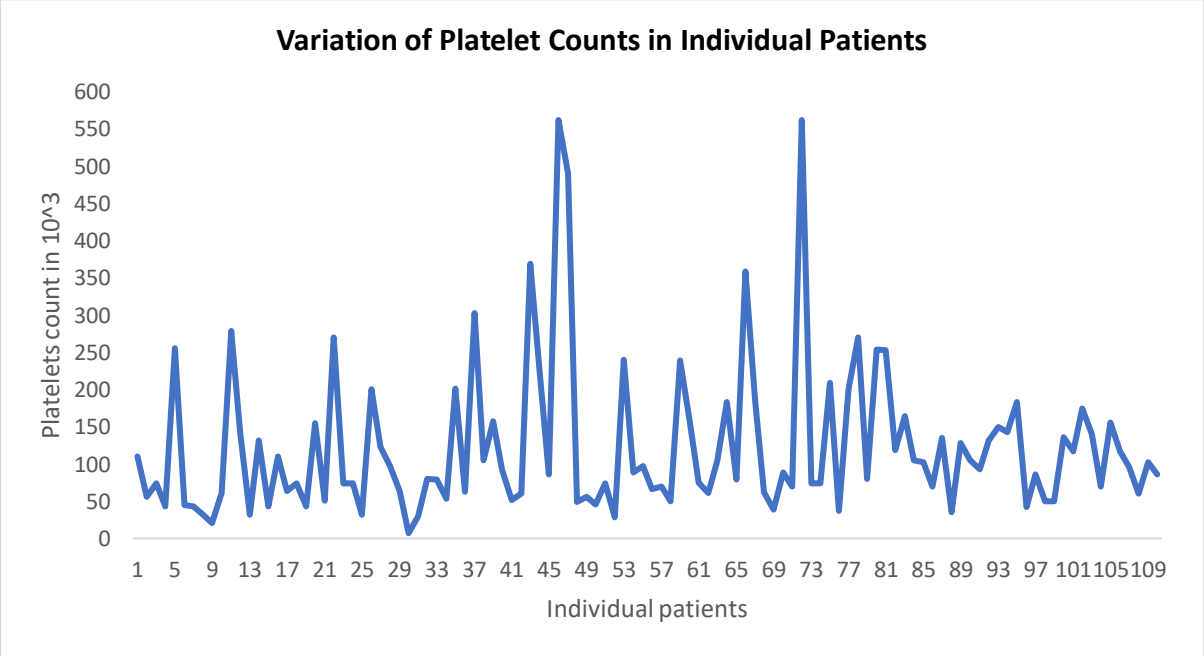


Table 10 and Figure 12 reveal distribution of patients according to platelet count. 61(55.5%) of the patients had platelet count less than 100 x 10^3 cells/microL, whereas, 32(29.1%) of the patients had platelet count between 100 x 10^3 cells/microL and 17(15.5%) of the people had platelet counts more than 200 x $10^{3.}$ Figure 13 reveals variation of platelet counts in individual patients.

Table 11: Distribution of patients according to presence of esophageal varices

	ESOPHAGEAL VARICES				
	Absent(n=30)		**Present(n=80)**		**p-value [1]**
	Median	**IQR**	**Median**	**IQR**	
Platelet count	174	144	74	56	<0.05*
PC/SD	1265	811	509	354	<0.05*
Spleen diameter	14	2	15	2	<0.05*

*[1] p-value is obtained by Mann-whitney U test

Table 9 shows descriptive analysis of distribution of patients according to presence of esophageal varices with respect to platelet count, spleen diameter and PC/SD ratio. The median PC/SD ratios were 1265 with IQR 811 and 509 with IQR 354 (p value <0.05) for patients who did not show evidence of esophageal varices on endoscopy and who did show evidence of varices respectively

The respective platelet count median were 174 x 10^3 cells/microL with IQR 144 x 10^3 cells/microL and 74 x 10^3 cells/microL with IQR 56 x 10^3 cells/microL (p value <0.05) in patients who did not and who did show evidence of esophageal varices respectively.

The respective median of spleen diameters were 140 cm with IQR 2 cm and 15 cm with IQR 2 cm (p value < 0.05).

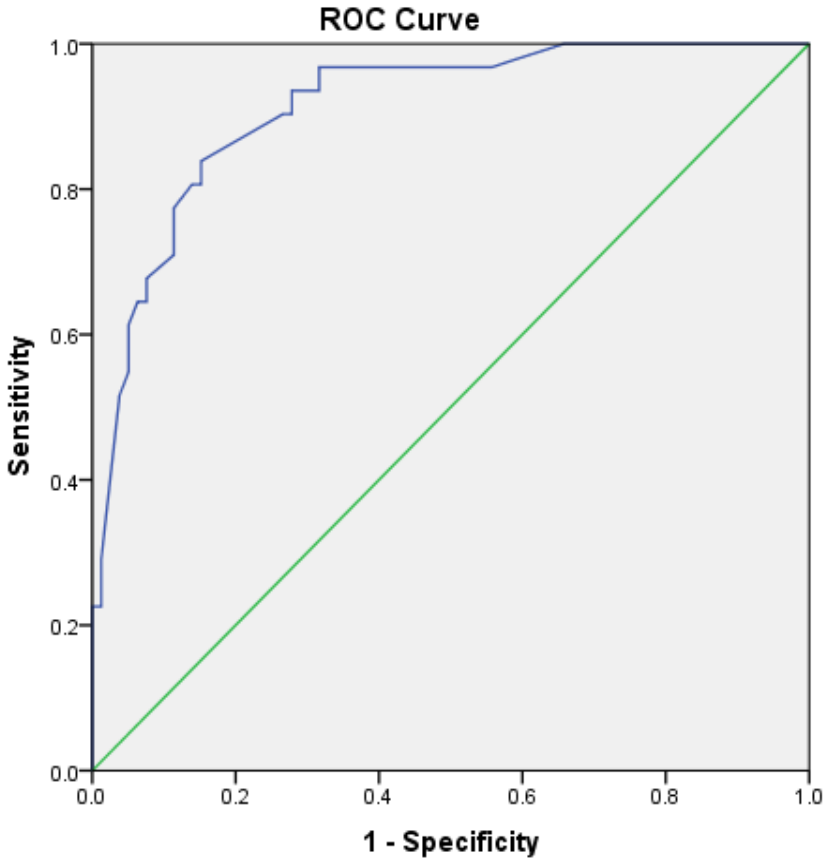
Table 12: Child-Turcotte-Pugh (CTP) Score wise distribution of patients

CTP score	**ESOPHAGEAL VARICES**		**p-value [2]**
	Absent	**Present**	
A	6 (20)	3 (3.8)	<0.001*
B	20 (66.7)	41 (51.3)	
C	4 (13.3)	36 (45)	

*[2] p-value is obtained by chi-square test

Table 10 shows correlation of CTP score with presence and absence of esophageal varices. Among patients with class A, varices were present in 3(3.8%) and absent in 6(20%) of the patients. Among class B subjects, varices were present in 41(51.3%) and absent in 20(66.7%). Among class C, varices were present in 36(45%) and absent in 4(13.3%) of the patients. This analysis is significant with p value of < 0.001.

Figure 14: ROC Curve of platelet count/diameter of the spleen ratio diagnostic test using the **cutoff value of ≤896** to predict the esophageal varices.



The AUC is 0.913, which makes the study significant.

Table 13: Descriptive analysis of PC/SD ratio

Sensitivity	Specificity	PPV	NPV	LR+	LR-
91.3	80.0	0.924	0.774	4.56	0.11

Table 14: Descriptive analysis of PC/SD ratio

EV		AUC	95% C. I		P value
Present	Absent		LL	UL	
73	24	0.913	0.857	0.969	<0.05

AUC=Area under curve

PPV=Positive predicted value

NPV=Negative predicted value

LR+=Positive likelihood ratio

LR-=Negative likelihood ratio

Table 13 and 14 reveal descriptive analysis of PC/SD ratio. The platelet count and spleen diameter ratio to determine esophageal varices, used in this study was

independent of the grade of varices and showed a cut off value of 896. This cut off value had a sensitivity of 91.3% and specificity of 80%. 73 patients within cutoff of 896 had esophageal varices and 24 patients did not. The positive and negative predictive values were 92% and 77.4% respectively. LR+ was 4.56 and LR- was 0.11.

DISCUSSION

A total of 110 patients with cirrhosis of the liver, were included in our study, who consulted Medicine OPD or were admitted in the Dept. of Medicine in "KLES Dr. Prabhakar Kore Hospital and Medical Research Centre" during the period January 2023 to December 2023. The objective of the study was "to validate whether the platelet count/spleen diameter ratio can be used to predict the presence of esophageal varices."

DISTRIBUTION

The mean age in our study population was 50.88 years. Maximum age was observed in the age group of 46-70 years (46.2%). This was in concordance with a study conducted by Gonzalez et al.,[1] where, mean age of 53.75 years was

found. Similarly, in another analysis conducted by Mattos A Z et al[3], the mean age was found to be 56.6 years.

In our study, etiology of cirrhosis of liver was found to be alcohol for 96(87.2%) of the patients, Hepatitis B for 8(7.2%) of the patients, NAFLD for 4(3.6%) of the patients and Wilson's disease for 2(1.8%) of the patients. This was in concordance with the study conducted by Gonzalez et al where the most common cause for cirrhosis in the people included in the study was alcohol 52.7%, whereas second most common cause was hepatitis c infection (26.3%), cryptogenic cirrhosis in 9.8% and primary biliary cirrhosis in 7.6%[1].

In general physical findings, 86 (78.1%) of the patients had pallor, 72(65.4%) of the patients had icterus, 45 (40.9%) of the patients had edema, whereas any of these were absent in 12 (10.9%) of the patients. In another study conducted by Wan-dong-hong, among the participants, 77% of the patients had pallor, 54% of the patients had icterus and 25% of the patients had edema as one of the general physical findings.

CHILD TURCOTT PUGH SCORE

In our study, it was observed, that distribution based on Child Turcotte Pugh (CTP) score : Class B was most common (55.5%). Significant correlation between CTP score and presence of esophageal varices ($p < 0.001$). In a study conducted by Gonzalez et al., the distribution based on Child-Pugh classification was Class A (17 patients), Class B (37 patients), and Class C (37 patients). In another study conducted by, 56.7 % patients belonged to Child Pugh class A, 39.3% belonged to class B and 4% patients belonged to class C[1].

PC/SD SCORE AND PRESENCE OF ESOPHAGEAL VARICES

In our study, PC/SD ratio cutoff of 896 had 91.3% sensitivity and 80% specificity for varices detection. Positive and negative predictive values were 92% and 77.4% respectively, positive and negative likelihood ratios were 4.56 and 0.11 respectively. This was in agreement with an analysis done by Mattos et al, who used a cutoff value of 909 for the ratio.

Similarly, in a study conducted by Payal et al., a PC/SD ratio cutoff value of 1054 was obtained, which concluded with PPV and NPV values with sensitivity and specificity of 97.14% and 100% respectively[2].

In another study conducted by Giannini et al., a cutoff value of 909 for the platelet count and spleen diameter ratio had a sensitivity of 90.5%, specificity of 67.0%, PPV of 76.6% and npv of 100% for diagnosing esophageal varices.[4]

Study conducted by Gonzalez et al., revealed a cutoff of < 884 for the platelet count and spleen diameter ratio had sensitivity of 84% and specificity of 70%, PPV of 94% and NPV of 40% for detecting esophageal varices, suggesting its potential utility in clinical practice.[1]

Study conducted by W W Baig et al(2014), assesses the diagnostic utility of platelet count to splenic diameter ratio for the same purpose, and derived a cutoff value of <1014 with a PPv of 95.4% and a NPV of 95.1%. the AUROC was 0.942(95% CI:0,890 to 0.995). This ratio was much larger than the ratio derived from our study.[14]

In another study conducted by Augustino Colli et al (2017), the aim was to compare the diagnositic accuracy of platelet count, spleen length and PC/SD ratio

against EGD in detecting varices. The key results were in concordance with our study, with PC/SD cutoff value of <909 was found to be highly accurate in identifying individuals at high risk of varices but should be used alongside EGD rather than replacing it.[12]

Another study conducted by Yang-Chun Du et al (2023), used PSR + PSVR (platelet count to splenic volume ratio) to predict esophageal varices in liver cirrhosis and found that this specific entity potentially offered superior diagnostic capability compared to PSR (platelet count to splenic diameter ratio) alone.

Hence more studies with larger sample size and in different subsets of population are needed to assess the authenticity of PC/SD as a prognostic marker.

CONCLUSION

110 patients were enrolled in our study based on diagnosis of cirrhosis of liver. The diagnosis was mainly done using ultrasound findings of the liver. Size of the spleen was measured as maximum bipolar diameter and readings of the same for each patient were recorded. Each of these patients was subjected to blood investigations and following which, platelet count to splenic diameter ratio was calculated.

This study was done to calculate platelet count / splenic diameter ratio (PC/SD) and to use it as a diagnostic marker for prediction of esophageal varices in cirrhosis of liver patients. The study was further validated by performing upper GI scopy for all the patients to look for presence or absence of esophageal varices. Our study revealed that

there is a significant positive correlation between PC/SD ratio and presence of esophageal varices, i.e., lesser the ratio, higher the chances of esophageal varices.

All the subjects in the study were male patients. The mean age in this study group was 50.88 years with standard deviation of 11.810, with maximum distribution in the age group of 46-70 years, 74(67.2%).

Among the study subjects, 96, (87.2%) of them were alcoholic and 14(12.7%) subjects were non alcoholic. In the study, the most common etiology of cirrhosis of liver was found to be alcohol for 87.2% of the patients, Hepatitis B for 7.2% of the patients, NAFLD for 3.6% of the patients and Wilson's disease for 1.8% of the patients.

Among the study population and analysis of grades of ascites, grade 2 ascites was most common, found in 44.5% of the subjects, followed by grade 3 ascites found in 33.6% of the subjects, Grade 4 ascites in 18.2% of the patients and least was grade 1 ascites seen in 3.6% of the patients.

In case of general physical findings, in our study, 78.1% of the patients had pallor, 65.4% of the patients had icterus, 40.9% of the patients had edema.

Signs of hepatic encephalopathy were seen in 75.4% of the patients, whereas, 24.5% of the patients didn't have any signs of hepatic encephalopathy.

In our analysis, it was found that, 84.5% of the patients had shrunken liver with coarse echotexture and surface irregularity while 15.5% of the patients had normal sized liver with coarse echotexture and surface irregularity.

Among the study subjects, 94.5% of the patients had enlarged spleen and 5.5% of the patients had normal spleen. When it comes to distribution of CTP score among patients.

It was found that Child Pugh class B is the most common, seen in 55.5% of the patients, followed by class C, seen in 36.4% of the patients, and then class A seen in 8.2% of the patients.

We also correlated the CTP score with presence of absence of esophageal varices and it revealed, among patients with class A, varices were present in 3(3.8%) and absent in 6(20%) of the patients. Among class B subjects, varices were present in 41(51.3%) and absent in 20(66.7%). Among class C, varices were present in 36(45%) and absent in 4(13.3%) of the patients. This amalysis is significant with p value of < 0.001.

In our study, the distribution of patients according to platelet count. 55.5% of the patients had platelet count less than 100 x 10 3 cells/microL, whereas, 29.1% of the patients had platelet count between 100 x 10 3 cells/microL and 15.5% of the people had platelet counts more than 200 x 10 3.

In our study, descriptive analysis of distribution of patients according to presence of esophageal varices with respect to platelet count, spleen diameter and PC/SD ratio. The median PC/SD ratios were 1265 with IQR 811 and 509 with IQR 354 (p value<0.05) for patients who did not show any evidence of esophageal varices on endoscopy when compared to those who did have varices respectively.

The respective platelet count median were 174 x 10 3 cells/microL with IQR 144 x 10 3 cells/microL and 74 x 10 3 cells/microL with IQR 56 x 10 3 cells/microL (p value <0.05) in patients who did not and who did show evidence of esophageal varices respectively.

The respective median of spleen diameters were 140 cm with IQR 2 cm and 15 cm with IQR 2 cm (p value < 0.05).We obtained a cut off value of 896 in our study for

ratio of platelet count to splenic diameter as a diagnostic test to predict esophageal varices and a ROC curve was developed using the same. The AUC was 0.913, which makes the study significant.

Our study was based on appraising PC/SD ratio as a prognostic marker for prediction of esophageal varices. The PC/SD ratio to detect esophageal varices, used in this study was independent of the grade of varices and showed a cut off value of 896. This cut off value had a sensitivity of 91.3% and specificity of 80%. 73 patients within cutoff of 896 had esophageal varices and 24 patients did not. The positive and negative predictive values were 92% and 77.4% respectively. 4.56 LR+ and 0.11 LR-.

Our study, found that there was a fair strength of reliability on the PC/SD ratio as a diagnostician marker for esophageal varices. Hence, a simple, non-invasive laboratory test such as PC/SD ratio, which has a significant association with esophageal varices in cirrhosis of liver, can be considered for use to predict esophageal varices in cirrhosis of liver in future clinical practice, specially in resource poor countries where endoscopy is still not available and such an invasive procedure can be avoided.

LIMITATIONS OF THE STUDY

This study has a few limitations-

1. The patients of cirrhosis were taken for the study using the ultrasonography technique instead of liver biopsy, which is the gold standard for the diagnosis of cirrhosis.

2. The patients who had thrombocytopenia were not worked up for other causes and hence it could be a confounding factor as platelet count is one of the parameters used to calculate the PC/SD ratio.
3. Need for validation for presence of esophageal varices using endoscopy.
4. Studies with larger sample pool are needed to establish a stronger relationship between PC/SD ratio and existence of esophageal varices.

STRENGTH OF THE STUDY

1. This analysis was conducted on 110 patients of liver cirrhosis, and we could strongly conclude that a significant positive correlation existed between PC/SD ratio and presence of esophageal varices in these patients.
2. The two most important parameters needed for the study , platelet count and the diameter of the spleen are routinely done, easily accessible in all patients of liver cirrhosis and hence is easier to derive and does not require out of the box tests to be done.
3. Therefore this test can be considered to be a potential marker that is not invasive to predict esophageal varices=in cirrhosis liver patients in future clinical practice, whenever resources for an invasive procedure like endoscopy are not available and the patient prefers not to undergo such invasive diagnostic procedures.

SUMMARY

Cirrhosis of liver is characterized by portal hypertension which mainly results in formation of esophageal varices and complications such as bleeding. Therefore cirrhosis of liver patients routinely undergo diagnostic and therapeutic endoscopy procedures for the same. Our study is based on using PC/SD ratio as a predictive marker for presence of esophageal varices in order to avoid invasive diagnostician procedures such as endoscopy and to compensate for its non-availability in resource poor regions.

The study involved 110 male patients with liver cirrhosis to assess the platelet count/splenic diameter ratio (PC/SD) as a diagnostic indicator for esophageal varices. Key findings include:

1. Correlation with Esophageal Varices:
- Lower PC/SD ratio was linked to a higher likelihood of esophageal varices.
2. Patient Demographics:
- Mean age was 50.88 years, with a majority in the 46-70 age group.
- Alcoholism was the primary cause of cirrhosis (87.2% of cases).
3. Clinical Features:
- Grade 2 ascites was most prevalent, followed by grades 3 and 4.
- 89.09% of the patients had findings of general physical examination such as pallor, icterus, edema or more than one.
4. Imaging and Lab Results:
- Most patients had a shrunken liver and an enlarged spleen.
- Child Pugh class B was the most common (55.5%).
- Platelet counts and PC/SD ratios significantly differed between patients with and without varices.

5. Diagnostic Marker Performance:

- PC/SD ratio cutoff of 896 had 91.3% sensitivity and 80% specificity for varices detection.
- Positive and negative predictive values were 92% and 77.4% respectively.

In conclusion, the study underscores the PC/SD ratio as a valuable and independent prognostic tool with high sensitivity and specificity for identifying esophageal varices in cirrhotic patients.

BIBLIOGRAPHY

1. González-Ojeda A, Cervantes-Guevara G, Chávez-Sánchez M, Dávalos-Cobián C, Ornelas-Cázares S, Macías-Amezcua MD, Chávez-Tostado M, Ramírez-Campos KM, Ramírez-Arce Adel R, Fuentes-Orozco C. Platelet count/spleen diameter ratio to predict esophageal varices in Mexican patients with hepatic cirrhosis. World J Gastroenterol. 2014 Feb 28;20(8):2079-84. doi: 10.3748/wjg.v20.i8.2079. PMID: 24616574; PMCID: PMC3934478.

2. Payal B, M S. Ratio of Platelet Count to Splenic Diameter as a Predictor Of Esophageal Varices in Cirrhosis of Liver. J Assoc Physicians India. 2022 Apr;70(4):11-12. PMID: 35443533.
3. Mattos AZ, Mattos AA, Vianna FF, Musskopf MI, Pereira-Lima JC, Maciel AC. Platelet count/spleen diameter ratio: analysis of its capacity as a predictor of the existence of esophageal varices. Arq Gastroenterol. 2010 Jul-Sep;47(3):275-8. doi: 10.1590/s0004-28032010000300012. PMID: 21140089.
4. Giannini E, Botta F, Borro P, Risso D, Romagnoli P, Fasoli A, Mele MR, Testa E, Mansi C, Savarino V, Testa R. Platelet count/spleen diameter ratio: proposal and validation of a non-invasive parameter to predict the presence of oesophageal varices in patients with liver cirrhosis. Gut. 2003 Aug;52(8):1200-5. doi: 10.1136/gut.52.8.1200. PMID: 12865282; PMCID: PMC1773759.
5. Schwarzenberger E, Meyer T, Golla V, Sahdala NP, Min AD. Utilization of platelet count spleen diameter ratio in predicting the presence of esophageal varices in patients with cirrhosis. J Clin Gastroenterol. 2010 Feb;44(2):146-50. doi: 10.1097/MCG.0b013e3181a745ff. PMID: 19593164.
6. Agha A, Anwar E, Bashir K, Savarino V, Giannini EG. External validation of the platelet count/spleen diameter ratio for the diagnosis of esophageal varices in hepatitis C virus-related cirrhosis. Dig DisSci 2009; 54: 654-60.
7. Chawla S, Katz A, Attar BM, Gupta A, Sandhu DS, Agarwal R. Platelet count/spleen diameter ratio to predict the presence of esophageal varices in patients with cirrhosis: a systematic review. Eur J Gastroenterol Hepatol. 2012;24:431–436.
8. Jamil Z, Malik M, Durrani AA. Platelet count to splenic diameter ratio and other noninvasive markers as predictors of esophageal varices in patients with liver cirrhosis.

Turk J Gastroenterol. 2017 Sep;28(5):347-352. doi: 10.5152/tjg.2017.17090. Epub 2017 Aug 4. PMID: 28776495.

9. Yu S, Chen W, Jiang Z (2021) Platelet count/spleen volume ratio has a good predictive value for esophageal varices in patients with hepatitis B liver cirrhosis. PLoS ONE 16(12): e0260774. https://doi.org/10.1371/journal.pone.0260774
10. Yoav Lurie, Muriel Webb, Ruth Cytter- Kuint, Shimmon Shteingart, Gerardo Z Lederkremer; Non invasive diagnosis of Liver Fibrosis and Cirrhosis, world Journal of Gastroenterology, November 7,2015.
11. Garcia-Tsao G, Sanyal AJ, Grace ND, Carey WD. Prevention and management of gastroesophageal varices and variceal hemorrhage in cirrhosis. Am J Gastroenterol. 2007;102:2086–2102.
12. Baig W. W., Nagaraja M. V., Varma M., Prabhu R. Platelet count to spleen diameter ratio for the diagnosis of esophageal varices: is it feasible? Canadian Journal of Gastroenterology . 2008;22(10):825–828. doi: 10.1155/2008/287274.
13. Colli, et al., Platelet count, spleen length, and platelet count-to-spleen length ratio for the diagnosis of oesophageal varices in people with chronic liver disease or portal vein thrombosis, Cochrane Database Syst. Rev. 4 (4) (2017) Cd008759.
14. Hong, Wd., Zhu, Qh., Huang, Zm. *et al.* Predictors of esophageal varices in patients with HBV-related cirrhosis: a retrospective study. *BMC Gastroenterol* **9**, 11 (2009).
15. James S. Dooley, Anna S.F. Lok, Guadalupe Garcia-Tsao, Massimo Pinzani; Sherlock's Diseases of the Liver and Biliary System.
16. Dooley JS, Burroughs AK, Heathcote EJ. Sherlock's disease of liver and biliary system, 12th edition, chapter 7-Hepatic cirrhosis; 2011:103.
17. Schiff ER, Maddrey WC, Reddy KR. Schiff's disease of liver, 11th Ed, Hepatic fibrosis; 2017:297-311.

18. Mark E Mailliard, Micheal F Sorell. Alcoholic Liver Disease. Harrison's Principles of Internal Medicine. 19th ed. New York. McGraw Hill; 2015:2052.
19. Kumar V, Abbas A, Aster J. Robbins textbook of basic pathology, 8th Ed; 2012:633-639.
20. de Franchis R. Revising consensus in portal hypertension: report of the Baveno V consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol. 2010;53:762–768.
21. Schepis F, Cammà C, Niceforo D, Magnano A, Pallio S, Cinquegrani M, D'amico G, Pasta L, Craxì A, Saitta A, et al. Which patients with cirrhosis should undergo endoscopic screening for esophageal varices detection. Hepatology. 2001;33:333–338.
22. Madhotra R, Mulcahy HE, Willner I, Reuben A. Prediction of esophageal varices in patients with cirrhosis. J Clin Gastroenterol. 2002;34:81–85.
23. Zein CO, Lindor KD, Angulo P. Prevalence and predictors of esophageal varices in patients with primary sclerosing cholangitis. Hepatology. 2004;39:204–210.
24. Zaman A, Becker T, Lapidus J, Benner K. Risk factors for the presence of varices in cirrhotic patients without a history of variceal hemorrhage. Arch Intern Med. 2001;161:2564–2570.
25. Riggio O, Angeloni S, Nicolini G, Merli M, Merkel C. Endoscopic screening for esophageal varices in cirrhotic patients. Hepatology. 2002;35:501–502.
26. Zimbwa TA, Blanshard C, Subramaniam A. Platelet count/spleen diameter ratio as a predictor of oesophageal varices in alcoholic cirrhosis. Gut. 2004;53:1055.
27. Sanyal AJ, Fontana RJ, Di Bisceglie AM, Everhart JE, Doherty MC, Everson GT, Donovan JA, Malet PF, Mehta S, Sheikh MY, et al. The prevalence and risk factors associated with esophageal varices in subjects with hepatitis C and advanced fibrosis. Gastrointest Endosc. 2006;64:855–864.

28. Chalasani N, Imperiale TF, Ismail A, Sood G, Carey M, Wilcox CM, Madichetty H, Kwo PY, Boyer TD. Predictors of large esophageal varices in patients with cirrhosis. Am J Gastroenterol. 1999;94:3285–3291.
29. Hong, Wd., Zhu, Qh., Huang, Zm. *et al.* Predictors of esophageal varices in patients with HBV-related cirrhosis: a retrospective study. *BMC Gastroenterol* **9**, 11 (2009). https://doi.org/10.1186/1471-230X-9-11
30. Zhang W, Wang L, Wang L, et al. Liver stiffness measurement, better than APRI, Fibroindex, Fib-4, and NBI gastroscopy, predicts portal hypertension in patients with cirrhosis. Cell Biochem Biophys 2015; 71: 865-73.
31. Chawla S, Katz A, Attar BM, Gupta A, Sandhu DS, Agarwal R. Platelet count/spleen diameter ratio to predict the presence of esophageal varices in patients with cirrhosis: a systematic review. Eur J Gastroenterol Hepatol 2012; 24: 431-6.
32. Lutz H. H., Gassler N., Tischendorf F. W., Trautwein C., Tischendorf J. J. Doppler ultrasound of hepatic blood flow for noninvasive evaluation of liver fibrosis compared with liver biopsy and transient elastography. Digestive Diseases and Sciences . 2012;57(8):2222–2230. doi: 10.1007/s10620-012-2153-0.
33. Fukui H., Saito H., Ueno Y., et al. Evidence-based clinical practice guidelines for liver cirrhosis 2015. Journal of Gastroenterology . 2016;51(7):629–650. doi: 10.1007/s00535-016-1216-y.
34. Dzeletovic I., Baron T. H. History of portal hypertension and endoscopic treatment of esophageal varices. Gastrointestinal Endoscopy . 2012;75(6):1244–1249. doi: 10.1016/j.gie.2012.02.052.
35. Afsar A., Nadeem M., Shah S. A. A., Hussain H., Rani A., Ghaffar S. Platelet count can predict the grade of esophageal varices in cirrhotic patients: a cross-sectional study. F1000Res . 2021;10:p. 101. doi: 10.12688/f1000research.28005.2.

36. Sm S., Tripathi B. K. Platelet count/spleen length ratio to predict the presence of esophageal varices in patients with cirrhosis. Journal of the Association of Physicians of India . 2022;70(4):11–12.
37. Ying L., Lin X., Xie Z. L., Hu Y. P., Shi K. Q. Performance of platelet count/spleen diameter ratio for diagnosis of esophageal varices in cirrhosis: a meta-analysis. Digestive Diseases and Sciences . 2012;57(6):1672–1681. doi: 10.1007/s10620-012-2058-y.
38. The general rules for recording endoscopic findings on esophageal varices. The Japanese Journal of Surgery . 1980;10(1):84–87. doi: 10.1007/BF02468653.
39. Abbasi A., Butt N., Bhutto A. R., Munir S. M. Correlation of thrombocytopenia with grading of esophageal varices in chronic liver disease patients. Journal of the College of Physicians and Surgeons–Pakistan . 2010;20(6):369–372.
40. Chang M. H., Sohn J. H., Kim T. Y., et al. Non-endoscopic predictors of large esophageal varices in patients with liver cirrhosis. The Korean Journal of Gastroenterology . 2007;49(6):376–383.
41. Nada L., Samira el F., Bahija B., Adil I., Nourdine A. Noninvasive predictors of presence and grade of esophageal varices in viral cirrhotic patients. The Pan African Medical Journal . 2015;20:p. 145. doi: 10.11604/pamj.2015.20.145.4320.
42. Shibata S, Umemura T, Yamazaki T, et al. Liver stiffness-spleen size-to-platelet ratio risk score identifies esophageal varices in Japanese patients with chronic hepatitis C. Hepatol Res. 2016;46(9):884–889. doi:10.1111/hepr.12631
43. O'Brien J, Triantos C, Burroughs AK. Management of varices in patients with cirrhosis. Nat Rev Gastroenterol Hepatol. 2013;10(7):402–412. doi:10.1038/nrgastro.2013.51
44. Thabut D, Moreau R, Lebrec D. Noninvasive assessment of portal hypertension in patients with cirrhosis. Hepatology. 2011;53(2):683–694.doi:10.1002/hep.24129

45. Petta S, Sebastiani G, Bugianesi E, et al. Non-invasive prediction of esophageal varices by stiffness and platelet in non-alcoholic fatty liver disease cirrhosis. J Hepatol. 2018;69(4):878–885. doi:10.1016/j.jhep.2018.05.019
46. Cherian JV, Deepak N, Ponnusamy RP, Somasundaram A, Jayanthi V. Non-invasive predictors of esophageal varices. Saudi J Gastroenterol. 2011;17(1):64–68. doi:10.4103/1319-3767.74470
47. Berzigotti A, Seijo S, Arena U, et al. Elastography, spleen size, and platelet count identify portal hypertension in patients with compensated cirrhosis. Gastroenterology. 2013;144(1):102–111.e101. doi:10.1053/j.gastro.2012.10.001
48. Crisan D, Radu C, Lupsor M, et al. Two or more synchronous combination of noninvasive tests to increase accuracy of liver fibrosis assessement in chronic hepatitis C; results from a cohort of 446 patients. Hepat Mon. 2012;12(3):177–184. doi:10.5812/hepatmon.853
49. European Association for the Study of the Liver. EASL-ALEH clinical practice guidelines: non-invasive tests for evaluation of liver disease severity and prognosis. J Hepatol. 2015;63(1):237–264. doi:10.1016/j.jhep.2015.04.006
50. Fukui H., Saito H., Ueno Y., et al. Evidence-based clinical practice guidelines for liver cirrhosis 2015. Journal of Gastroenterology. 2016;51(7):629–650. doi: 10.1007/s00535-016-1216-y.

ANNEXURES

ANNEXURE I- INFORMED CONSENT FORM

TITLE OF THE STUDY: "RATIO OF PLATELET COUNT TO SPLENIC DIAMETER AS A PREDICTOR OF OESOPHAGEAL VARICES IN CIRRHOSIS OF LIVER.- A CROSS SECTIONAL STUDY."

Registration no. of Student/Principal Investigator: BG0121016

Objective: To validate whether the platelet count/spleen diameter ratio can be used to predict the presence of esophageal varices.

Introduction:

Portal hypertension is the principal complication of hepatic cirrhosis(1). More than 80% of patients with cirrhosis will develop esophageal varices at some point, and 30% of these patients will have at least one bleeding episode because of rupture of a varix(2). Most first bleeding episodes happen during the first year after the detection of the varices, with a 5%-10% mortality attributed to the initial hemorrhage[3]. Today's guidelines are clear that there are no substitute markers to determine the presence and size of esophageal varices, and endoscopy is still the only valid method to investigate varices and is a fundamental part of the diagnostic work-up in patients with cirrhosis.[4]

Today's guidelines are clear that there are no substitute markers to determine the presence and size of esophageal varices, and endoscopy is still the only valid method to investigate varices. However, access to endoscopy and other resources is limited in some cities.

At any given time, a variable proportion of patients will not have varices, and the reported prevalence of esophageal varices is 24%-80%[5]. The use of noninvasive methods to predict the presence of esophageal varices would help restrict endoscopic studies to those with a high probability of having varices.

Explanation of procedure:

- Informed consent will be obtained and then patient will be enrolled for the study.
- All patients fulfilling inclusion criteria are subjected to a questionnaire and thorough clinical examination.

- Routine workup for cirrhosis of liver is done. Complete blood counts, including platelet count, coagulation profile will be done. Liver function tests including serum proteins. USG abdomen will be done.
- Child Pugh score will be calculated for each participant.
- Data will be analysed and tabulated.

Withdrawal from participation in the study: Participation in this study is voluntary. You will be free to decide whether to participate in this study or continue participation once enrolled. In case you decide to withdraw your participation, you are free to do so. However, please convey the decision to the principal investigator.

Possible benefits from participating in the study: You will/will not have nor get any benefits by participating in this study. The data gathered will help the population at large.

Possible risks from participating in the study: There are no risks involved in participating in this study.

Privacy and confidentiality: The information collected from you will be coded, to prevent any person from identifying you. Your identity will never be revealed. The data collected from you will be kept confidential and only processed or aggregated data will be used for publication.

Financial incentives: You will not receive any payment for participating in this study.

Authorization for publication of aggregated data: Results obtained after processing of the aggregated data will be published for scientific purposes and or presented to scientific groups. However, your identity will never be revealed.

Questions: In case of any questions with regard to this study, you are free to contact: Dept of General Medicine – Reg no: BG0121016 ” If you have any question or complaints with regard to your right as study participant you may contact Dr Harsha Hegde, Chairperson, Ethical committee of JNMC, 0831-2473777 Extension 4052.

Legal rights: By signing this consent form, we are not waving any of your legal rights.

Name of the participant:

Signature of the participant:

Date and time:

ANNEXURE II- PROFORMA

TITLE OF THE STUDY: "RATIO OF PLATELET COUNT TO SPLENIC DIAMETER AS A PREDICTOR OF OESOPHAGEAL VARICES IN CIRRHOSIS OF LIVER.- A CROSS SECTIONAL STUDY."

CASE NO:

NAME:

AGE/SEX:

IP NO.:

ADDRESS:

OCCUPATION:

CHIEF COMPLAINTS AT PRESENTATION:

Diagnosis:

Treatment history:

PHYSICAL EXAMINATION:

GENERAL CONDITION:

- PALLOR- YES/NO
- ICTERUS-YES/NO
- LYMPHADENOPATHY-YES/NO
- CYANOSIS- YES/NO
- CLUBBING-YES/NO
- EDEMA-YES/NO

VITALS:

- TEMPERATURE:
- PULSE:
- RESPIRATORY RATE:
- BLOOD PRESSURE:

SIGNS OF HEPATOCELLULAR FAILURE	SIGNS OF HEPATIC ENCEPHALOPATHY
Loss of auxiliary hair-	Asterixis-
Leuconychia-	Constructional apraxia-
Dupuytren's contracture-	Drowsiness-
Gynaecomastia-	Altered sensorium-
Spider naevi-	
Testicular atrophy-	
Ascites-	

SYSTEMIC EXAMINATION:

C.V.S.:

R. S.:

P.A.:

C.N.S.:

INVESTIGATIONS-

BLOOD PICTURE	**LIVER FUNCTION TEST**
HB -	T.BILIRUBIN -
WBC –	D. BILIRUBIN-
Platelet count-	I. BILIRUBIN
Neutrophils -	SGOT -
Lymphocytes -	SGPT -
Monocytes -	ALP-
Eosinophils -	TOTAL PROTEINS-
Basophils –	Sr. Albumin-
COAGULATION PROFILE	A/ G Ratio-
PT -	
APTT-	CTP score-
INR –	

USG ABDOMEN -

LIVER SIZE AND ECHO TEXTURE-

SPLEEN DIAMETER-

PORTAL VEIN DIAMETER AND COLLATERALS (IF ANY)-

ESOPHAGEAL VARICES ON ENDOSCOPY(IF ANY)-

ANNEXURE III- KEY TO MASTERCHART

- CLD- Chronic liver disease
- DCLD- Decompensated liver disease
- PHTN- Portal hypertension
- HE- Hepatic encephalopathy
- T2DM- Type 2 diabetes mellitus
- IHD- Ischemic heart disease
- PE- Pulmonary effusion
- PHTN- Portal hypertension
- HRS- Hepato renal syndrome
- UGI- Upper gastro intestinal
- AKI- Acute kidney injury
- SBP- Spontaneous bacterial peritonitis
- HPS- Hepato pulmonary syndrome
- RVD- Retro viral disease
- B/L- Bilateral
- NVBS- Normal vesicular breath sounds
- HB- Hemoglobin
- WBC- White blood cells
- T. BIL- Total bilirubin
- D. BIL- Direct bilirubin
- I. BIL- Indirect bilirubin

NAME	AGE/SE	DIAGNOSIS	GEN PHYSICAL EXAM	SIGNS OF HEPATOCELLULA	SIGNS OF HEPATIC ENCEPH	Ascites	RS	HB	WBC	PLATEL	PT	INR	T. BIL	I. BIL	D. BIL	SGOT	SGPT	ALP	ALBUM	LIVER SIZE AND ECHOTEXTURE	SPLEEN SIZE	ESOPHAGEAL VA	PC/SD	CTP score
Dayanand	55/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	9.2	10.6	110	1.3	1.34	3.2	1.1	2.1	76	51	119	2.6	Shrunken, coarse echotexture, surface irregul	13	Present	846.1	B
Irappa	60/M	CLD with PHTN with ascites, secondary to NAFLD in HE T2I	Pallor, icterus, edema	Present	Present	Grade 2 ascites	B/l nvbs	8.4	11.2	56	1.6	1.6	6.2	1.2	4.8	82	98	132	2.5	Shrunken, coarse echotexture, surface irregul	17	Present	329.4	B
Appasaheb	64/M	CLD with PHTN with ascites, secondary to Hepatitis B, recurr	Pallor, icterus, edema	Present	Present	Grade 3 ascites	Decreased air entry on right	8.4	8.2	74	1.92	1.9	14.3	6.1	8.2	107	46	122	2.2	Shrunken, coarse echotexture, surface irregul	16.3	Present	453.9	C
Salim	28/M	DCLD with PHTN with ascites, with SBP, in HE	Pallor, icterus, edema	Present	Present	Grade 4 ascites	B/l basal crepts	5.9	11.8	43	2	2.1	13.4	1.14	12.32	93	51	119	1.9	Shrunken, coarse echotexture, surface irregul	14.3	Present	300.6	C
Mallappa	46/M	DCLD with PHTN with ascites, in HRS, right pleural effusion	Pallor, icterus, edema	Present	Absent	Grade 2 ascites	B/l bvbs	9.3	7.4	255	1.9	1.9	28.3	9	19.3	107	46	122	2.2	Shrunken, coarse echotexture, surface irregul	13.3	Present	850.6	B
Anand	41/M	DCLD with PHTN with ascites, with UGI bleed in HE	Pallor, icterus, edema	Present	Present	Grade 3 ascites	B/l nvbs	8.2	11.4	45	2.4	2.42	18.9	7.4	11.5	136	38	60	2.6	Shrunken, coarse echotexture, surface irregul	15.2	Present	296	C
Anappa	65/M	CLD with PHTN with ascites, secondary to hepatitis b	Pallor, icterus,	Present	Absent	Grade 2 ascites	B/l nvbs	9.9	11.8	43	1.9	1.8	3.46	1.14	2.32	93	51	119	1.9	Shrunken, coarse echotexture, surface irregul	14.1	Absent	300.4	C
Kiran	37/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 3 ascites	B/l nvbs	9.1	4.8	32	2.4	2.58	6.2	2.1	4.1	136	63	89	3.1	Normal, coarse echotexture, surface irregular	14.6	Absent	219.1	B
Shrikant	34/M	Dcld, with PHtn with ascites, in HRS	Pallor, icterus, edema	Present	Absent	Grade 4 ascites	Decreased air entry on right	7	12.1	21	2.8	2.83	17.8	4.9	12.3	65	37	47	2.4	Normal, coarse echotexture, surface irregular	16	Present	131.2	B
Yallappa	55/M	DCLD with PHTN with ascites, with right pleural effusion, in	Pallor, icterus	Present	Present	Grade 3 ascites	Decreased air entry on right	8.9	4.7	61	1.4	1.5	4.5	2.06	2.4	60	25	122	2.9	Shrunken, coarse echotexture, surface irregul	15.2	Present	401.3	C
Raniprakasl	39/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	8.8	20	279	1.3	1.34	14.6	4.6	10	129	29	221	2.9	Normal, coarse echotexture, surface irregular	16	Present	1743	A
Lacchappa	55/M	CLD with PHTN with ascites with left pleural effusion	Pallor, icterus	Present	Absent	Grade 4 ascites	Decreased air entry on right	9.2	4.7	142	3.4	3.59	4.5	1.006	3.4	60	25	122	2.7	Shrunken, coarse echotexture, surface irregul	15.3	Absent	928.1	B
Shivaling	52/M	DCLD with PHTN in HRS	Pallor, icterus, edema	Present	Absent	Grade 2 ascites	B/l nvbs	8	12.1	32	2.6	2.6	7.8	2.9	4.8	65	37	99	2.4	Shrunken, coarse echotexture, surface irregul	14.2	Present	225.3	B
Kallappa	48/M	CLD with pHTN with ascites	Pallor	Present	Absent	Minimal ascites	B/l nvbs	10.1	4.8	132	1.4	1.4	2.8	0.8	1	136	88	240	3.1	Normal, coarse echotexture, surface irregular	14.6	Absent	904.1	B
Kistappa	65/M	DCLD with PHTN with ascites with UGI bleed, in HE	Pallor, icterus, edema	Present	Present	Grade 4 ascites	B/l nvbs	6.2	11.8	43	1.9	1.9	3.46	1.14	2.32	93	51	119	1.9	Shrunken, coarse echotexture, surface irregul	14.1	Present	304.9	C
Balappa	60/M	CLD with PHTN with ascites	Icterus	Present	Absent	Grade 2 ascites	B/l nvbs	11.2	10.6	110	1.3	1.34	3.2	1.1	2.1	76	51	119	2.9	Shrunken, coarse echotexture, surface irregul	13.1	Absent	839.6	B
Fakeerappa	60/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	8.4	7.2	64	1.62	1.6	3.4	1.2	2.3	182	98	232	3	Shrunken, coarse echotexture, surface irregul	17.6	Present	363.6	B
Annargoud	64/M	CLD with PHTN with ascites with recurrent pleural effusion	Pallor	Present	Absent	Grade 3 ascites	Decreased air entry on right	8.4	8.2	74	1.6	1.6	2.3	0.3	1	107	46	122	2.9	Shrunken, coarse echotexture, surface irregul	14	Absent	528.5	B
Laxman	30/M	DCLD with PHTN with ascites with SBP in HE	Pallor, icterus, edema	Present	Present	Grade 3 ascites	B/l basal crepts	7.2	11	43	2	1.99	13.4	1.14	12.3	93	51	220	1.9	Shrunken, coarse echotexture, surface irregul	15.2	Present	282.8	C
Balappa	52/M	DCLD with PHTN with ascites, in AKI , in HE	Pallor, icterus, edema	Present	Present	Grade 2 ascites	B/l basal crepts	9.3	7.4	155	1.9	1.98	17.3	8	9.3	107	46	186	2.2	Shrunken, coarse echotexture, surface irregul	13.3	Present	1165	C
Mahadev	46/M	CLD with PHTN with ascites with acute pancreatitis	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	10.6	4.7	51	2.4	2.5	8.5	3.6	4.4	60	25	156	3.1	Shrunken, coarse echotexture, surface irregul	15.2	Present	335.5	B
Babu	41/m	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	11	12	270	1.3	1.3	24.6	6.9	17.7	346	144	277	2.8	Shrunken, coarse echotexture, surface irregul	13.2	Absent	2045	A
Iranna	57/M	CLD with PHTN secondary NAFLD, T2DM, IHD	Absent	Present	Absent	Grade 3 ascites	B/l nvbs	11.4	9.7	74	1.28	1.29	1.43	0.46	0.97	42	32	157	2.7	Shrunken, coarse echotexture, surface irregul	16	Present	462.5	B
Nagappa	64/M	CLD with PHTN with ascites with recurrent pleural effusion	Pallor	Present	Absent	Grade 3 ascites	Decreased air entry on right	8.4	8.2	74	1.6	1.6	2.3	0.3	1	107	46	122	2.9	Shrunken, coarse echotexture, surface irregul	14	Present	528.5	B
Ramappa	52/M	DCLD with PHTN with ascites in HRS	Pallor, icterus, edema	Present	Absent	Grade 2 ascites	B/l nvbs	8	12.1	32	2.6	2.6	7.8	2.9	4.8	65	37	99	2.4	Shrunken, coarse echotexture, surface irregul	14.2	Present	228.5	C
Laxman	41/M	CLD with PHTN with ascites with UGI bleed with acute pancr	Pallor, icterus, edema	Present	Present	Grade 4 ascites	B/l nvbs	7.1	15.8	200	2.28	2.26	6.9	0.25	6.72	56	26	101	2.4	Shrunken, coarse echotexture, surface irregul	15.1	Present	1324	C
Praveen	24/M	CLD with PHTN with Ascites sec to wilson's disease	Icterus	Present	Absent	Grade 2 ascites	B/l nvbs	14.2	5.2	123	2.6	2.6	32.4	6	26.4	569	725	200	3.5	Shrunken, coarse echotexture, surface irregul	14	Absent	1065.3	B
Basavraj	64/M	CLD with PHTN with ascites secondary NAFLD, T2DM	Pallor, icterus, edema	Present	Absent	Grade 4 ascites	B/l nvbs	8.6	26	97	1.44	1.44	2.4	1.1	1.2	29	14	70	3	Normal, coarse echotexture, surface irregular	13.7	Present	708	B
Mahesh	60/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	8.4	7.2	64	1.62	1.6	3.4	1.2	2.3	182	98	232	3	Shrunken, coarse echotexture, surface irregul	17.6	Present	363.6	B
Adappa Har	56/M	CLD with PHTN with ascites	Pallor, icterus, pedal edema	Present	Absent	Grade 2 ascites	B/l nvbs	9.3	4800	7	1.3	1.48	4.03	2.8	1.23	105	41	367	2.7	Shrunken, coarse echotexture, surface irregul	15.4	Present	754.2	C
Shivaling	59/M	CLD with PHTN with ascites	Pallor, edema	Present	Absent	Grade 3 ascites	B/l nvbs	9.2	2600	30	2.15	2.13	4.26	2.38	1.8	39	22	85	2.4	Shrunken, coarse echotexture, surface irregul	16.4	Present	182.9	C
Basvaraj	43/M	DCLD with PHTN with ascites, in HRS, in HE	Pallor, icterus, edema	Present	Present	Grade 4 ascites	Decreased air entry on right	8.8	14.8	80	2.8	2.9	8.42	5.7	2.7	45	21	95	1.8	Shrunken, coarse echotexture, surface irregul	16.3	Present	490.7	C
Ramachand	65/M	DCLD with PHTN with ascites, SBP, in HRS, in HE	Pallor, icterus, edema	Present	Present	Grade 3 ascites	B/l basal crepts	8.5	13.3	79	1.62	1.62	6.7	2.25	4.47	46	25	101	3.1	Shrunken, coarse echotexture, surface irregul	13.2	Present	598.4	C
Shivaling	22/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	10.5	4.5	53	2.2	2.2	16.9	2.9	14	168	145	188	3.4	Shrunken, coarse echotexture, surface irregul	12.6	Present	420.6	B
Chumappa	39/M	CLD with PHTN with ascites	Pallor, edema	Present	Absent	Grade 2 ascites	B/l nvbs	8.4	10	201	1.39	1.39	2.41	0.94	1.47	43	15	138	3	Shrunken, coarse echotexture, surface irregul	15.2	Absent	1322	A
Rajesh	57/M	DCLD with PHTN with ascites with UGI bleed	Pallor	Present	Absent	Grade 3 ascites	B/l nvbs	7.7	8.7	63	1.57	1.57	2.56	1.38	1.18	93	34	68	2.2	Shrunken, coarse echotexture, surface irregul	13.5	Present	466.6	B
Ajit	46/M	DCLD with phtn withn ascites, SBP, in HRS	Pallor, icterus, edema	Present	Absent	Grade 2 ascites	B/l nvbs	6.2	15.9	303	1.62	1.62	0.56	0.26	0.3	17	16	130	2.8	Shrunken, coarse echotexture, surface irregul	17	Present	745.9	A
Dundappa	55/M	CLD with PHTN with ascites with acute pancreatitis	Absent	Present	Absent	Grade 3 ascites	B/l nvbs	12.2	7.5	105	1.57	1.57	1.79	0.27	1.52	11	12	175	3	Shrunken, coarse echotexture, surface irregul	15.7	Present	668.7	B
Tangayya	67/M	CLD with PHTN with ascites, with SBP, right pleural effusion	Pallor, icterus, edema	Present	Present	Grade 3 ascites	Decreased air entry on right	9.7	30	157	1.59	1.56	4.4	1.2	3.2	111	77	164	3.1	Shrunken, coarse echotexture, surface irregul	14.8	Present	1060	C
Manohar	69/M	DCLD with PHTN with ascites	Pallor, icterus, edema	Present	Absent	Grade 4 ascites	B/l basal crepts	10.1	5.6	92	2.1	2.1	2.4	1	1.2	66	52	159	2.4	Shrunken, coarse echotexture, surface irregul	15.1	Present	609.2	B
Prakash	44/M	CLD with PHTN with ascites with SBP	Edema	Present	Absent	Grade 3 ascites	B/l basal crepts	10.7	22.3	52	1.7	1.7	1.6	0.3	1.3	56	44	128	2.6	Shrunken, coarse echotexture, surface irregul	13.9	Present	374.1	B
Shankar	55/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	11.2	4	60	1.69	1.69	5.2	2.1	3.1	156	89	221	3	Shrunken, coarse echotexture, surface irregul	16	Present	375	B
Ballappa pa	53/M	CLD with PHTN with ascites	Pallor, edema	Present	Absent	Grade 2 ascites	B/l nvbs	9.9	7.2	369	1.2	1.24	0.78	0.47	0.31	27	12	78	3.8	Shrunken, coarse echotexture, surface irregul	10	Absent	369	B
Sham rama	58/M	CLD with PHTN with ascites	Pallor, icterus, edema	Present	Absent	Grade 3 ascites	B/l nvbs	9	4.3	220	1.4	1.3	3.48	1.88	1.6	42	23	95	2.9	Shrunken, coarse echotexture, surface irregul	11	Absent	1200	B
Maruti doda	58/M	CLD with PHTN with ascites secondary to hepatitis b	Pallor, icterus, edema	Present	Absent	Grade 3 ascites	B/l nvbs	9	9.2	86	1.6	1.6	2.53	1.64	0.89	67	41	124	2.4	Normal size, coarse echotexture, surface irreg	13.2	Present	651.5	B
Ishwar gom	48/M	CLD with PHTN with ascites with SBP, pancreatic ascites	Edema	Present	Absent	Grade 3 ascites	B/l nvbs	14.5	12.2	562	1.15	1.1	0.63	0.14	0.49	83	54	281	3.2	Shrunken, coarse echotexture, surface irregul	12	Absent	1468	B
Somshekha	78/M	CLD with PHTN with ascites with CA colon	Pallor, icterus, edema	Present	Absent	Grade 3 ascites	B/l nvbs	5.5	5.3	490	1.1	1.16	0.25	0.16	0.09	15	12	74	2.2	Shrunken, coarse echotexture, surface irregul	11.2	Absent	1859	B
Bhima nanc	49/M	CLD with PHTN with ascites in HRS	Pallor, icterus, edema	Present	Absent	Grade 4 ascites	B/l nvbs	7.8	15.8	49	1.6	1.6	5.3	1.3	4	134	82	195	1.8	Shrunken, coarse echotexture, surface irregul	15	Present	326.6	B
Shivanand	38/M	CLD with PHTN with ascites in HRS	Icterus, edema	Present	Absent	Grade 2 ascites	B/l basal crepts	11.3	4.8	56	2.2	2.27	5.92	5.75	0.17	184	43	200	2.4	Shrunken, coarse echotexture, surface irregul	15.5	Present	361.2	B
Gopal sana	37/M	CLD with PHTN with ascites	Absent	Present	Absent	Grade 2 ascites	B/l basal crepts	11.6	3.9	46	1.57	1.57	3.53	2.35	1.18	32	14	166	3.6	Normal size, coarse echotexture, surface irreg	16.1	Present	285.7	C
Sangamesh	20/M	CLD with PHTN with Ascites sec to wilson's disease	Icterus	Present	Absent	Grade 2 ascites	B/l nvbs	13.1	4	74	2.6	3.08	29.8	24.3	5	583	658	198	3.5	Shrunken, coarse echotexture, surface irregul	18	Absent	411.1	B
Nagesh	56/M	CLD with PHTN with ascites	Pallor	Present	Absent	Grade 2 ascites	B/l nvbs	8.3	3.1	28	1.73	1.72	3.07	1.94	1.13	39	17	86	3.4	Normal size, coarse echotexture, surface irreg	13.5	Present	207.4	A
Madivalapp	41/M	CLD with PHTN with ascites with acute pancreatitis, old koch	Pallor	Present	Absent	Grade 3 ascites	Decreased air entry on right	9.1	14	240	1.7	1.69	3.01	2.58	0.43	78	41	184	2.1	Shrunken, coarse echotexture, surface irregul	13.8	Absent	1739	A
Madiwalapp	50/M	DCLD with PHTN with ascites	Pallor, icterus, edema	Present	Present	Grade 3 ascites	B/l nvbs	8.8	5.4	89	1.5	1.5	4.8	1.4	3.4	89	44	220	3	Shrunken, altered echotexture, surface irregul	14.4	Present	618	C
Kalappa	64/M	CLD with PHTN with ascites secondary to hepatitis B, T2DM	Pallor, icterus, edema	Present	Absent	Grade 4 ascites	B/l nvbs	8.6	26	97	1.44	1.44	2.4	1.1	1.2	29	14	70	3	Normal, coarse echotexture, surface irregular	13.7	Present	708	B
Shivananda	47/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	10	2.9	66	1.62	1.6	3.98	1.36	2.62	62	28	115	2.2	Shrunken, coarse echotexture, surface irregul	14.9	Present	442.9	B
Shankargou	56/M	CLD with PHTN with ascites	Absent	Present	Absent	Grade 3 ascites	B/l nvbs	11.2	6.2	70	1.56	1.55	2.34	0.63	1.71	45	27	151	2.5	Shrunken, coarse echotexture, surface irregul	15.2	Present	460.5	B
Raju	47/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 4 ascites	B/l nvbs	7.4	6.7	50	2.55	2.52	7.17	2.8	4.3	35	29	170	2.3	Shrunken, coarse echotexture, surface irregul	15.9	Present	314.4	B
Vishwanath	65/M	CLD with PHTN with ascites	Pallor, icterus, edema	Present	Absent	Grade 2 ascites	B/l nvbs	9.3	16.6	239	2	1.99	25.7	5.6	20.1	87	28	154	2.5	Shrunken, coarse echotexture, surface irregul	13.3	Absent	1796	A
Vijay	39/M	CLD with PHTN with ascites, with SBP, right pleural effusion	Pallor, icterus, edema	Present	Present	Grade 3 ascites	Decreased air entry on right	9.7	30	157	1.59	1.56	4.4	1.2	3.2	111	77	164	3.1	Shrunken, coarse echotexture, surface irregul	14.8	Present	822.6	C
Bhartesh	50/M	CLD with PHTN with ascites with right pleural effusion	Pallor, icterus	Present	Present	Grade 2 ascites	Decreased air entry on right	12.9	6.1	75	1.79	1.8	4.8	1.5	3.3	95	53	151	2.8	Shrunken, coarse echotexture, surface irregul	13.4	Absent	1559.4	C
Mahadev	48/M	CLD with PHTN with ascites	Absent	Present	Absent	Grade 3 ascites	B/l nvbs	11	6.5	61	1.6	1.6	1.4	0.49	1	65	38	166	1.7	Shrunken, coarse echotexture, surface irregul	14.1	Present	432.6	B
Ramesh	59/M	CLD with PHTN with ascites	Icterus	Present	Absent	Grade 2 ascites	B/l nvbs	13.2	10.4	104	2	2.04	10.2	0.2	10	77	19	96	3	Shrunken, coarse echotexture, surface irregul	15.8	Present	658.2	B
Dadasaheb	60/M	DCLD with PHTN with ascites in HE	Pallor, edema	Present	Present	Grade 4 ascites	B/l nvbs	8.8	8.7	183	1.48	1.5	2.2	0.9	1.28	31	16	111	2.6	Normal, coarse echotexture, surface irregular	14.5	Present	654.2	C
Maruti	58/M	CLD with PHTN with ascites	Absent	Present	Absent	Grade 2 ascites	B/l nvbs	11.5	5	79	1.5	1.5	2.7	1.56	1.14	68	38	109	2.9	Shrunken, coarse echotexture, surface irregul	15.9	Present	496	B
Mahesh	48/M	CLD with PHTN with ascites in SBP	Pallor, icterus	Present	Absent	Grade 3 ascites	B/l nvbs	10.3	22	359	1.9	1.9	7.62	1.52	6.1	94	23	154	2.7	Shrunken, coarse echotexture, surface irregul	13.5	Absent	2659	A
Rama	68/M	DCLD with PHTN with ascites with UGI bleed	Pallor	Present	Absent	Grade 2 ascites	B/l nvbs	7.4	8.9	187	1.19	1.19	1.6	1.11	0.53	21	9	84	3	Shrunken, coarse echotexture, surface irregul	17	Present	870.5	B
Gangadhar	47/M	CLD with PHTN with ascites with RVD positive	Pallor, icterus	Present	Absent	Grade 3 ascites	B/l nvbs	9.4	8.7	62	3.3	3.26	9.07	1.08	7.9	69	38	86	3.2	Shrunken, coarse echotexture, surface irregul	16.3	Present	380.3	B
Shivaling	59/M	CLD with PHTN with ascites	Absent	Present	Absent	Grade 2 ascites	B/l nvbs	10.4	4.9	39	2.2	2.24	2.8	0.4	1.4	82	27	112	2.3	Shrunken, coarse echotexture, surface irregul	15.9	Present	245.2	B
Iranna	42/M	CLD with PHTN with ascites with hepatitis B	Pallor	Present	Absent	Grade 2 ascites	B/l nvbs	6.6	7.1	89	1.5	1.5	3.05	0.77	2.7	45	16	89	3	Shrunken, coarse echotexture, surface irregul	14.2	Present	626.7	B
Nagesh	54/M	CLD with PHTN with ascites with UGI bleed	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	7.9	5.2	70	1.7	1.7	3.29	0.93	2.36	95	42	200	2.6	Shrunken, coarse echotexture, surface irregul	13.5	Present	518.5	B
Ishwar	58/M	CLD with PHTN with ascites with SBP	Absent	Present	Absent	Grade 2 ascites	B/l nvbs	14.5	10.2	562	1.2	1.2	0.63	0.49	0.14	83	54	281	3.2	Shrunken, coarse echotexture, surface irregul	13.2	Absent	1506.8	A
Thotayya	52/M	DCLD with PHTN with ascites with peritoneal TB	Pallor, icterus	Present	Absent	Grade 4 ascites	B/l nvbs	9	11.4	74	1.67	1.67	5.2	2.1	3.1	44	25	133	2.2	Shrunken, coarse echotexture, surface irregul	15.2	Present	486.8	B
Sunil	57/M	CLD with PHTN secondary NAFLD, T2DM, IHD	Absent	Present	Absent	Grade 3 ascites	B/l nvbs	11.4	9.7	74	1.28	1.29	1.43	0.46	0.97	42	32	157	2.7	Shrunken, coarse echotexture, surface irregul	16	Present	462.5	B
Manjunath	31/M	DCLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	10.2	10.3	209	1.63	1.6	18.6	3.7	14.9	71	36	153	2.6	Shrunken, coarse echotexture, surface irregul	16	Absent	1306	B
Sachin	41/M	DCLD with PHTN with ascites with UGI bleed	Pallor	Present	Absent	Grade 3 ascites	B/l nvbs	6.2	4.7	37	1.3	1.37	2.35	0.5	1.85	89	22	95	2.9	Shrunken, coarse echotexture, surface irregul	15.6	Present	237.1	B
Madivalapp	41/M	CLD with PHTN with ascites with UGI bleed with acute pancr	Pallor, icterus, edema	Present	Present	Grade 4 ascites	B/l nvbs	7.1	15.8	200	2.28	2.26	6.9	0.25	6.72	56	26	101	2.4	Shrunken, coarse echotexture, surface irregul	15.1	Present	752.4	C
Ramling	60/M	CLD with PHTN with ascites with UGI bleed	Pallor	Present	Absent	Grade 4 ascites	B/l nvbs	6.2	27	270	2.2	2.21	2.15	0.7	1.45	47	24	88	2.5	Shrunken, coarse echotexture, surface irregul	14.2	Absent	1901	B
Vitthal	40/M	CLD with PHTN with ascites with UGI bleed	Pallor, edema	Present	Absent	Grade 3 ascites	B/l nvbs	8.8	5.2	80	1.9	1.9	6.4	1.5	4.9	99	45	169	2.6	Shrunken, coarse echotexture, surface irregul	15.7	Absent	960.4	B
Huvappa	44/M	CLD with PHTN with ascites	Pallor	Present	Absent	Grade 2 ascites	B/l nvbs	9.1	7	254	1.7	1.7	3.19	1.58	1.61	38	24	69	3	Shrunken, coarse echotexture, surface irregul	13.2	Absent	1924	B
Ramesh	52/M	CLD with PHTN with ascites	Pallor	Present	Absent	Grade 4 ascites	B/l nvbs	13	7.8	253	1.19	1.19	2.67	1.03	1.62	40	25	215	3.4	Shrunken, coarse echotexture, surface irregul	14.4	Absent	1756	B
Siddalingap	50/M	DCLD with PHTN with ascites	Absent	Present	Absent	Grade 2 ascites	B/l nvbs	14.5	4.3	119	1.1	1.1	2.1	1.14	0.86	23	38	75	2.8	Shrunken, coarse echotexture, surface irregul	15.8	Present	753.1	B
Basayya	55/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	8.6	10.6	164	1.5	1.5	3.5	0.54	2.96	1.4	66	219	2.2	Shrunken, coarse echotexture, surface irregul	13.2	Absent	1242	B
Rama	55/M	CLD with PHTN with ascites with acute pancreatitis	Absent	Present	Absent	Grade 3 ascites	B/l nvbs	12.2	7.5	105	1.57	1.57	1.79	0.27	1.52	11	12	175	3	Shrunken, coarse echotexture, surface irregul	15.7	Present	668.7	B
Nandu	65/M	DCLD with PHTN with ascites with HRS, in HE	Pallor, edema	Present	Present	Grade 3 ascites	B/l basal crepts	8.6	11.2	102	1.56	1.56	0.77	0.16	0.61	36	25	122	2.8	Shrunken, coarse echotexture, surface irregul	16	Present	668.7	C
Rama	49/M	CLD with PHTN with ascites	Pallor	Present	Absent	Grade 2 ascites	B/l nvbs	8.7	8	70	1.65	1.65	1.83	0.46	1.37	44	19	82	2.2	Shrunken, coarse echotexture, surface irregul	13.1	Present	534.3	C
Sangappa	56/M	DCLD with PHTN with ascites with UGI bleed	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l nvbs	7.5	8.5	135	1.79	1.79	1.88	0.7	1.18	42	20	122	2.3	Shrunken, coarse echotexture, surface irregul	14.1	Present	957.4	B
Tanveer	35/M	DCLD with PHTN with UGI bleed	Pallor, icterus	Present	Absent	Minimal ascites	B/l nvbs	6.8	3.3	35	1.53	1.52	4.11	1.59	2.52	139	29	149	3.3	Shrunken, coarse echotexture, surface irregul	13.5	Present	259.2	C
Shrishail	65/M	CLD with PHTN with ascites with SBP	Pallor, icterus	Present	Absent	Grade 4 ascites	B/l nvbs	9.9	17.4	128	1.44	1.42	4.7	0.3	4.3	94	20	565	2.8	Shrunken, coarse echotexture, surface irregul	15.1	Absent	1050.4	C
Mallikarjun	45/M	DCLD with PHTN with ascites with UGI bleed	Pallor	Present	Absent	Grade 2 ascites	B/l nvbs	7.7	8.3	105	1.97	1.96	1.27	0.14	1.13	32	15	95	3.3	Normal, coarse echotexture, surface irregular	14.5	Present	724.1	C
Maruti	72/M	CLD with PHTN with ascites in HE, Herpes labialis	Pallor, icterus	Present	Present	Minimal ascites	B/l nvbs	8.4	15.6	93	1.3	1.29	6.5	1.02	5.4	27	23	134	3.2	Increased, coarse echotexture, surface irregul	16.5	Absent	1483.2	C
Shivanand	36/M	CLD with PHTN with ascites in HE	Pallor	Present	Present	Minimal ascites	B/l nvbs	6.6	4.9	132	1.5	1.52	1.28	0.4	0.6	64	32	121	1.8	Shrunken, coarse echotexture, surface irregul	13.8	Present	956.5	C
Chodappa	86/M	CLD with PHTN with UGI bleed	Pallor	Present	Absent	Grade 2 ascites	B/l nvbs	7.1	15.6	150	1.6	1.36	0.2	0.09	0.11	13	10	78	2.9	Shrunken, coarse echotexture, surface irregul	12.8	Present	674.6	B
Nagesh	56/M	CLD with PHTN with ascites in HE	Pallor, icterus, edema	Present	Present	Grade 2 ascites	B/l nvbs	8.2	5.3	143	1.69	1.68	3.08	2.8	0.28	40	13	81	2.7	Shrunken, coarse echotexture, surface irregul	14.6	Present	979.4	C
Pradeep	40/M	CLD with PHTN with ascites	Pallor, icterus	Present	Absent	Grade 3 ascites	B/l nvbs	8.5	23.7	183	2.32	2.3	24.5	3.7	20.8	114	26	144	2.4	Normal, coarse echotexture, surface irregular	14.2	Absent	1288	B
Prashant	35/M	DCLD with PHTN with ascites in HE	Pallor, icterus, edema	Present	Present	Grade 3 ascites	B/l nvbs	5.9	9.3	42	3.2	3.28	24.3	5.1	19.2	46	19	107	2.4	Shrunken, coarse echotexture, surface irregul	15.6	Present	269.2	C
Sadanand	48/M	CLD with PHTN with ascites in HE	Pallor, icterus	Present	Present	Grade 3 ascites	B/l nvbs	9.3	11.2	86	2.5	2.54	10.3	6.02	2.28	106	40	85	2.2	Shrunken, coarse echotexture, surface irregul	13.8	Present	623.1	C
Kalappa	55/M	CLD with PHTN with ascites with UGI bleed, pneumonia, in l	Pallor, icterus, edema	Present	Present	Grade 2 ascites	B/l basal crepts	7.4	20.7	50	2.62	1.6	3.4	0.9	2.4	62	35	177	2	Shrunken, coarse echotexture, surface irregul	14.4	Present	347.2	C
Mahadev	48/M	CLD with PHTN with ascites secondary to hepatitis b, HIV +	Pallor, icterus	Present	Absent	Grade 2 ascites	B/l basal crepts	8.9	10.7	50	1.98	1.9	5.3	0.86	4.5	36	14	131	2.6	Shrunken, coarse echotexture, surface irregul	15.5	Present	322.5	C
Sadanand	46/M	CLD with PHTN with ascites, in SBP with HPS	Pallor, icterus	Present	Absent	Grade 2 ascites	Decreased air entry on right	8.7	16.7	136	1.9	1.98	9.2	2.4	6.8	102	34	133	2.4	Shrunken, coarse echotexture, surface irregul	14.1	Absent	964.5	B
Rajendra	56/M	CLD with PHTN with ascites	Pallor	Present	Present	Grade 4 ascites	B/l basal crepts	8.5	4.1	117	1.72	1.75	2.5	0.9	1.6	21	13	83	2.5	Shrunken, coarse echotexture, surface irregul	17.2	Present	680.2	C
Maruti	57/M	CLD with PHTN with ascites	Icterus, edema	Present	Absent	Grade 2 ascites	Decreased air entry on right	12	6.6	175	1.33	1.3	3.04	0.8	2.15	68	20	132	3.1	Normal, coarse echotexture, surface irreugula	14	Present	540.6	B
Bhimappa	41/M	CLD with PHTN with ascites	Absent	Present	Absent	Grade 2 ascites	B/l nvbs	12	7.5	140	1.6	1.6	1.4	0.6	0.87	38	16	96	3.4	Normal, coarse echotexture, surface irreugula	17.5	Present	800	C
Sumeet	22/M	CLD with PHTN secondary to Budd Chiari in HPS	Pallor, icterus	Present	Absent	Grade 3 ascites	B/l nvbs	10.6	6.4	70	1.6	1.6	4.9	4.1	0.8	46	15	236	3.8	Normal, coarse echotexture, surface irreugula	14	Present	500	C
Ramesh	54/M	DCLD with PHTN with ascites in HE	Icterus	Present	Present	Grade 4 ascites	B/l nvbs	10	5.3	156	1.23	1.21	4.29	2.4	1.8	57	26	202	2.5	Normal, coarse echotexture, surface irreugula	14.4	Present	880	C
Bhimappa	41/M	CLD with PHTN with ascites	Edema	Present	Absent	Grade 3 ascites	B/l nvbs	11.1	5.4	117	1.26	1.26	1.46	0.7	0.74	36	13	136	3.3	Shrunken, coarse echotexture, surface irregul	12	Absent	975	B
Ashok	62/M	DCLD with PHTN with ascites, secondary to hepatitis b	Icterus, edema	Present	Absent	Grade 4 ascites	B/l nvbs	12.2	5.4	96	1.3	1.35	3.9	2.8	1.14	54	39	60	3.3	Shrunken, coarse echotexture, surface irregul	11	Present	872.7	B
Afzal	45/M	DCLD with PHTN with ascites with UGI bleed, in HE	Pallor, icterus, edema	Present	Present	Grade 3 ascites	B/l basal crepts	6.2	14.2	60	2.07	2.06	5.37	2.41	2.96	46	44	148	1.8	Shrunken, coarse echotexture, surface irregul	14.2	Present	422.5	C
Vinay	50/M	CLD with PHTN with ascites	Icterus	Present	Absent	Grade 2 ascites	B/l nvbs	12.1	3.3	102	1.59	1.59	6.59	1.8	4.79	14	49	128	3.5	Shrunken, coarse echotexture, surface irregul	15.6	Present	653.8	C
Shivappa	63/M	CLD with PHTN with ascites secondary to hepatitis B	Pallor, icterus, edema	Present	Absent	Grade 3 ascites	B/l nvbs	9	9.2	86	1.6	1.6	2.53	1.64	0.89	67	41	124	2.4	Normal size, coarse echotexture, surface irreg	13.2	Present	651.5	C