Study of Portal vein Doppler indices and other noninvasive markers as predictors of esophageal varices in cirrhotic patients

Dissertation Submitted to

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

in partial fulfillment of the regulations

for the award of the degree of

D.M (GASTROENTEROLOGY)
BRANCH – IV

DEPARTMENT OF MEDICAL GASTROENTEROLOGY

GOVT. STANLEY MEDICAL COLLEGE & HOSPITAL

THE TAMIL NADU DR. M.G.R. MEDICAL UNIVERSITY

CHENNAI, INDIA.

AUGUST 2012

CERTIFICATE

This is to certify that the dissertation entitled "**STUDY OF PORTAL VEIN DOPPLER INDICES AND OTHER NONINVASIVE MARKERS AS PREDICTORS OF ESOPHAGEAL VARICES IN CIRRHOTIC PATIENTS**" is the bonafide original work of **Dr. V. ARULSELVAN** in partial fulfillment of the requirements for **D.M (GASTROENTEROLOGY) BRANCH – IV** Examination of the Tamilnadu Dr. M.G.R. Medical University to be held in August 2012.

The period of study was from April 2010 to January 2012.

Dr. S. GEETHALAKSHMI., M.D.,Ph.D.
DEAN
Govt. Stanley Medical College & Hospital,
Chennai-600 001.

Prof. A.R.VENKATESWARAN,
M.D.,D.M,
Professor & Head
Department of Medical Gastroenterology
Govt. Stanley Medical College
Chennai-600 001.

DECLARATION

I, **Dr.V.ARULSELVAN,** solemnly declare that the dissertation titled, "STUDY OF PORTAL VEIN DOPPLER INDICES AND OTHER NONINVASIVE MARKERS AS PREDICTORS OF ESOPHAGEAL VARICES IN CIRRHOTIC PATIENTS" is a bonafide work done by me at Govt. Stanley Medical College & Hospital during 2009-2012 under the guidance and supervision of **Dr. A.R.VENKATESWARAN, M.D., D.M,** Professor and Head, Department of Medical Gastroenterology, Stanley Medical College, Chennai-600 001.

The dissertation is submitted to Tamilnadu, Dr. M.G.R. Medical University, towards partial fulfillment of requirement for the award of **D.M Degree (BRANCH – IV) in Gastroenterology.**

Place: Chennai.

Date:

(Dr. V. ARULSELVAN)

ACKNOWLEDGEMENT

I express my profound gratitude to **Dr. S. GEETHALAKSHMI., M.D.,Ph.D.,** Dean of Government Stanley Medical College and Hospital, Chennai–600001 for permitting me to use all the needed resources for this dissertation work.

I sincerely express my grateful thanks to **Dr.A.R.VENKATESWARAN, M.D., D.M,** Professor and Head, Department of Medical Gastroenterology, Stanley Medical College for his unstinted support and advice rendered throughout my study. I sincerely thank him for his valuable guidance, suggestions and constant encouragement.

I am very much thankful to **Prof.V.Jayanthi, MD.,DM,** formerly my unit chief &professor for her valuable guidance and help in doing this study. I thank her for being a constant source of encouragement, inspiration, not only in this study but in all my professional endeavors.

I express my sincere thanks to all the Assistant Professors, **Dr. M. S. Revathy, MD., DM, Dr. R. Murali, MD., DM, Dr. S. Chitra, MD., DM, Dr. M. Manimaran, MD., DM,** Department of Medical Gastroenterology, SMC, Chennai, for their support and guidance in completing the study.

I also extend my sincere thanks to formerly my unit Assistant Professors **Dr A. Murali, MD.,DM, Dr T. Rajkumar Solomon, MD.,DM,** for their support, interest and enthusiasm in completion of this study.

I express my sincere thanks to **Dr. C. Amarnath**, **MDRD,** Professor and Head, Department of Radiology, Stanley Medical College for his guidance and help for completing the study.

I also thank **Dr. R. Gangadevi, MDRD, Dr. B. Suhashini, MDRD, Dr. K. Sivasankaran, MDRD,** Assistant Professors, and Dr. P. Prabakaran, Postgraduate student, Department of Radiology, Stanley Medical College for their help in doing Doppler study.

I also sincerely thank Ethical Committee, SMC, Chennai for approving my study.

I extend my sincere thanks to my subjects but for them the project would not have been possible.

I thank my wife, **Dr. K. Arunadevi, DNB**, who stood by me in successfully completing this study.

I am greatly indebted to all my friends, Postgraduate colleagues who have been the greatest source of encouragement, support, enthusiasms, criticism, friendly concern and timely help.

CONTENTS

INTRODUCTION

Portal Hypertension is the most common and lethal complication of chronic liver diseases. It is responsible for the development of gastroesophageal varices, variceal hemorrhage, ascites, renal dysfunction, portosystemic encephalopathy, hypersplenism and hepatopulmonary syndrome.

Portal hypertension commonly accompanies liver cirrhosis. The development of esophageal varices (EV) is one of the major complications of portal hypertension.[1] The prevalence of EV in patients with liver cirrhosis ranges from 60% to 80%. [2,3] Numerous evidences suggest that varices develop and enlarge with time. Christensen and colleagues showed that the cumulative incidence of varices in patients with cirrhosis increased from 12% to 90% over 12 years.[4] In a study, Cales and Pascal et al. showed 20% of patients who did not have varices developed new varices and 42% of patients with small varices showed definite enlargement.[5]

The risk of bleeding from these varices is associated with the severity of the liver disease and the size of varices, which are the most important predictors of bleeding.[6,7] It is estimated that approximately 60%–80% of patients with

cirrhosis develop esophageal varices during their life at a rate of 8% per year, and the progression from small to large varices occurs in 5%–10% of patients after the first year.[8-11]

Portal hypertension related upper GI bleeding accounts for 15-20% of all upper GI bleeding cases in Western population[12] and around 45% cases in Indian population.[13] Large EVs (LEVs) are more likely to bleed than small EVs (SEVs)[14] due to high variceal wall tension.[15] Among 60% of cirrhotic patients who develop gastroesophageal varices, 50% will experience an episode of variceal hemorrhage within 2 years of the diagnosis of the varices.[16,17] Majority of initial bleeds occurring with 1 year from the time of detection of varices.[6, 18] Up to one-third to half of the patients with advanced liver disease and large varices die after the first attack of variceal bleeding.[19]

The mortality rate from first episode of bleeding is 40%.[20] Mortality from each rebleeding episode is 20-30%. The reported overall mortality from variceal bleeding ranges from 17% to 57%. [2, 3] Introduction of prophylactic antibiotics and pharmacotherapy have shown to reduce the mortality.[2, 3, 21]

The American Association for the Study of Liver Disease and the Baveno V Consensus Conference on portal hypertension recommended that all cirrhotic

patients should be screened for the presence of EV when liver cirrhosis is diagnosed.[22, 23] Other authors have suggested repeating endoscopy at 2–3 year intervals in patients without varices and at 1–2 year intervals in patients with small varices so as to evaluate the development or progression of varices.[9, 24]

Upper gastrointestinal endoscopy, which is the most common and accurate procedure for evaluation of varices, is at times inconvenient for patients.[25,26] It also bears a small risk of complications like esophageal perforation, aspiration of gastric contents and bacteremia.[27,28] Moreover, sedation with benzodiazepines usually used for this procedure can significantly exaggerate hepatic encephalopathy.[29]

Investigators have attempted to identify characteristics that '**noninvasively'** predict the presence of varices. These studies have shown that biochemical, clinical, and ultrasonographic parameters alone or together have good predictive power for noninvasively assessing the presence of EV.[30-43] Overall, the most common result of these studies was that parameters such as splenomegaly, thrombocytopenia, Childs score, ascites, portal flow patterns, and platelet count—spleen diameter ratio were predictors of the presence of EV.

Doppler ultrasonography can be regarded as an attractive and non-invasive alternative method and may provide useful functional information. Many investigations reported correlations between different hepatic vasculature Doppler indices and the severity of portal hypertension and the resultant esophageal varices.[20, 35, 44-50]

Our study aimed to determine what Doppler indices of hepatic vessels can be used to predict the presence of esophageal varices and to evaluate the severity of esophageal varices.

AIM OF THE STUDY

1. To evaluate portal hypertension parameters in liver cirrhosis by using Doppler ultrasound.

2. To evaluate other non-invasive parameters in predicting esophageal varices.

3. To correlate portal hypertension parameters in predicting Esophageal varices and upper GI bleed from esophageal varices.

REVIEW OF LITERATURE

Portal hypertension (PHT) is defined by a pathologic increase in portal pressure above normal range of 6-10 mm or the pressure gradient between the portal vein and inferior vena cava (the portal pressure gradient [PPG]) is increased above the upper normal limit of 5 mm Hg.[51-53]

Portal hypertension becomes clinically significant when the PPG increases above the threshold value of 10 mm Hg (e.g., formation of varices) or 12 mm Hg (e.g., variceal bleeding, ascites). PPG values between 6 and 10 mm Hg represent subclinical portal hypertension.[51]

PPG is determined by the product of blood flow and vascular resistance within the portal venous system. The importance of portal hypertension is defined by the frequency and severity of complications: Massive upper gastrointestinal bleeding from ruptured gastroesophageal varices and portal hypertensive gastropathy (PHG), ascites, renal dysfunction, hepatic encephalopathy, arterial hypoxemia, disorders in the metabolism of drugs or endogenous substances that are normally eliminated by the liver, bacteremia, and hypersplenism.[54] These

complications are major causes of death and the main indications for liver transplantation in patients with cirrhosis.

Anatomical features of portal venous system:

The name “portal vein” derives from the notion that it is the gate into which the splanchnic circulatory system is connected to the liver (*porta* = gate). This venous system originates in the capillaries of the intestine and terminates in the hepatic sinusoids.

The portal vein is 6 to 8 cm long, formed by the union of the superior mesenteric vein and the splenic vein at the level of the second lumbar vertebra, just behind the neck of the pancreas which drains the stomach, the large and small intestine, the pancreas, and the spleen. The left gastric (coronary) vein joins the portal vein at its origin 50% of the time, and it joins the splenic instead of the portal vein in the other 50% of subjects.

The segment of the portal vein after the last afferent branch runs in the hepatoduodenal ligaments (the free edge of the lesser omentum) in a plane dorsal to the bile duct and the hepatic artery. This segment extends for approx 6–8 cm before entering the liver and it is 1–1.2 cm in diameter. The portal trunk

divides into two lobar veins before entering the portal fissure. The right lobar branch, short and thick, then receives the cystic vein. The left lobar vein is longer than the right and consists of a transverse and an umbilical part. Then the segmental branches of the portal vein split dichotomously into equal sized branches, constituting a tree of conducting vessels that terminate in venules.

The recanalized umbilical or paraumbilical veins arise from the umbilical portion of the left portal vein and pass through the round ligament to the anterior abdominal wall, where they may become evident, in the presence of portal hypertension, in the umbilical varices.

The portal vein is not provided with valves, so the pressure is transmitted freely back to the afferent branches. The portal vein pressure normally ranges between 5 and 10 mmHg (depending on the method of measurement). Normal fasting hepatic blood flow is approx 1500 ml/min. About two-thirds of the total hepatic blood flow and about one-half of the oxygen consumption are supplied by the portal vein, whereas the remainder is supplied by the hepatic artery. This dual hepatic blood supply makes the liver rather resistant to hypoxia.

Portal Collateral Circulation:

The portal system has numerous collaterals that interconnect with the systemic circulation. When portal pressure rises above 10 mmHg potential portosystemic collaterals may develop. The most important sites for the development of portosystemic collateral vessels are:

(1) esophageal submucosal veins, supplied by the left gastric vein and draining into the superior vena cava through the azygos vein; (2) paraumbilical veins, although normally non-functional, can serve as an anastomosis between the umbilical part of the left portal vein and the epigastric veins of the anterior abdominal wall that drain into the superior or inferior vena cava, and in special circumstances may form caput medusa at the umbilicus (Cruveilhier–Baumgarten syndrome); (3) rectal submucosal veins, supplied by the inferior mesenteric vein through the superior rectal vein and draining into the internal iliac veins through the middle rectal vein; (4) splenorenal shunts, in this case venous blood may be carried to left renal vein, either directly or by way of the diaphragmatic, pancreatic, or gastric veins; (5) short gastric veins communicate with the esophageal plexus; (6) within the cirrhotic liver, there is significant collateral flow in small veins that connect branches of the portal and hepatic veins.

The Gastroesophageal Junction:

Clinically, the most significant collaterals are the intrinsic veins of the gastroesophageal junction, which are located close to the mocusal surface. They are the collaterals most likely to bleed when dilated because of increased blood flow. According to Vianna et al.[55] There are four distinct zones of esophageal venous drainage (from distal to proximal): (1) the gastric zone, which extends for 2–3 cm just below the gastroesophageal junction. Veins from this zone drain into the short gastric and left gastric veins. (2) The palisade zone extends 2–3 cm superiorly from the gastric zone into the lower esophagus and represent the watershed between the portal and systemic circulation. (3) The perforating or transitional zone extends approx 2 cm above the palisade zone. Characteristic features of this zone is presence of perforating veins through the muscle wall of the esophagus linking the submucosal and paraesophageal venous plexuses that are tributaries of the azygos venous system. These perforating veins run circumferentially around the esophageal wall. In PHT patients, dilated perforating veins become incompetent and allow retrograde blood flow from the paraesophageal to the submucosal veins. (4) The truncal zone is 8–10 cm long and is characterized by four of five longitudinal veins in the lamina propria. In this zone, perforating veins penetrate from the submucosa at irregular intervals to the external esophageal venous plexus.

Flow through the perforating veins is unidirectional toward the extrinsic plexus and systemic circulation. When portal hypertension develops, the valves of the perforating veins become incompetent and allow reversal of flow from the extrinsic to the intrinsic system.

Varices of the gastroesophageal junction usually are classified by location as esophageal or gastric. Esophageal varices consist of three or four large trunks that are further characterized by size. The classification is important because the larger the varix, the more likely it is to bleed. Gastric varices, on the other hand, are by convention classified only by location.

Pathphysiology of portal hypertension :

Portal venous pressure is directly related to portal venous blood flow and resistance through the liver as described by Ohm's law, i.e., P = Q x R, where P is pressure along a vessel, Q is the flow, and R is the resistance to the flow.

Portal hypertension may result from both increases in flow and in resistance. Resistance is due to architectural distortion and regenerating nodules which cause resistance to the portal blood flow. There is an active intrahepatic vasoconstriction that accounts for 20-30% of the increased intrahepatic

resistance,[56] which is mostly due to a decrease in the endogenous production of nitric oxide.[57,58] Endothelin-1 also contributes to increased intrahepatic vasoconstriction in PHT by enhancing hepatic stellate cell contractility. Clinical trials of endothelin receptor blockers in treatment of PHT are underway. Other vasoconstrictive mediators are angiotensin, thromboxane, and cysteinyl leukotrienes.

In addition to resistance, increase in flow is also responsible for PHT. There is hyperdynamic circulation characterized by splanchnic vasodilatation, reduced mean arterial pressure, and increased portal blood flow.

Figure 1 - Schematic representation of the pathophysiology of portal hypertension:

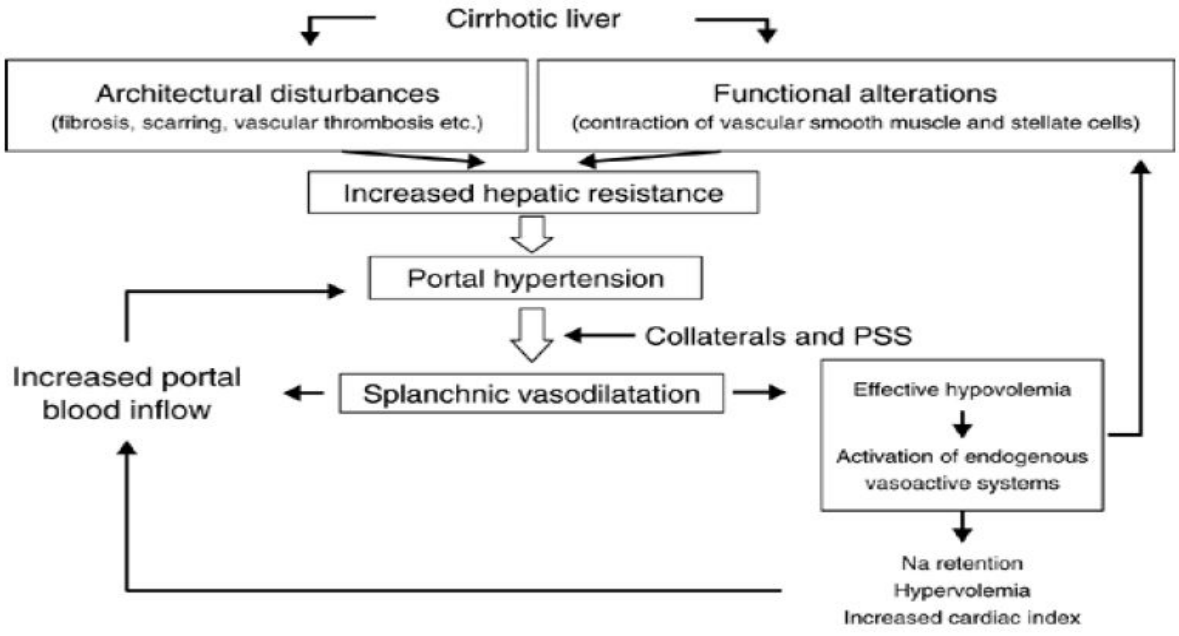
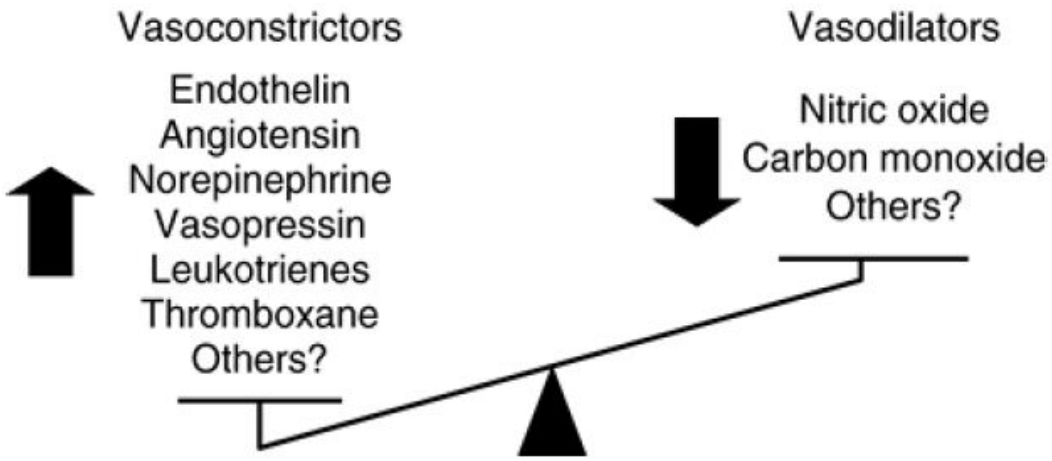


Figure 2 - Factors modulating intrahepatic resistance in cirrhosis:



Classification of Portal Hypertension:

The most useful classification of PHT is the anatomical classification, which categorizes it according to the site of pathology. Some causes and sites of the increased resistance are listed below:

- Prehepatic
    - Splenic vein thrombosis
    - Portal vein thrombosis
    - Exrtrinsic compression of the portal vein

- Intrahepatic
    - ❖ Presinusoidal
        - o Schistosomiasis
        - o Primary biliary cirrhosis
        - o Idiopathic portal hypertension
        - o Noncirrhotic portal fibrosis
        - o Congenital hepatic fibrosis
    - ❖ Sinusoidal
        - o Alcoholic cirrhosis
        - o Cryptogenic cirrhosis
        - o Postnecrotic cirrhosis
        - o Alcoholic hepatitis
    - ❖ Postsinusoidal
        - o Veno-occlusive disease of liver
- Posthepatic
    - o Budd-Chiari syndrome
    - o Thrombosis of Inferior Vena Cava
    - o Constrictive pericarditis

Pathophysiology of variceal Hemorrhage and prediction of variceal hemorrhage:

In cirrhosis, PHT is aggravated by the increase in the portal venous inflow due to splanchnic vasodilatation. When portal pressures rise, blood flow is diverted to venous collaterals that dilate to form vairces. All cirrhotic patients with varices do not experience bleeding episodes. Bleeding occurs in only about 30%. Various factors including physical factors such as the elastic properties of the vessel and the intravariceal and intraluminal pressure are important determinants of whether rupture will occur. The likelihood that any one varix will rupture and bleed depends on its wall tension. Variceal wall tension is determined by the application of Poiseuille's and Laplace's laws.

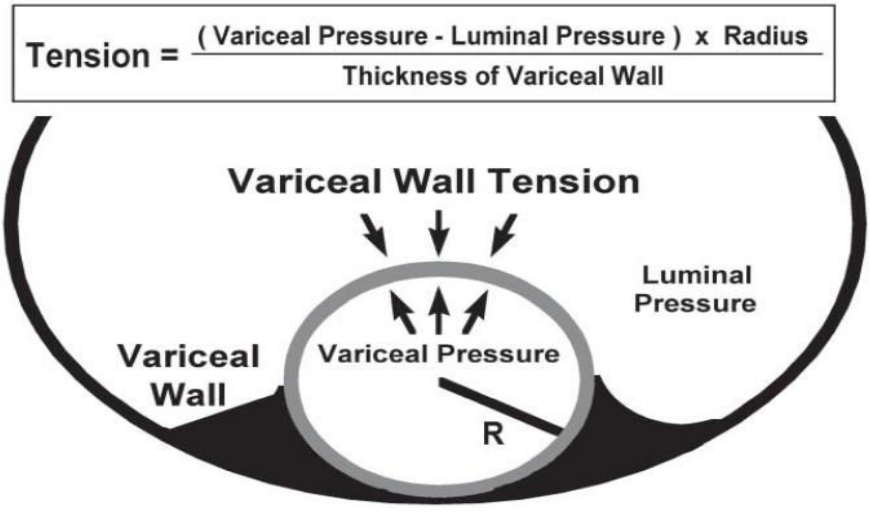


Figure 3 - Laplace's Law applied to esophageal varices - factors interact in the pathophysiology of variceal bleeding.

In practice, this means that a large(R), long varix with a high flow rate and a thin wall is most likely to rupture and bleed. Thus, Highest risk of first hemorrhage (15% per year) occurs in patients with large varices.[6] Other predictors of hemorrhage are decompensated cirrhosis (Child B/C) and the endoscopic presence of red wale marks (raised red streaks) and cherry red spots.[6] Hemodynamic measurements of portal pressure including hepatic vein pressure gradient (HVPG), intravariceal pressure, and Doppler ultrasound are the valuable predictors for the same. Rebleeding usually occurs within the first 6 weeks after an initial bleed.

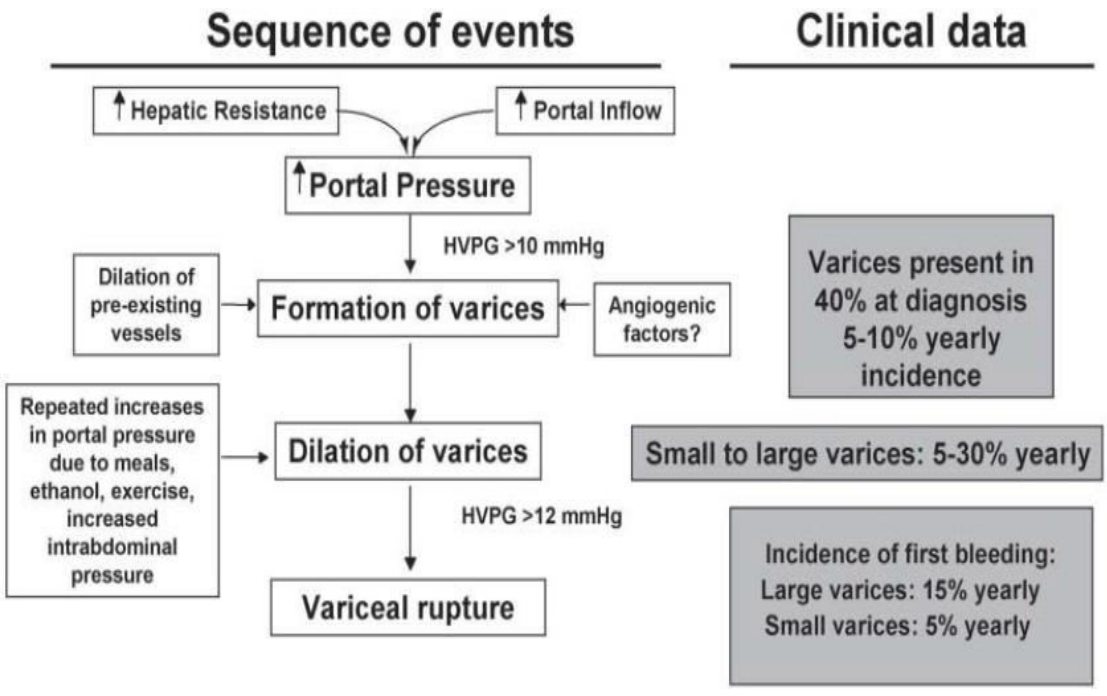


Figure 4- Variceal bleeding is the final step of a chain of events initiated by an increase in portal pressure, followed by the development and progressive dilation of varies until these finally rupture and bleed

Because it is not feasible to shorten a varix or increase its wall thickness, therapies for PHT aim to decrease variceal flow. This decrease is achieved by reducing either portal venous inflow (e.g., by splanchnic vasoconstriction) or resistance to portal outflow (e.g., by creation of a shunt).

Natural History varices:

The natural history of varices in cirrhotic patients with PHT evolves from a patient without varices to the development of varices and variceal bleed. Varices and variceal hemorrhage are a direct consequence of PHT. Patients with varices almost invariably have a portal pressure (as determined by the hepatic venous pressure gradient [HVPG] of at least 12 mmHg, while normal HVPG is 3-5 mmHg.[7,59] Gastroesophageal varices occur in 50% of patients with cirrhosis, at the time of diagnosis. Their presence correlates with the severity of liver disease; Almost 40% of Child A patients have varices, compared to 85% of Child C patients.[60] Patients without varices develop them at a rate of 8% per year.[10,61,62] Initially varices are small, but they enlarge with increasing blood flow. Variceal hemorrhage occurs at yearly rate of 5-15%. In cirrhotic patients 80-90% of bleeding episodes are due to variceal hemorrhage and one third of deaths in cirrhotic patients can be attributed to variceal hemorrhage. Although bleeding from esophageal varices ceases spontaneously in up to 40% of

patients, the mortality of an episode of variceal hemorrhage is of at least 20% at 6 weeks, and it occurs mostly in patients with severe liver disease and in those with early re-bleeding. Late rebleeding occurs in approximately 60% of untreated patients within 1-2 years of the index hemorrhage.[7, 63]

Table 1: Risk factors for early rebleeding

Gastric varices
Encephalopathy
Alcoholic cirrhosis
Large varices
Active bleeding at endoscopy
High HVPG

Diagnosis of Variceal bleeding:

Early diagnosis of variceal bleeding is important, as early treatment of bleeding cat be lifesaving one. The gold standard in the diagnosis of varices is esophagogastroduodenoscopy (EGD). It confirms the variceal cause of bleeding and also determines the exact site of it. It also allows immediate management of the varices by banding or sclerotherapy. However, it should only be performed in a hemodynamically stable patient. Endoscopically varices are classified as per Paquet's grading[64]

Grade 1 – Small varices without luminal prolapsed

Grade II – Moderate varices with luminal prolapsed and minimal obscuring of OG junction

Grade III- Large varices substantially obscuring the OG junction.

Screening for esophageal varices:

There are no reliable methods of predicting which cirrhotic patients will have esophageal varices without endoscopy.[43] The American Association for the Study of Liver Disease and the Baveno V Consensus Conference on portal hypertension recommended that all cirrhotic patients should be screened for the presence of EV when liver cirrhosis is diagnosed.[22, 23] The optimal surveillance intervals for esophageal varices have not been determined. For patients found to have no varices on initial screening endoscopy, repeat endoscopy at 3-year intervals has been suggested, whereas patients with small varices should undergo endoscopy in 1-2 years.[9, 24] Varices may grow faster in patients with cirrhosis secondary to alcohol abuse or severe liver impairment and in those with endoscopy stigmata of high risk (red wale markings); this subgroup of patients should undergo yearly upper endoscopy.

Upper gastrointestinal tract endoscopy, which is the most common and accurate procedure for evaluation of varices, is at times inconvenient for patients.[25,26] It

also bears a small risk of complications like esophageal perforation, aspiration of gastric contents and bacteremia.[27,28] Moreover, sedation with benzodiazepines used for this procedure can significantly exaggerate hepatic encephalopathy.[29]

Doppler ultrasonography is a non-invasive alternative method and may provide useful functional information. Many investigations reported correlations between different hepatic vasculature Doppler indices and the severity of portal hypertension and the resultant esophageal varices.[20,35,44-50] Concerning abdominal ultrasound and Doppler studies, inter-observer and inter-equipment variability limit their applicability in clinical practice. Thus the current recommendation is that the accuracy of non invasive tests (Doppler ultrasound) for the diagnosis of PHT should be further assessed before their use can be recommended in clinical practice. As a consequence, upper GI endoscopy is the main tool used for screening and monitoring patients.

Assessment of Severity:

The prognosis of the patient is best assessed by Child-Pugh criteria. If the total score is 5 or 6, cirrhosis is designated classA; if the score is 7-9, the cirrhosis is Class B; and if the score is ≥10, the cirrhosis is class C. The prognosis is

directly related to the score. Further, higher the score , more are the chances that the patient will have varicel bleeding in near future.

Table 2: Child-Pugh Classification of Severity of cirrhosis

Variable	1 point	2 point	3 point
Encephalopathy	Absent	Mild to moderate	Severe to coma
Ascites	Absent	Slight	Moderate
Bilirubin (mg/dl)	<2	2-3	>3
Albumin (g/dl)	>3.5	2.8 – 3.5	<2.8
Prothrombin time (seconds above normal	1-4	4-6	>6

Hemodynamic measurement of portal hypertension:

The evaluation of patients with portal hypertension is based on the visualization of varices at endoscopy, definition of the portal collateral anatomy and portal vein hemodynamics by Doppler ultrasonography or angiography, and the measurement of portal pressure.

A. Measurement of Portal Pressure

Measurement of portal pressure is still the single most important hemodynamic measurement in portal hypertension. Portal pressure should be expressed in terms of the pressure gradient between the portal vein and the IVC, which represents the perfusion pressure within the portal and hepatic circulation. Normal values of PPG are up to 5 mm Hg.

Portal pressure can be assessed by direct or indirect methods.

1. **Direct measurement**:

Direct measurements of portal pressure are invasive investigations based on the surgical, percutaneous transhepatic, or transvenous (transjugular) catheterization of the portal vein. Measurement of splenic pulp pressure and direct measurement of the portal vein pressure are invasive, cumbersome, and infrequently used approaches. Esophageas variceal pressure also can be measured but is not routinely performed in clinical practice.

2. **Indirect measurement:**

The indirect and safe approach of hepatic vein catheterization, with measurements of the wedged hepatic venous pressure (WHVP) and free hepatic vein pressure (FHVP) and hepatic vein pressure gradient (HVPG) is the preferred technique to estimate portal pressure. The FHVP, measured when the tip of the catheter is maintained "free" in the hepatic vein, is close to the IVC pressure. WHVP is obtained by placing a catheter in the hepatic vein and wedging it into a small branch or by inflating a balloon and occluding a larger branch of the hepatic vein. The WHVP has been shown to correlate very closely with portal pressure in cirrhosis.[65]

The HVPG is the difference between the WHVP and FHVP. The HVPG has been validated as the best predictor for the development of complications of PHT. The normal HVPG is 3-5 mmHg. HVPG has to be above 10 mm Hg for varices to develop and above 12 mm Hg for variceal bleeding. The threshold values define "clinically significant portal hypertension."[59, 61] HVPG is helpful in determining the cause of PHT, like presinusoidal, sinusoidal, or postsinusoidal because HVPG is normal in presinusoidal cause of PHT. It is also helpful to monitor patients in prevention of variceal bleeding. If HVPG decreased below 12 mmHg with therapy, variceal bleeding is prevented and varices decrease in size. It has also been shown that a reduction of 20% or more in HVPG (even if the HVPG remains more than 12 mmHg) is associated with a

reduction in the risk of variceal bleeding. The achievement of these targets also lowers the risk of ascites, hepatic encephalopathy, and death.[66] However, the Baveno IV consensus states that routine use of HVPG cannot be recommended, because of its invasiveness.[67]

B. Assessment of portal hemodynamics by Color Doppler Ultrasound:

Because HVPG cannot be recommended for routine clinical practice for assessing PHT and upper GI endoscopy is somewhat inconvenient and not cost effective for assessing esophageal varices, various attempts have been made to find less invasive techniques to assess severity of PHT and its correlation with development of esophageal varices. Doppler ultrasonography is a non-invasive alternative method for assessing PHT. Many investigations reported correlations between different hepatic vasculature Doppler indices and the severity of portal hypertension and the resultant esophageal varices. [20,35,44-50] It is widely used to explore the relationship between EV hemodynamic associated with PHT and cirrhosis.[68,69] Several Doppler ultrasonographic parameters have been measured to assess PHT such as Liver and spleen sizes, portal and splenic vein diameters, portal vein mean velocity (PVV), hepatic artery resistive index (HARI), hepatic artery pulsatility index (HAPI), portal vein cross sectional area, Splenic artery resistive index (SARI), presence of portal-systemic collaterals and following

indices have been calculated : liver vascular index (LVI), congestion index (CI), portal hypertension index (PHI)

1. **Liver and Spleen Sizes:**

Liver size is determined by measuring the longitudinal length from the dome of the right lobe to the inferior end on the midclavicular line. USG may reveal changes of cirrhosis such as coarsened echo texture with or without altered echogenicity, nodularity of liver surface, presence of regenerating nodules and reduction in the number of visible portal or hepatic veins. Reduction of liver size with a combination of segmental hypertrophy and atrophy is seen. A caudate to right lobe ratio>0.65 and reduction of the transverse diameter of segment IV, i.e., left wall of gallbladder to ascending portion of left portal vein to less than 30 mm has been reported.[70] Splenic size is found by measurement of its longest diameter. If splenomegaly is absent, PHT is unlikely though not ruled out.

2. **Portal and Splenic Vein Diameters:**

The normal portal vein diameter is 9-12 mm in quiet respiration. A portal vein diameter of >13 mm indicates portal hypertension with a high degree of

specificity but low sensitivity and a calibre over 17 mm is 100% predictive for large varices.[71] Normal portal vein dimensions, however, do not exclude PHT. Portal vein diameter is done at the centre of the portal vein at a known angle of insonation less than 60˚. This is usually done 1 cm proximal to its bifurcation. Measurement of portal vein diameter is performed from the hilar segment, which has least interobserver variability.

Splenic vein diameter is measured at the splenic hilum. The upper limit of normal splenic vein ranges from 10-12 mm with the vessel become prominent in PHT.

3. Portal flow direction, velocity, and waveform:

The normal portal flow is always directed towards the liver (hepatopedal) and has a fairly uniform flow velocity with slight phasicity in the spectral tracing secondary to respiration, and to a lesser extend cardiac activity. The fasting mean flow velocity is approximately 12-18 cm/sec (range12-23cm/sec) and has respiratory cycle variation decreasing on inspiration and increasing on onset of expiration. In normal subjects, the average portal flow is 500-900 ml/min.[72]
Flow velocity is measured from the hilar segment with subjects in the supine

position and during inspiration. All subjects are examined by the intercostal route. Flow measurements consisted of peak, lowest, and mean venous velocity. Measurement of a cross-sectional area of the vessel lumen is done by showing it in a transverse plane. Flow direction recorded from both the main portal vein and right and left branches of it. Flow direction of the splenic vein is established to evaluate the flow direction in the portal system. Mean velocity should be calculated over tracing 4-6sec long in order to avoid fluctuation in flow velocity. As PHT develops, the flow velocity in the portal vein decreases and the flow in the portal vein lose its undulatory pattern and becomes monophasic. As severity of PHT increases, flow becomes biphasic and finally hepatofugal. A decrease in the portal venous flow velocity (<12 cm/s) is a characteristic feature of PHT.[73,35,44-46] Both the volume flow and velocity are affected by the development of collaterals. Koda et al[74] showed that a decrease in PVV is more sensitive than portal vein flow volume for the advancement of fibrosis stage.

4. Hepatic Artery:

The normal hepatic artery (HA) supplies only about 25% to 30% of blood to the liver. It lies anterior to the portal vein and measures about 4.6 mm. In a fasting patient, HA has a systolic velocity of 10-15 cm/sec. HA diastolic velocity normally is less than the peak portal vein velocity of about 18 cm/sec. If HA

diastolic velocities greater than the portal vein, cirrhosis should be considered. Measurements of the right HA are taken where it crosses the portal vein near the porta hepatis. The hepatic artery is evaluated via an intercostal approach by demonstration of right and left portal veins under a 60° angle. Resistive and pulsatility index values are measured in the intrahepatic main branches.

The resistive index (RI) of the HA in a fasting subject varies from 0.55 to 0.81 (mean 0.62-0.74). HARI increases in normal subjects after a meal.[50,75] The pulsatility index (PI) of the HA varies from 1.16 to 1.24 in normal subjects. The RI and PI of the hepatic artery are increased in chronic liver disease due to an increase in intrahepatic vascular resistance. The most commonly used measurement is the HARI which is an indirect estimation of the impedance of arterial flow into the liver. In patients with advanced hepatic cirrhosis and chronic hepatitis, the normal increase in RI after a meal is also absent. The HARI falls steadily following acute portal vein thrombosis.

Doppler impedance indices of spleen such as splenic artery resistive index (SARI) measured in the intraparenchymal branches of splenic artery, which is increased in patients with cirrhosis. Zhang et al. demonstrated that HAPI (P=0.036), SARI(0.046) are closely correlated with PHT.[76]

5. Doppler Indices for PHT:

(A) Liver vascular Index (LVI) :

LVI= PV velocity / HARI, aids in the diagnosis of cirrhosis and PHT. A LVI less than 12 cm/sec identified cirrhosis and PHT with a specificity of 97% and a sensitivity of 93%.[77] LVI in the patients with PHT was reduced compared with that in the healthy control subjects. This illustrates that patients with PHT are incapable of activating the hepatic artery to maintain liver perfusion.

(B) Congestive Index (CI):

Indirect assessment of portal pressure can be made using the congestive index. CI correlates with portal pressure or with portal resistance. The CI is the ratio between the cross sectional area and the mean flow velocity of portal trunk. It takes into account the fact that in PHT, the portal vein tends to dilate and blood velocity to decrease, so that higher values are found in patients with more severe portal resistance, pressure and larger varices. CI above 0.13 cm/sec has 67% sensitivity in predicting PHT. Haag et al.[78] used the congestion index (CI) of the portal vein, exceeding 0.1 cm/s-1 to diagnose PHT. The CI reflects the

portal vascular outflow resistance and was sensitive for the diagnosis of PHT at 0.08 cm/s-1 with 100% specificity.[76]

(C)Portal Hypertensive Index (PHI) :

Piscaglia et al.[79] proposed a PHI cut-off of 1.2 s/m as the parameter with the highest accuracy (≈75%) for PHT. PHI is considered a comprehensive index, concerned with both intrahepatic and extra hepatic hemodynamic changes. PHI in the patients with PHT was significantly higher than that in the control group; the results of resistance from both upstream blood flow and intrahepatic blood flow increased.

With the use of PVV and the HAPI, HARI, Splenic Artery RI, liver vascular index (VI), congestion index (CI), portal hypertension index (PHI) are calculated. Table 3 shows various Doppler indices measured.

Table 3 Indices, Their Explanations, and Units Used in the study

Index	Explanation	Unit
PVV[80]	Peak venous velocity in portal vein	Cm/sec
PV Dm	Portal vein diameter	cm
PV CSA	Portal vein cross sectional area	Cm^2
HARI[81]	Hepatic Artery Resistive Index = (peak systolic velocity – end diastolic velocity)/peak systolic velocity	*
HAPI[81]	Hepatic Artery Pulsatility Index = (peak systolic velocity – end diastolic velocity)/time-averaged peak velocity	*
SARI[82]	Splenic Artery Resistive Index	*
LVI[83]	Liver vascular index = PVV / HAPI	*
CI[84]	Congestive index = PVCSA(cm^2) / PVV	*
PHI[83]	Portal hypertensive index = (HARI*0.69)*(SARI*0.87) / PVV	*

*indices without units

6. Assessment of Hepatic Veins:

Doppler spectral traces from normal hepatic veins have a triphasic appearance, consisting of two large antegrade waves that represent atrial and ventricular diastole and a small retrograde wave that occurs in atrial systole.[77] Altered hepatic vein waveforms are seen in atleat 50% of patients with cirrhosis with flattening of the phasic oscillations.

7. Portosystmic venous collaterals :

Portosystmic venous collaterals are a clear indication of PHT. Ultrasonagraphy is reported to visualize 65-90% of collaterals. The important collaterals are Left gastric, short gastric vein, Paraumbilical vein, Splenorenal collaterals, collaterals within the gallbladder, splenoretroperitoneal, splenocaval, omphaloiliocaval, splenoportal.

Correlation between portal vein doppler indices and EV:

Tarzamni et al. showed that most of the echo-Doppler parameters were related to presence of EV and Portal vein flow velocity and liver vascular index was significantly higher in patients with EV while they had lower portal vein diameter, CI, portal hypertensive index, and hepatic and splenic artery RI.[83]

In study of Korner, the overall sensitivity for prediction of variceal bleeding in case of decreased portal vein mean velocity was 88% and that of its reduced volume flow was 65%.[45] Iwao et al.[50] Proposed LVI for the diagnosis of PHT and also showed significant association between low PVV, High HAPI in patients with large EV. Comparisons of the correlations between resistive indices and portal pressure showed that the HARI had the highest linear correlation with portal pressure. HAPI & LVI has weaker correlation and no significant correlations are present in the SARI.[76]

Haktanir et al. demonstrated that mean HARI and HAPI values were significantly larger and Portal and splenic vein diameters and spleen sizes of patients with cirrhosis were significantly greater than those of the Chronic Viral Hepatitis and control groups. LVI and Mean PVV of the cirrhosis group was smaller than those in the control and CVH groups ($P < 0.001$).[86]

C. Other Non-invasive Markers predicting esophageal varices:

Various studies have shown that different biochemical, clinical and ultrasonographic parameters alone or together have good predictive power for non-invasively assessing the presence of EV. They include splenomegaly,[30,33,37,87-89] thrombocytopenia,[30,33,34,37,87-90] ascites,[33,88,] hepatic encephalopathy,[33]

serum albumin concentration,[90] serum bilirubin levels,[90] and Child–Turcotte–Pugh (CTP),[88,89] Model for End-stage Liver Disease (MELD),[91] AST to platelet ratio index (APRI).[91]

In a study by Amarapurkar N et al. splenomegaly was detected in 86.7% of patients with liver cirrhosis, and it was 89.7% sensitive in predicting the presence of esophageal varices in patients with cirrhosis.[36] Various authors have reported similar findings of an enlarged spleen being associated with the presence of varices.[26,30-34,37]

Ismail et al. showed the presence of EV could be predicted by MELD score higher than 8 points (sensitivity: 80.10%; specificity: 51.20%; P=0.02) and APRI higher than 1.64 (sensitivity: 56.70%; specificity: 69.80%; P=0.01).[91] He also demonstrated that platelet count <91,000, palpable splenomegaly, splenic size of 158 mm, hemodynamic instability, cirrhosis with hepatocellular carcinoma, and a previous history of gastroesophageal variceal hemorrhage are indicators of large EV.[92]

Giannini A et al used platelet count/spleen diameter ratio cut off value of 909 had 100% negative predictive value for a diagnosis of EV. He showed the platelet count/spleen diameter ratio seems to represent an acceptable surrogate

with highest accuracy for clinically relevant portal hypertension and for non-invasively predicting the presence of EV.[89]

AST to platelet ratio index (APRI) values higher than 1.64 were correlated with the presence of EV, because they indicate more severe hepatic parenchymal architectural distortion and increased intrahepatic resistance, resulting in PHT.[91] Sanyal et al. studied 1016 clinically stable cirrhotic patients and reported a correlation between high values of APRI, low platelets count and elevated AST, and the presence of EV.[93] Sebastiani et al. found a weak correlation between APRI and the presence of any EV (APRI=1.4; sensitivity 54%; specificity: 69%) and large varices.[94] In a recent study by Tafarel et al. showed that presence of large EV require prophylactic therapy, could be predicted by MELD score higher than 8 points (sensitivity 80.1%;specificity 51.2%:p=0.02), APRI higher than 1.64 (sensitivity 56.7%; specificity 69.8% p=0.01) and thrombocytopenia of 93,000/mm^3 or less (sensitivity 56.1%; specificity 61.2%, p<0.01).[91]

Schepis F et al.[26] recommends endoscopic examination in liver cirrhosis with prothrombin activity below 75%, platelet count is less than 1,00,000/uL, and portal vein diameter exceeds 13 mm on ultrasound examination. Zaman et al.[31] in their study, found that a platelet count less than 88,000/uL significantly

predicts the development of esophageal varices. Chalasani et al.[30] in a multivariate analysis found thrombocytopenia (< 88,000/uL) and splenomegaly to be strong predictors of esophageal varices. Giannini et al, in their study in Italy in 2003, found a cut off point of platelet count > 112,000 (p= 0.0001; 95% CI 0.815 – 0.928), splenic diameter > 121 mm (p= 0.0001; 95% CI 0.850 – 0.951).[89] Prihatini J etal. used platelet count of equal to or less than 82,000/uL, portal vein diameter of 11.5 mm or more, and an anteroposterior splenic measurement of 103 mm or more to detect esophageal varices in liver cirrhosis.[20] Fook-Hong Ng et al. recommends endoscopic screening in liver cirrhosis in the presence of thrombocytopenia (<150,000/ul) or ascites.[33]

Treatment of acute variceal hemorrhage:

Treatment of variceal bleeding can be divided into immediate and definitive treatment. The goals of the treatment are: Control of acute bleeding, Prevent rebleeding by reducing portal pressure in patients who are at risk and by obliterating the varices, Prevent initial bleeding if possible in patients with diagnosed varices.

Regarding nonspecific management, immediate treatment includes admission in ICU, correction of hypovolemia with crystalloids(isotonic saline solution),

tracheal intubation for airway protection prior to endoscopy, blood volume replacement with the goals of maintaining hemodynamic stability and a haemoglobin level of approximately 8 g/dl.[67] Current guidelines recommend prophylactic antibiotic therapy for cirrhotic patients admitted with acute variceal hemorrhage and should be instituted form admission.[67] The recommended antibiotic schedule is norfloxacin 400 mg BD for 7 days or ceftriaxone (2 g/day) in advanced cirrhosis.[43]

The recommended specific management consists of the combination of endoscopic therapy plus a safe vasoactive drug (terlipressin or analogues, somatostatin or analogues). The advantage of these drugs is that they can be started at admission and before diagnostic/therapeutic endosocopy and continued for 2-5 days to prevent early variceal rebleeding. Baik et al.[95] suggested that 2 mg IV terlipressin may be of greater benefit than somatostatin and its analogues. Although both drugs decreased HVPG significantly, the effect of terlipressin was sustained over the 25 minutes, while the effect of octreotide was transient.

Regarding the best endoscopic therapy for the control of acute variceal hemorrhage, a meta-analysis of 10 randomised controlled trials shows an almost significant benefit of Endoscopic Variceal band Ligation (EVL) in the initial

control of bleeding compared to sclerotherapy.[96] HVPG increased significantly immediately after both EVL and sclerotherapy, and it remained elevated for 5 days for sclerotherapy group, while HVPG had decreased to baseline levels by 48 hours after EVL.[97] Therefore in the Baveno consensus report, EVL is the recommended form of endoscopic therapy for acute esophageal variceal bleeding although sclerotherapy is recommended if EVL is technically difficult.[67] Failures of initial therapy with combined pharmacological and endoscopic therapy are best managed by a second attempt at endoscopic therapy or, in the case of fundal gastric varices, by transjugular intrahepatic portosystemic shunt (TIPS).[67]

Prevention of recurrent variceal hemorrhage:

Either combination pharmacological therapy (nonselective b-blockers plus nitrates) or EVL are considered the therapy of choice in the prevention of variceal re-bleeding. The choice depends on tolerance and local expertise. However, with either of these therapies, re-bleeding rates are still quite high (30%-42% in studies of b-blockers plus nitrates; 20%-43% with EVL).[63] The lowest re-bleeding rates of 7%-13% have been described in studies on pharmacological therapy in which HVPG decreased by >20% from baseline or to levels below 12 mmHg.[63] Randomized controlled trials show combining EVL and b-blockers significantly reduces re-bleeding rates than EVL alone.[98,99]

Additionally, 1-year variceal recurrence was lower in the EVL+nadolol group (54%) than in the EVL alone group (77%). Therefore the current recommendation is still to use EVL+b-blocker± nitrates as first line therapy. Side effects of EVL include hemorrhage from ulcers, chest pain, dysphagia, and odynophagia. Because gastric acid may exacerbate post-EVL ulcers, acid suppressors may reduce EVL related side effects.

In patients who fail combined endoscopic and pharmacological treatment for prevention of re-bleeding, TIPS or surgical shunts should be considered, depending on local availability and the surgical candidacy of the patient. Patients with decompensated cirrhosis are not candidates for shunt therapy and should be evaluated for liver transplantation.

MATERIALS AND METHODS

Ninety nine Cirrhotic patients registered in liver clinic and admitted in ward (both old and new patients), Dept. of medical gastroenterology, Govt. Stanley medical college were included in this prospective study. The study period from April 2010 to January 2012. All subjects included in the study provided informed consent to participate. The study was approved by the Ethics Committee of our institution. All patients underwent a detailed clinical evaluation at entry, with the following data:

Age, Gender, Duration of illness, Details of treatment prior to registration. Etiology of cirrhosis was arrived based on history of alcohol intake including quantity & total duration of consumption; blood for viral serology (HBsAg &HBV DNA assay for hepatitis B and HCV RNA & Anti-HCV for hepatitis C); serum ceruloplasmin (<20mg%), presence of Kayser Fleischer ring and 24 hours urine copper estimation (>100mg%) for Wilsons disease; and antinuclear antibody, hypergammaglobulinemia (>3.5 gm%) for autoimmune related cirrhosis.

Apart from details of past blood transfusion, surgery, family members with liver disease, details of associated co-morbid illness were also recorded.

Relevant history and physical characteristics including symptoms and signs of liver failure (spider angioma, palmar erythema etc.), hepatomegaly, splenomegaly, and abdominal vein collaterals were recorded. Ascites was graded as none, mild (detectable only on ultrasound), moderate (visible moderate symmetrical abdominal distension) or severe (marked abdominal distension). Hepatic encephalopathy was graded from grade 0 to IV, as per the Conn's grading. Diagnosis of cirrhosis was based on clinical, biochemical, and ultrasonographic findings.

Exclusion criteria:

1. Patients with evidence of hepatocellular carcinoma on ultrasonography
2. Portal vein thrombosis
3. Previous H/O surgical intervention for portal hypertension

Blood tests:

Hematological and biochemical workup included measurement of hemoglobin, total leukocyte count, platelet count, prothrombin time, and serum concentrations of bilirubin (total and conjugated), protein, albumin, alanine aminotransferase and aspartate aminotransferase. All patients were tested for HBsAg and antibodies to hepatitis C virus to determine the cause of liver

cirrhosis. Tests for other causes of cirrhosis (serum ceruloplasmin and slit lamp examination for Wilson's disease, tests for autoantibodies for autoimmune liver disease, iron studies for hemochromatosis) were carried out only if there was a suggestive clinical clue. For each patient, a modified Child-Pugh score was calculated.

Child Turcotte Pugh (CTP) score was applied to grade the severity of cirrhosis. CTP score is based on serum bilirubin, serum protein, ascites, prothrombin time and hepatic encephalopathy. Minimum score of CTP is 5 and maximum score is 15. Based on scoring system, cirrhosis was classified as Childs A when the total score was 5 & 6, Childs B when the total score was 7 to 9, and Childs C when the total score is exceeded 9.

Colour Doppler Ultrasound:

All patients underwent ultrasonography and the following details were recorded: maximum vertical span of the liver; nodularity of liver surface; spleen size (length of its longest axis); diameter of the portal and splenic veins; presence of portal-systemic collaterals; and presence of ascites. All Doppler assessments were performed by a single radiologist using a 3.5 MHz curvilinear transducer of **EASOATE MyLab40** (Germany) machine. Patients were examined while fasting and in supine position and quiet respiration. Subcostal

or intercostals ultrasonic windows were used to obtain longitudinal view of middle hepatic vein, portal vein and hepatic artery (in front of portal vein) with an ultrasound beam incidence angle of less than 60º. Outer-to-outer main portal vein diameter (mm) was measured in midway between the spleno-portal junction and its intrahepatic bifurcation.

Several Doppler ultrasonographic parameters were measured such as Liver and spleen sizes, portal and splenic vein diameters, portal vein mean velocity (PVV), hepatic artery resistive index (HARI), hepatic artery pulsatility index (HAPI), portal vein cross sectional area, Splenic artery resistive index (SARI), presence of portal-systemic collaterals and following indices were calculated : liver vascular index (LVI), congestion index (CI), portal hypertension index (PHI).

Table 3: Indices, Their Explanations, and Units Used in the study

Index	Explanation	Unit
PVV[80]	Peak venous velocity in portal vein	Cm/sec
PVDm	Portal vein diameter	cm
PV CSA	Portal vein cross sectional area	Cm^2
HARI[81]	Hepatic Artery Resistive Index = (peak systolic velocity – end diastolic velocity)/peak systolic velocity	*
HAPI[81]	Hepatic Artery Pulsatility Index = (peak systolic velocity – end diastolic velocity)/time-averaged peak velocity	*
SARI[82]	Splenic Artery Resistive Index	*
LVI[83]	Liver vascular index = PVV / HAPI	*
CI[84]	Congestive index = PVCSA(cm^2) / PVV	*
PHT[83]	Portal hypertensive index = (HARI*0.69)*(SARI*0.87) / PVV	*

*indices without units

Endoscopic evaluation:

All patients underwent upper gastrointestinal endoscopy for assessment of esophageal and gastric varices after Doppler ultrasound examination. If EVs were present, their size was graded as I-IV, using the Paquet grading

system. Grade 0: No varices, grade I: Varices, disappearing with insufflation, grade II: Larger, clearly visible, usually straight varices, not disappearing with insufflation, grade III: More prominent varices, locally coil-shaped and partly occupying the lumen, grade IV: Tortuous, sometimes grape-like varices occupying the esophageal lumen.[64] Further, patients were classified dichotomously either as having large EVs (grade III-IV) or as not having these (no varices) or small EV (grade I-II). Presence of gastric varices, portal hypertensive gastropathy, and duodenopathy were recorded wherever appropriate.

Statistical analysis:

Data were analyzed with Analysis of Variance (ANOVA) Technique. Descriptive statistics including means, standard deviations, and frequencies were computed. For determining associations, univariate analysis was performed by ANOVA. A p value less than 0.05 was considered statistically significant. A multivariate ordinal logistic regression (OLR) model was used for determining the adjusted associations between size of esophageal varices and hepatic hemodynamic determinants.

RESULTS

Ninety nine cirrhotic patients (70 men, 29 women) were enrolled in the study. Mean age of the study population was 44 ± 11.5 years. Cirrhosis was predominantly observed in men (Male: Female - 2.4:1). Table 1 shows the patients' baseline characteristics.

Table 4: Demographic characteristics of the study population

Characteristics	**No (%)**
Total No of cases	99
Mean age (years)	44±11.5
Gender distribution	
Male	70 (71%)
Female	29 (29%)
Literacy status	
Yes	78 (79%)
No	21 (21%)
Alcohol Ingestion	
Yes	43 (43%
No	56(57%)
Smoking	
Yes	33(33%)
No	66(67%)

Figure 5: Gender distribution

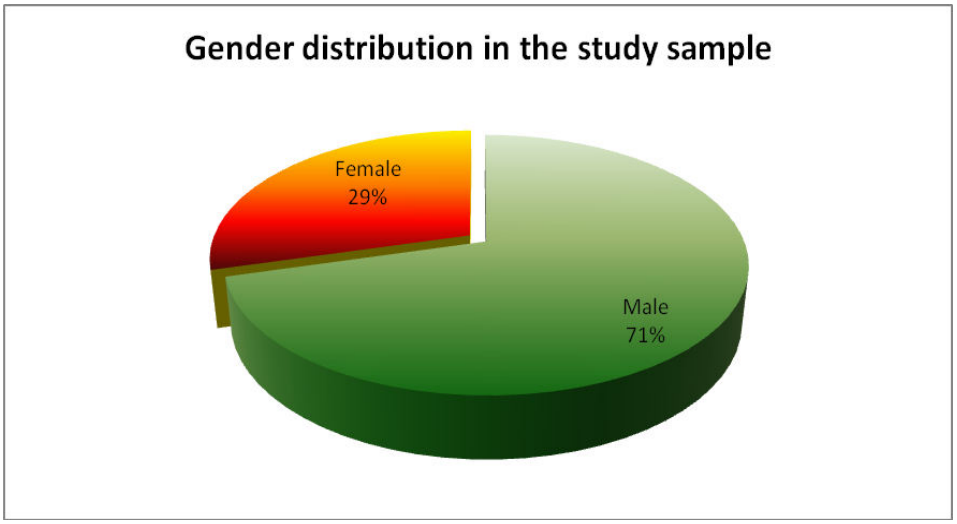
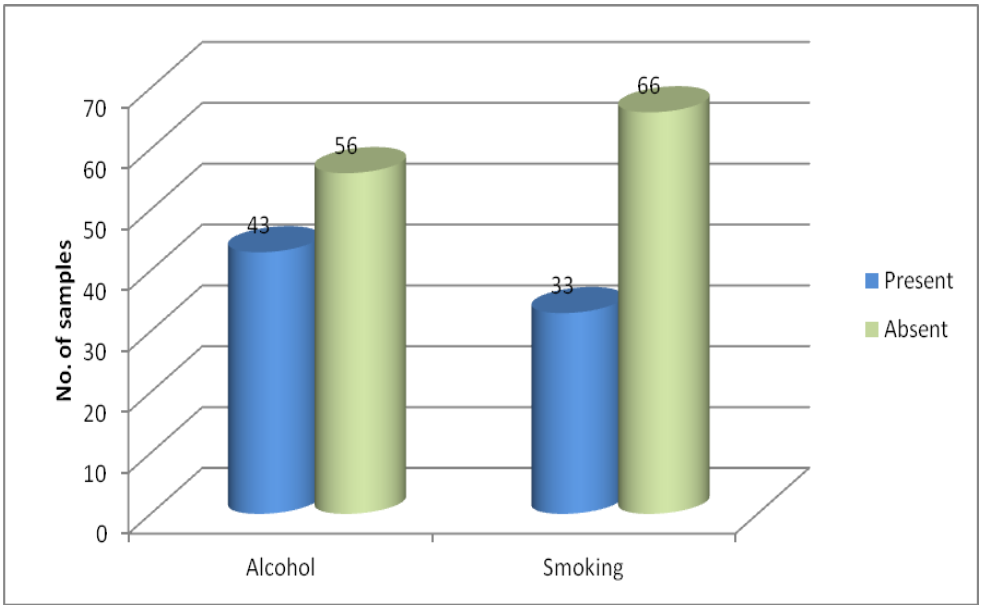


Figure 6: Alcohol and Smoking pattern

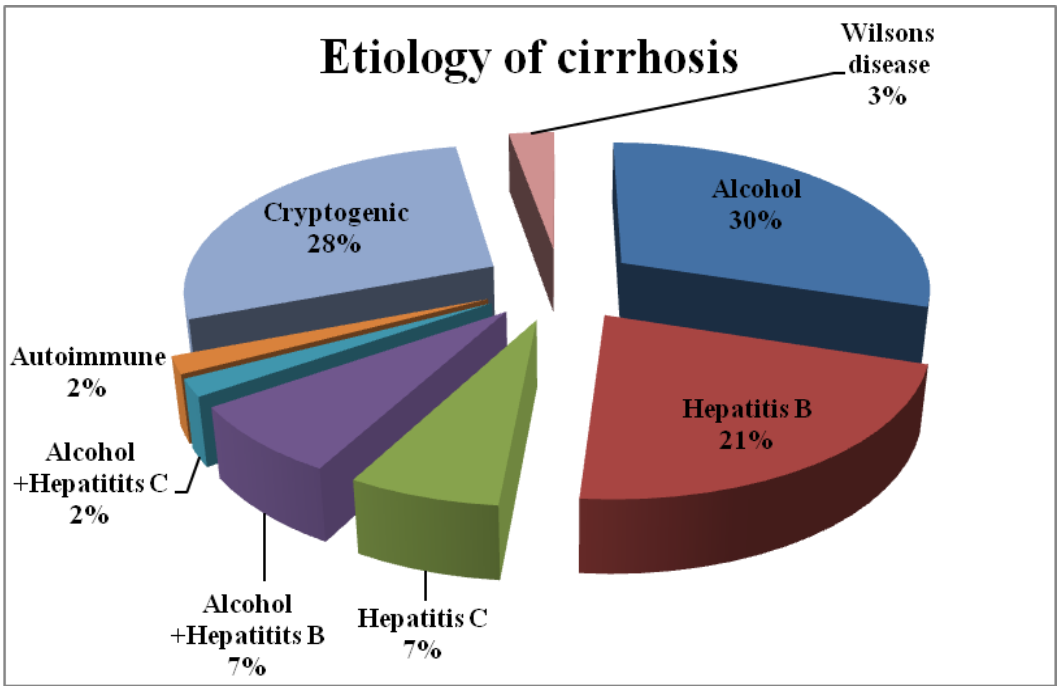


Alcohol related liver disease constitutes the most common etiology of cirrhosis in our study followed by cryptogenic and Hepatitis B related liver disease. (Table 5)

Table 5: Etiology of cirrhosis

Etiology	n	%
Alcohol	32	29.63%
Alcohol+Hepatits B	8	7.41%
Alcohol+Hepatits C	2	1.85%
Autoimmune	2	1.85%
Cryptogenic	30	27.78%
Hepatitis B	23	21.30%
Hepatitis C	7	6.48%
Wilsons disease	3	2.78%

Figure 7: Etiology of cirrhosis



The severity of liver disease as assessed by Child Pugh scoring was: Child A in 31%, Child's B and C in 45% and 23% respectively.

Table 6: Severity of liver disease as assessed by CTP score

CTP Grade	n	%
Grade A	31	31%
Grade B	45	45%
Grade C	23	23%
Total	**99**	**100%**

Table 7: Mean values of Laboratory investigation

Investigation	Mean	Std dev
Hb (gms%)	9.64	2.61
Platelet Count(no.)	95818.18	42936.68
PT (sec.)	18.49	4.64
INR	1.42	0.40
Creatinine (mg/dl)	2.23	13.48
TB mg/dl)	2.60	2.74
AST (U/L)	77.63	60.24
Albumin (g/dl)	2.84	0.73

Figure 8: Mean prothrombin time

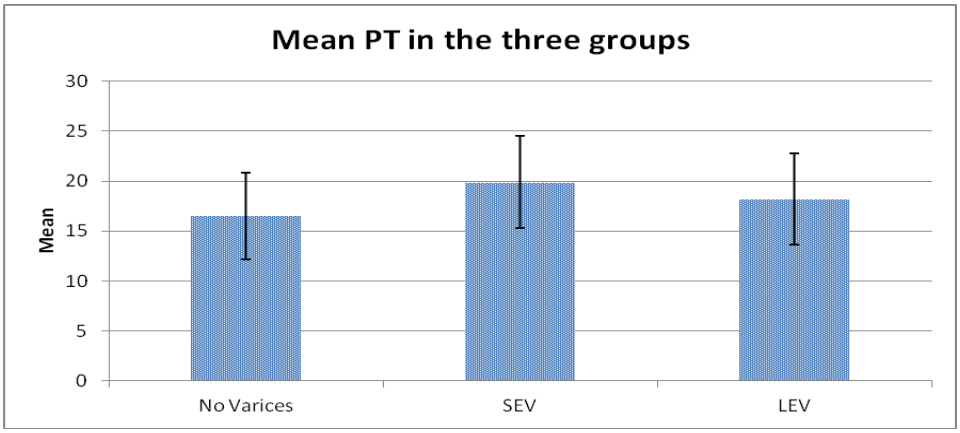


Table 8: Mean values of Doppler study parameters

Doppler study	Mean	Std dev
Liver size (cms)	11.98	1.45
PV DM (cms)	1.20	0.26
PV velocity (cm/sec)	12.97	4.60
HARI	0.71	0.12
HAPI	1.32	0.44
PV cs area(cm2)	1.05	0.58
Spleen Size (cms)	14.85	3.36
SARI	0.67	0.08
Liver Vascular Index	10.73	5.01
Congestive Index	0.11	0.14
PHT Index	0.05	0.11

Of the 99 patients, 19 (19%) did not have esophageal varices at endoscopy, 36 (36%) had small esophageal varices (SEV) and 44 (45%) have large esophageal varices (LEV).

Figure 9: Mean of different Doppler parameters

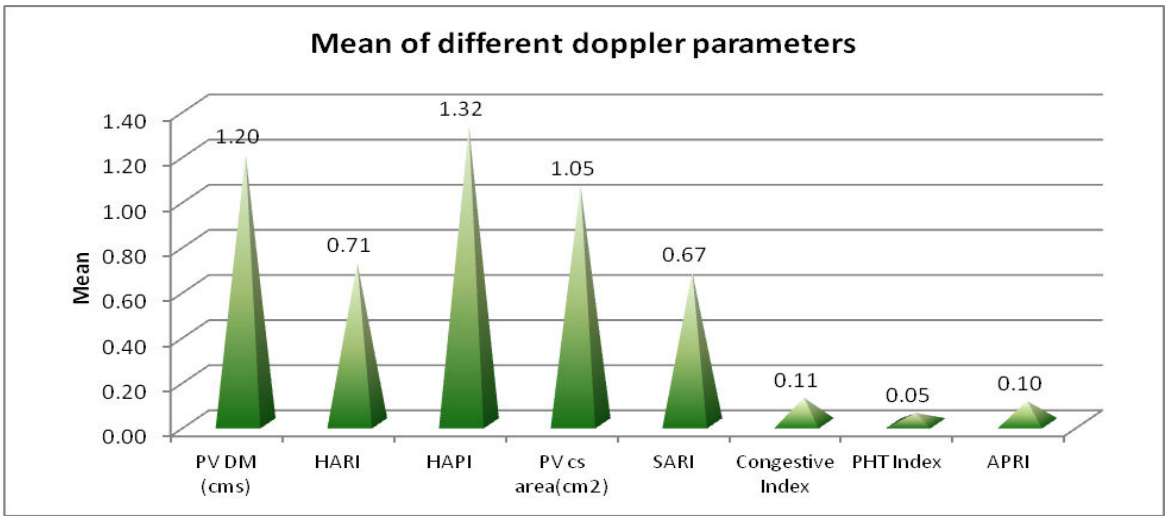


Figure 10: Mean of significant Doppler parameters

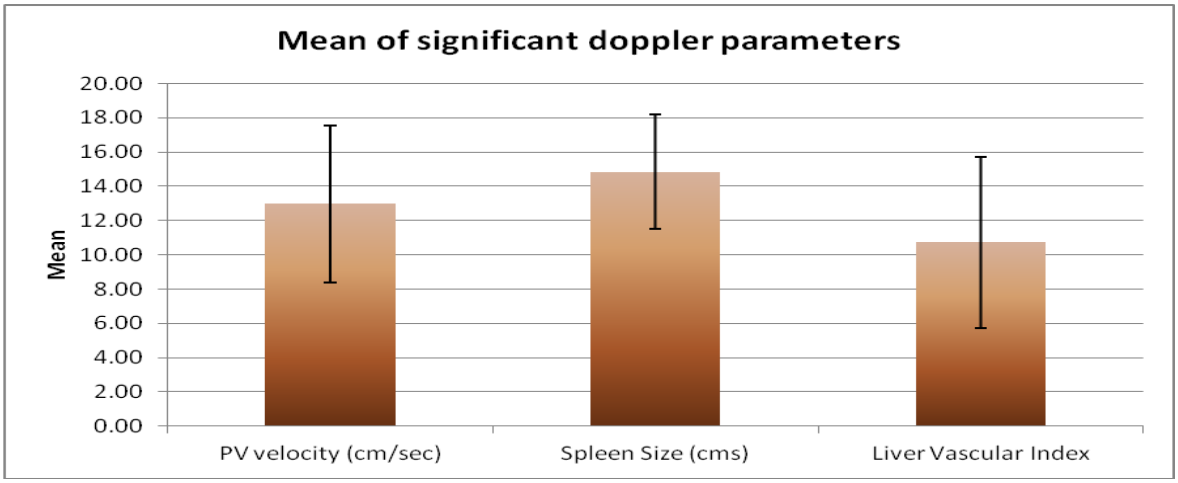


Table 9 shows the relationship of absence of EV or presence of SEV/LEV with various clinical, laboratory and ultrasonographic characteristics on univariate analysis.

Table 9: Comparison of different parameters according to Varices:

Parameter	No Varices (n=19)	SEV (n=36)	LEV (n=44)	P-Value
MELD	11.00± 4.22	14.39±5.59	12.39±4.94	0.500
Platelet Count	114578±53732	95333±44346	78113±34290	0.049*
PT(sec)	16.53±4.33	19.90±4.59	18.18±4.55	0.030*
INR	1.34±0.37	1.48±0.37	1.41±0.43	0.490
TB (mg/dl)	1.94±1.86	3.25±3.09	2.35±2.69	0.176
AST (U/L)	83.74±69.87	78.94±47.42	73.91±66.06	0.830
PV Velocity (cm/sec)	15.44±4.63	12.96±4.90	11.91±3.97	0.019*
HARI	0.67±0.08	0.74±0.15	0.71±0.09	0.108
HAPI	1.13±0.34	1.38±0.53	1.35±0.38	0.117
PV cs area (cm^2)	1.08±0.78	0.98±0.51	1.10±0.55	0.638
Spleen Size (cm)	13.34±4.20	15.10±2.56	16.29±3.42	0.05*
SARI	0.71±0.09	0.66±0.07	0.67±0.08	0.092
Liver Vascular Index	14.38±5.56	10.46±4.99	9.38±4.01	0.001*
Congestive Index	0.10±0.13	0.11±0.14	0.12±0.15	0.816
PHT Index	0.04±0.06	0.03±0.03	0.06±0.16	0.444
P/S Ratio	957.00±607.54	668.86±343.94	627.68±325.51	0.011*
APRI	0.07±0.04	0.10±0.07	0.11±0.12	0.288

Six factors were found to be significantly different between the three groups. These were Platelet count (P=0.049), Prothrombin time (P=0.030), Portal vein velocity (P=0.019), Liver vascular index (P=0.001), Spleen size (P=0.05) and Platelet count/spleen ratio (P=0.011). No significant difference is observed for the other parameters.

Figure 11: Mean PV Velocity in three groups

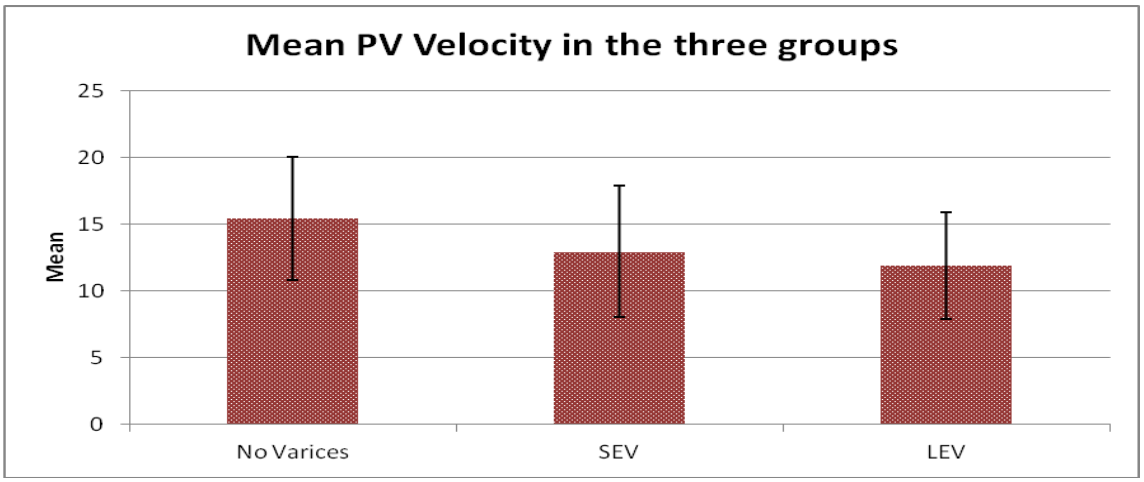


Figure 12: Mean Liver Vascular Index in the three groups

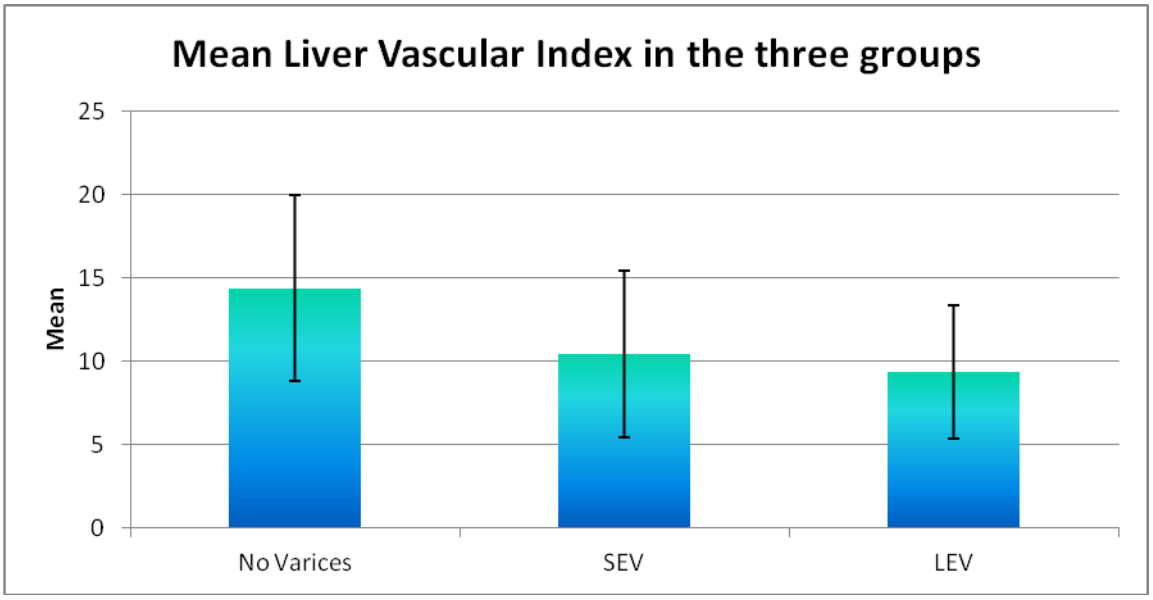


Figure 13: Comparison of significant Doppler parameters according to EV

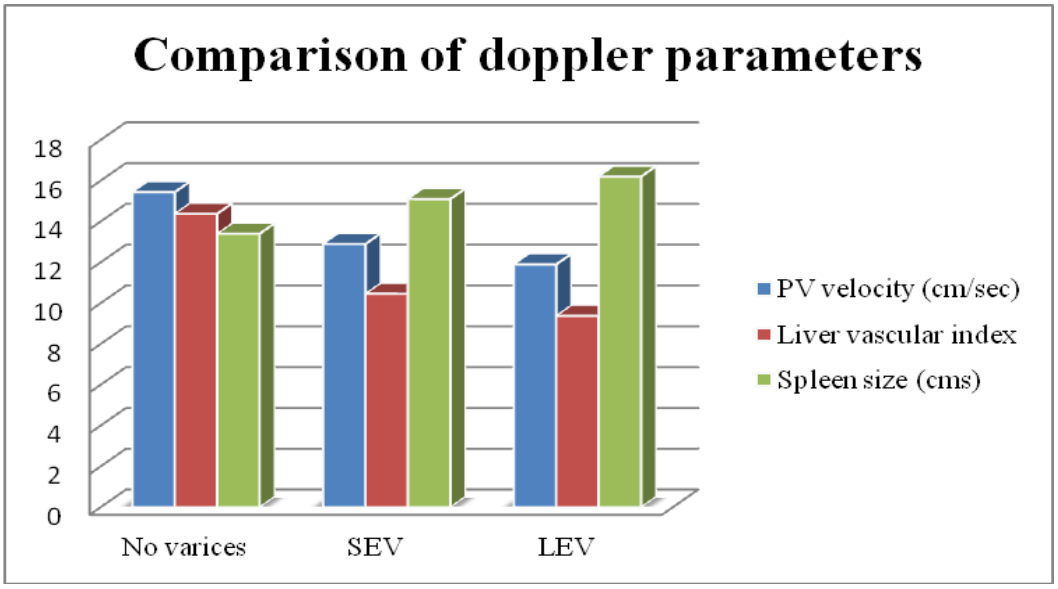


Figure 14: Mean P/S Ratio in the three groups

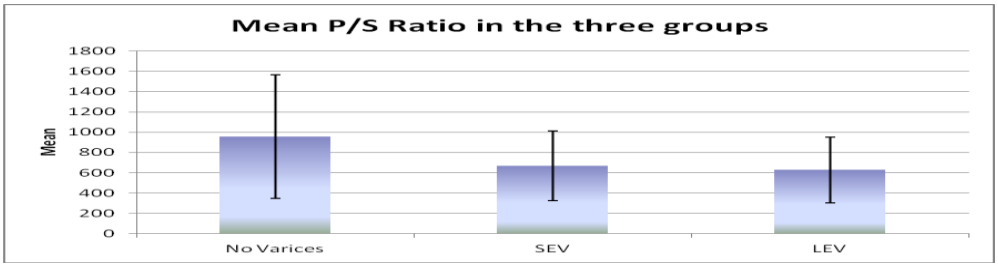


Figure 15: comparison of significant noninvasive parameters according to EV

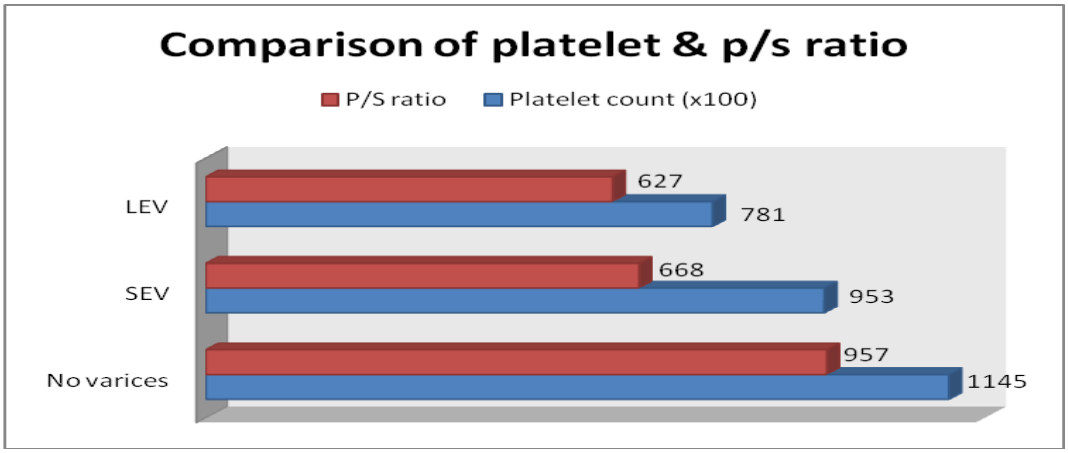


Table 10 shows the results of a logistic regression analysis in 99 patients. In this analysis no factors found to have independent predictive value for the presence of LEV.

Table 10: Logistic Regression Results:

Variables	P-Value	Odds Ratio
Group A	0.440	1[§]
Group B	0.761	1.778
Group C	0.360	5.511
MELD	0.559	0.880
Hb	0.904	0.975
Platelet	0.794	1.000
PT	0.047	0.692
INR	0.055	185.554
TB	0.764	0.915
AST	0.182	1.041
PV_DM	0.609	0.344
PV_Velocity	0.214	1.161
HARI	0.596	62.255
HAPI	0.613	0.393
PV_CS	0.764	1.333
Spleen size	0.114	0.692
SARI	0.053	168
Liver_Vascular index	0.315	1.212
Congestive_Index	0.271	19.845
PHT_Index	0.429	10.125
P/S_Ratio	0.568	0.999
APRI	0.175	0.000

DISCUSSION

Variceal gastrointestinal bleeding is a major complication of portal hypertension with significant morbidity and mortality. However, this complication occurs primarily in patients with LEV and is uncommon in those with small varices. Because the occurrence of variceal bleeding can be prevented using pharmacological agents like beta-adrenergic receptor antagonists, it is important to recognize patients who have LEV and are thus at a higher risk of developing variceal bleeding and likely to benefit from such interventions. It has therefore been recommended that patients with liver cirrhosis should be screened for the presence of LEV at the time of initial diagnosis and at periodic intervals thereafter throughout life. Efforts have been made to identify clinical, laboratory and imaging characteristics that may non-invasively predict the presence or absence of LEV with a high degree of accuracy, either reducing or eliminating the need for screening endoscopy. Few data have been published on predictors of LEV from India. Differences in the predictors found to be significant in various previous studies indicate that such studies may be necessary in our population. Such studies may be particularly indicated because of differences in the etiology of liver disease with a larger proportion of Indian patients being related to viral infections, the greater severity of liver disease in our patients because of their delayed presentation, and poorer nutritional status.

Our study, based on information achieved from 99 liver cirrhosis patients from tertiary care centre in south India, including 44 with LEV, showed that 6 factors had predictive ability for the presence of LEV on univariate analysis. These were Platelet count (P=0.049), Prothrombin time (P=0.030), Portal vein mean velocity (P=0.019), Liver vascular index (P=0.001), Spleen size (P=0.05) and Platelet count/spleen ratio (P=0.011). However, on multivariate analysis, no factors were found to have independent predictive value.

Our study population was composed mainly of patients with liver cirrhosis due to alcohol abuse or chronic hepatitis B infection, which represent more than 50% of the causes of liver cirrhosis, followed by cryptogenic in 27%.

In our study, there was no correlation between the presence of EV and CTP classification. These findings were also reported by other researchers, [31, 37, 92] but in a study by zaman et al. showed CTP class B or C were nearly 3 times more likely to have varices on endoscopy than CTP class A.[34]

MELD score also was not a good predictor of the presence of EV or LEV in our study. Burton et al.[100] and Levy et al.[101] also demonstrated no predictive value for MELD for the presence of EV. But in a recent study by Tafarel et al.[91]

showed that the presence of EV could be predicted by MELD score higher than 8 points (sensitivity 80.1% and specificity 51.2%).

The importance of platelet count has been alluded to in many studies. [30,33,34,37,87-90] The values of thrombocytopenia related to the presence of EV were different among various published studies.

Table 11: Comparison of platelet count in various studies

Study	Small varices	Large varices	P value
Zaman et al.[34]	107000	76000	0.001
Thomopoulos et al.[88]	126000	81000	<0.0001
FH Ng et al.[33]	160000	110000	0.01
Madhotra R et al.[37]	94000	62000	0.0003
Chalasani et al.[30]	-	<88000	0.0001
Giannini et al.[89]	177000	79000	<0.0001
Ismail et al.[92]	113000	91000	0.028
Tafrel et al.[91]	116000	90000	<0.01
Prihatini et al.[20]	161000	101000	0.003
Sharma SK et al.[87]	118000	84000	0.002
In this study	**95000**	**78000**	**0.049**

In our study platelet count less than 95000/mm^3 was associated with the presence of SEV and platelet count less than 78000/mm^3 associated with the presence LEV. This is in line with existing studies that have documented LEV with platelet count less than 100000/mm^3. Thrombocytopenia and EV are associated because both resulted from deterioration of liver functional reserve, leading to hemodynamic changes.

In our study, patients with LEV group had large spleen size (16.29±3.42 cm) in comparison to those in SEV (15.10±2.56cm) & no varices (13.34±4.20 cm) group with p =0.05. In the only available Indian study, Amarapurkar *et al.*[36] found that presence of splenomegaly was associated with presence of esophageal varices but not with LEV. In our study, LEV were more often associated with a splenomegaly (size>162mm, p=0.05) as has been observed in other parts of the world.

Table 12: comparison of Association of spleen size with presence of EV in various studies

Study	No varices	Small varices	Large varices	P value
Thomopoulos et al.[88]	-	131 mm	152 mm	0.032
FH Ng et al.[33]	-	102 mm	117 mm	0.02
Madhotra R et al.[37]	-	31%	62%	0.0001
Chalasani et al.[30]	-	-	75%	0.05
Giannini et al.[89]	-	110 mm	155 mm	<0.0001
Ismail et al.[92]	-	115 mm	158 mm	0.032
Amarapurkar N et al.[36]	17.9%	44%	37.7%	
Prihatini et al.[20]	-	101 mm	123 mm	0.007
In this study	**133 mm**	**151 mm**	**162 mm**	**0.05**

The presence of splenomegaly in cirrhotic patients is likely the result of vascular disturbances that are mainly related to portal hypertension. Even though low platelet count and splenomegaly were used as important predictors of presence of EV, the use of platelet count alone as a non-invasive predictor of EV can be misleading and cannot be solely attributed to portal hypertension. In patients with chronic liver disease the presence of decreased platelet count may depend on several factors other than portal hypertension, such as shortened

platelet mean lifetime, decreased thrombopoietin production, or myelotoxic effects of alcohol or hepatitis viruses. In this situation Giannini et al. [89] introduced a new parameter, platelet count/spleen diameter (p/s) ratio. He showed that platelet count/spleen diameter ratio with cut off value of 909 had 100% negative predictive value for non-invasively predicting the presence of EV in patients with either compensated or decompensated liver cirrhosis. 100% of patients with a p/s ratio >909 were free from EV. The platelet count/spleen diameter ratios represent an acceptable surrogate marker for clinically relevant portal hypertension. In concordance with the study by Giannini et al.[89] our study also showed similar results.

Table 13: comparison of Platelet/spleen diameter ratio for predicting EV

Study	No varices	SEV	LEV	P value
Giannini et al.	1638	-	533	<0.0001
In our study	957	668	627	0.011

In our study, **prothrombin time predicts** the presence of LEV in univariate analysis. But no other study demonstrated prothrombin time as a predictor of EV.

Among the hemodynamic characteristics of portal vein and hepatic artery, our study showed the Mean portal vein velocity (15.44±4.63 vs. 12.96±4.90 vs 11.91±3.97, p=0.019), and Liver vascular index (14.38±5.56 vs. 10.46±4.99 vs. 9.38±4.01, p=0.001) were the predictors of EV in univariate analysis. There was a significant association between the mean portal vein velocity and the size of varices (p = 0.019). According to Korner et al. the overall sensitivity for prediction of variceal bleeding in case of decreased portal vein mean velocity was 88%. Although none of other portal vein measurements had statistically significant associations with the size of varices, some articles have shown that portal vein diameter is also increased in large varices,[20,47] such a finding was not observed in our study. Iwao, et al, showed that not only portal venous velocity was significantly lower, but the hepatic arterial pulsatility index was also significantly higher in patients with esophageal varices.[50] Our study results did not disclose any significant associations between hepatic artery resistive index, pulsatility index and esophageal varices status. Various authors like Liu et al,[102] Shabestari et al.[85], Zhang et al.[76] also demonstrated no predictive value of HARI, HAPI, SARI for the presence of EV.

In a recent study by Tarzamni et al.[83] Portal vein diameter, congestion index (CI) (0.11 ± 0.03 vs. 0.06 ± 0.03, P < 0.0005), portal hypertensive index (2.62 ± 0.79 vs. 1.33 ± 0.53, P < 0.0005), and hepatic (0.73 ± 0.07 vs. 0.66 ± 0.07, P <

0.001) and splenic artery resistance index (SARI) (0.73 ± 0.06 vs. 0.62 ± 0.08, P < 0.0005) were found to be significantly higher in patients with LEV and portal vein flow velocity (13.25 ± 3.66 vs. 20.25 ± 5.05, P < 0.0005), liver vascular index (8.31 ± 2.72 vs. 17.8 ± 6.28, P < 0.0005) were significantly lower in patients with LEV. A logistic regression model confirmed spleen size >15.05cm, (P = 0.002) and portal hypertensive index (P = 0.040) as independent predictors for the occurrence of large esophageal varices (LEV). Our study correlates only with liver vascular index and portal vein velocity which was significantly lower in patient with LEV.

Table 14: Comparison Doppler parameters of our study with Tarzamni et al.[83]

Studies	Tarzamni et al.[83]			In our study		
Parameters	No EV	LEV	P value	No EV	LEV	P value
PVV (cm/sec)	15.26 ± 5.06	12.13 ± 2.59	0.001	15.44±4.63	11.91±3.97	**0.019**
PV DM (mm)	13.24 ± 2.55	14.54 ± 1.48	0.037	12.4±2.6	13.6±1.8	0.609
HARI	0.70 ± 0.06	0.80 ± 0.06	0.003	0.67±0.08	0.71±0.09	0.108
Spleen size (cm)	15.21 ± 2.99	17.62 ±3.1	0.003	13.34±4.20	16.29±3.42	**0.05**
SARI	0.69 ± 0.06	0.76 ± 0.11	< 0.0005	0.71±0.09	0.67±0.08	0.092
LVI	10.96 ± 5.05	6.48 ± 2.78	< 0.0005	14.38±5.56	9.38±4.01	**0.001**
CI	0.09 ± 0.03	0.14 ± 0.04	< 0.0005	0.10±0.13	0.12±0.15	0.816
PHTI	2.14 ± 0.77	3.18 ± 0.90	< 0.0005	0.04±0.06	0.06±0.16	0.444

Piscaglia et al[79] proposed a PHI cutoff of 1.2 s/m as the parameter with the highest accuracy (≈75%) for PHT and useful tool for detecting esophageal varices. Our study did not show significant association between PHI and esophageal varices.

Shabestari et al.[85] showed significant correlation between the size of esophageal varices and portal vein mean velocity (p=0.04) and logistic regression analysis did not show any significant associations between Doppler parameters and the size of esophageal varices. He also concluded that none of hepatic vasculature Doppler measurements had a significant role in predicting the size of esophageal varices.

Table 15: Comparison Doppler parameters of our study with Shabestari et al.[85]

Studies	Shabestari et al.			In our study		
Parameters	No EV	LEV	P value	No EV	LEV	P value
PVV (cm/sec)	15.5 (10.8-20.2)	10.47 (6.6-14.4)	0.08	15.44±4.63	11.91±3.97	**0.019**
PV DM (mm)	11.2 (9.9-12.5)	11.4 (9.3-13.5)	0.38	12.4±2.6	13.6±1.8	0.609
HARI	0.69 (0.60-0.79)	0.71 (0.68-0.75)	0.83	0.67±0.08	0.71±0.09	0.108
HAPI	1.46 (1.05-1.87)	1.38 (1.18-1.6)	0.70	1.13±0.34	1.35±0.38	0.117

De Bem, et al.[48] also revealed that there is no good correlation between Doppler ultrasound parameters of the portal system and the presence of gastroesophageal varices in cirrhotic patients. According to Liu et al.[102] mean PVV (*P* =0 .001), SARI (*P* =0.04), were predictive of the presence of esophageal varices at univariate analysis, but in multivariate logistic regression analysis, only mean PVV was independently associated with the presence of esophageal varices.

Table 16: Comparison Doppler parameters of our study with Liu et al.[102]

Studies	Liu et al.			In our study		
Parameters	No EV	LEV	P value	No EV	LEV	P value
PVV (cm/sec)	14.7±3.2	16.9±3.7	<0.001	15.44±4.63	11.91±3.97	**0.019**
HARI	0.75±0.06	0.76±0.08	0.42	0.67±0.08	0.71±0.09	0.108
HAPI	1.54±0.27	1.60±0.39	0.18	1.13±0.34	1.35±0.38	0.117
SARI	0.71±0.06	0.69±0.07	0.04	0.71±0.09	0.67±0.08	0.092

Our study is also in line with the above studies, in which portal vein diameter, HARI, HAPI, SARI, congestive index, portal hypertensive index did not predict the presence of esophageal varices. Multivariate logistic regression analysis did not show any significant association between Doppler parameters and esophageal varices.

SUMMARY

In the present study,

1. The most common etiology of cirrhosis in this part of country is Alcohol related liver disease (29%), followed by cryptogenic and Hepatitis B related liver disease (28% & 21% respectively).

2. Non-invasive parameters like Platelet count (P=0.049), Prothrombin time (P=0.030), Platelet count/spleen diameter ratio (P=0.011) predicted the presence of large esophageal varices.

3. Among the Colour Doppler Ultrasound study parameters, the Portal vein mean velocity (P=0.019), Liver vascular index (P=0.001), Spleen size>16.2 cm (P=0.05) predicted the presence of large esophageal varices, increasing the risk for upper gastrointestinal bleeding.

4. Other non-invasive parameters like CTP score, MELD, AST/Platelet ratio, Bilirubin, and Doppler parameters like portal vein diameter, hepatic artery resistive & pulsatility index, splenic artery resistive index, congestive index, portal hypertensive index did not predict the presence of either small or large varices.

CONCLUSION

In conclusion, results of our study indicate that non-invasive tools like platelet count, prothrombin time, platelet/spleen diameter ratio, spleen size >16.2 cm, and Doppler parameters like portal vein velocity, liver vascular index are predictors of presence of large esophageal varices.

But there were no independent noninvasive predictors of large esophageal varices by multivariate analysis in our study. Values for the noninvasive indicators from this study and comparables need to be validated by randomised prospective studies.

Applying the non-invasive techniques including hepatic vessel hemodynamics by Doppler study for the detection of esophageal varices and assess the risk for bleeding may be cost effective and safer than the “scope all strategy”. But further randomised studies are needed to evaluate the accurate predictors of esophageal varices in our population. Till then Upper Gastrointestinal endoscopy remains the gold standard procedure for screening esophageal varices and assessing risk for bleeding.

BIBILOGRAPHY

1. De Franchis R, Primignani M. Natural history of portal hypertension in patients with cirrhosis. Clin Liver Dis 2001; 5:645–63.
2. Garceau AJ, Chalmers TC. The Boston inter-hospital liver group. The natural history of cirrhosis: I. Survival with oesophageal varices. N Engl J Med 1963; 268:469–73.
3. Graham D, Smith JL. The course of patients after variceal hemorrhage. Gastroenterology 1981; 80:800–9.
4. Christensen E, Fauerholdt L, Schlichting P et al. Aspects of the natural history of gastrointestinal bleeding in cirrhosis and the effect of prednisone. Gastroenterology. 1981; 81:944-52.
5. Cales P, Pascal JP, Natural history of esophageal varices in cirrhosis (from origin to rupture) Gastroenterology Clin Biol. 1990; 12:245-54.
6. The Northern Italian Endoscopic Club for the Study and Treatment of Esophageal Varices. Prediction of the first variceal hemorrhage in patients with cirrhosis of the liver and esophageal varices: A prospective multicenter study. N Engl J Med 1988;319:983–9
7. D'Amico G, Pagliaro L, Bosch J. Pharmacological treatment of portal hypertension: an evidence-based approach. Semin Liver Dis 1999; 19: 475–505.

8. Christensen E, Fauerholtdt L, Schlichting P, JuhI E, Poulsen H, Tygstrap N, et al. Aspects of the natural history of gastrointestinal bleeding in cirrhosis and the effect of prednisone. Gastroenterology 1981; 81:944–52.
9. Cales P, Desmorat H, Vinel JP, Caucanas JP, Ravaud A, Gerin P, et al. Incidence of large oesophageal varices in patients with cirrhosis: application to prophylaxis of first bleeding. Gut 1990; 31:1298–1302.
10. Merli M, Nicolini G, Angeloni S, Rinaldi V, Desantis A, Markel C, et al. Incidence and natural history of small esophageal varices in cirrhotic patients. J Hepatol 2003; 38(3):266–72.
11. Zoli M, Merkel C, Magalotti D, Gueli C, Grimaldi M, Gatta A, et al. Natural history of cirrhotic patients with small esophageal varices: a prospective study. Am J Gastroenterol 2000; 95(2):503–8.
12. Jutabha R. Jensen DM. Management of upper gastrointestinal bleeding in the patient with chronic liver disease. Med Clin North Am. 1996:80(5):1035-68.
13. Anand CS, Tandon BN, Nundy S. The causes, management and outcome of upper gastrointestinal haemorrhage in an Indian hospital, Br J Surg. 1983:70(4):209-11.
14. Rigo GP, Merighi A, Chalen JN, et al. A prospective study of the ability of three endoscopic classifications to predict hemorrhage from esophageal varices. Gastrointest Endosc 1992; 38:425–9.

15. Nevens F, Bustami R, Scheys I, Lesaffre E, Fevery J. Variceal pressure is a factor predicting the risk of a first variceal bleeding: a prospective cohort study in cirrhotic patients. *Hepatology* 1998; 27: 15-19.

16. Navarro VJ, Garcia TSao G. Variceal haemorrhage. Crit Care Clin. 1991;11:312

17. Burroughs A, Hezzanolte G, Philps A et al., Cirrhosics with variceal haemorrhage: The importance of the time interval between admission and the start of analysis for survival and rebleeding rates. Hepatology. 1969; 9; 801.

18..Gores GJ, Weisner RH, Dickson ER, et al. Prospective evaluation of esophageal varices in primary biliary cirrhosis: Development, natural history and influence in survival. Gastroenterology 1989;96:1552–9

19. Silverstein FE, Gilbert DA, Tedesco FJ, et al. The national ASGE survey on upper gastrointestinal bleeding clinical prognostic factors. Gastroint. Endos. 1981; 27:80-93.

20. Prihartini J, Lesmana LA, Manan C, Gani RA et al. Detection of esophageal varices in liver cirrhosis using non-invasive parameters. Acta Med Indones – Indones J Intern Med. 2005; 37(3):126-131.

21. Bernard B, Grange JD, Khac EN et al. Antibiotic prophylaxis for the prevention of bacterial infections in cirrhotic patients with gastrointestinal bleeding: A meta-analysis. Hepatology. 1999; 29:1655-61.

22. Grace ND, Groszmann RJ, Garcia-Tsao G, et al. Portal hypertension and variceal bleeding: an AASLD single topic symposium. Hepatology 1998; 28:868–80.

23. De Franchis R et al. On behalf of the Baveno V Faculty, Revising consensus in portal hypertension: Report of the Baveno V consensus workshop on methodology of diagnosis and therapy in portal hypertension. Journal of Hepatology 2010 vol. 53: 762–768

24. D'Amico G, Pagliaro L, Bosch J. The treatment of portal hypertension: A meta- analytic review. Hepatology 1995; 22:332–54.

25. Spiegel BM, Targownik L, Dulai GS, Karsan HA, Gralnek IM. Endoscopic screening for esophageal varices in cirrhosis: is it ever cost effective? Hepatology 2003;37(2):366–77.

26. Schepis F, Camma C, Niceforo D, Magnano A, Pallio S, Cinquegrani M, et al. Which patients with cirrhosis should undergo endoscopic screening for esophageal varices detection? Hepatology 2001; 33:333- 8.

27. Nashef SA, Pagliero KM. Instrumental perforation of the esophagus in benign disease. Ann Thorac Surg 1987; 44(4):360–2.

28. Norfleet RG, Mitchell PD, Mulholland DD, Philo J. Does bacteremia follow upper gastrointestinal endoscopy? Am J Gastroenterol 1981; 76(5):420–2.

29. Assy N, Rosser BG, Grahame GR, Minuk GY. Risk of sedation for upper GI endoscopy exacerbating subclinical hepatic encephalopathy in patients with cirrhosis. Gastrointest Endosc 1999; 49:690–4.

30. Chalasani N, Imperiale TF, Ismail A, et al. Predictors of large esophageal varices in patients with cirrhosis. Am J Gastroenterol 1999; 94:3285–91.

31. Zaman A, Hapke R, Flora K, et al. Factors predicting the presence of esophageal varices or gastric varices in patients with advanced liver disease. Am J Gastroenterol 1999; 94:3292–6.

32. Pilette C, Oberti F, Aube C, et al. Non-invasive diagnosis of esophageal varices in chronic liver disease. J Hepatol 1999; 31: 867–73.

33. Ng FH, Wong SY, Loo CK, et al. Prediction of oesophagogastric varices in patients with cirrhosis. J Gastroenterol Hepatol 1999; 14:785–90.

34. Zaman A, Becker T, Lopidus J, et al. Risk factors for the presence of varices in cirrhotic patients without a history of variceal hemorrhage. Arch Intern Med 2001; 161:2564–70.

35. Gorka W, Al Mulla A, Al Sebayel M, et al. Qualitative hepatic venous Doppler sonography versus portal flow-metry in predicting the severity of esophageal varices in hepatitis C cirrhosis. Am J Roentgenol 1997; 169:511–5.

36. Amarapurkar DN, Parikh SS, Shankaran K, *et al.* Correlation between splenomegaly and oesophageal varices in patients with liver cirrhosis. *Endoscopy* 1994; 26:563.

37. Madhotra R, Mulcahy HE, Willner I, et al. Prediction of esophageal varices in patients with cirrhosis. J Clin Gastroenterol 2002; 34:81–5.

38. Zeijen RNM, Caenepeel P, Stockbru¨gger RW, et al. Prediction of esophageal varices in liver disease: preliminary results. Gastroenterology 1994; 106:A1013.

39. Lavergne J, Molina E, Reddy KR, et al. Ascites predicts the presence of high grade varices by screening gastroscopy. Gastrointest Endosc 1997; 45:A187.

40. Giannini EG, Botta F, Borro P, Dulbecco P, Testa E, Mansi C, et al. Application of the platelet count/spleen diameter ratio to rule out the presence of oesophageal varices in patients with cirrhosis: a validation study based on follow-up. Dig Liver Dis 2005; 37(10):779–85.

41. Garcia-Tsao G, Escorsell A, Zakko M, et al. Predicting the presence of significant portal hypertension and varices in compensated cirrhotic patients. Hepatology 1997; 26:A360.

42. Freeman JG, Darlow S, Cole AT. Platelet count as a predictor for the presence of oesophageal varices in alcoholic cirrhotic patients. Gastroenterology 1999; 116:A1211.

43. Riggio O, Angeloni S, Nicolini G, et al. Endoscopic screening for esophageal varices in cirrhotic patients. Hepatology 2002;35:
501–2.

44. Erdozain Sosa JC, Martin Hervas C, Morena Blanco MA, Zapata Aparicio I, Herrera Abian A, Conde Gacho P, et al. Color duplex Doppler ultrasonography in the evaluation of the risk of esophageal varices bleeding in cirrhotic patients. Gastroenterol Hepatol 2000; 23 (10):466-9 [Abstr].

45. Korner T. Portal duplex sonography in liver cirrhosis. A useful supplement to endoscopic evaluation of bleeding risk of esophageal varices? Scand J Gastroenterol 1996; 31(5):495-9.

46. Yin XY, Lu MD, Huang JF, Xie Xy, Liang LJ. Color Doppler velocity profile assessment of portal hemodynamics in cirrhotic patients with portal hypertension: correlation with esophageal variceal bleeding. J Clin Ultrasound 2001; 29(1):7-13.

47. Sarwar S, Khan AA, Alam A, Butt AK, Shafqat F, Malik K, et al. Non endoscopic prediction of presence of esophageal varices in cirrhosis. J Coll Physicians Surg Pak 2005; 15(9):528-31.

48. De Bem RS, Lora FL, De Souza RCA, Trippia MA, Amarante HM, Carmes ER. Correlation of Doppler ultrasound of the portal system with endoscopic changes caused by portal hypertension in cirrhotic patients. Arquivos de Gastroenterologia 2006; 43(3):178-83 [Abstr].

49. Letard JC, Boustiere C, Romy P, Jouffre C, Patouillard B, Etaix JP, et

al. Reversal of portal blood flow in cirrhosis: Clinical, endoscopic and endosonographic correlation in 72 patients. Gastroenterologie Clinique et Biologique 1993;17(8-9):578-83 [Abstr].

50. Iwao T, Toyonaga A, Oho K, Tayama C, Masumoto H, Sakai T, et al. Value of Doppler ultrasound parameters of portal vein and hepatic artery in the diagnosis of cirrhosis and portal hypertension. Am J Gastroenterol 1997; 92(6):1012-17.

51. Bosch J, D'Amico, Juan C. García-Pagán. Portal Hypertension and Nonsurgical Management. Schiff's Diseases of the Liver, 10th Edition, Lippincott Williams & Wilkins: 2007;vol(1):420-475

52. Boyer Thomas D. Portal hypertension and its complication. In Zakim and Boyer (Eds). WB Saunders Company. 1982;466

53. La Mont JT, Koff RS, Isselbacher KJ. Cirrhosis. In petersdorf, Adams, Braunwald, Isselbacher Martin, Wilson (Eds) Harrisons Principles of Medicine 10th ed. Tata McGraw Hill, 1983.

54. Bosch J, Garcia-Pagan JC. Complications of cirrhosis. I. Portal hypertension. *J Hepatol* 2000; 32(1 Suppl):141–156.

55. Vianna A, Hayes PC, Moscoso G, et al. Normal venous circulation of the gastresophageal junction. A route to understanding varices. Gastroenterology 1987; 93:876–889.

56. Bhathal PS, Grossman HJ. Reduction of the increased portal vascular resistance of the isolated perfused cirrhotic rat liver by vasodialtors. J Hepatol. 1985; 1:325-37.

57. Guptha TK, Chung MK, Toruner M, Groszmann RJ. Endothelial dysfunction in the intrahepatic microcirculation of the cirrhotic rat. Hepatology. 1998;28:926-31

58. Wiest R, Groszmann RJ. Nitric Oxide and portal hypertension: its role in the regulation of intrahepatic and splanchnic vascular resistance. Sem Liv Dis. 2000; 19:411-26.

59. Garcia-Tsao G, Groszmann RJ, Fisher RL, Conn HO, Atterbury CE, Glickman M. Portal pressure, presence of gastroesophageal varices and variceal bleeding. Hepatology 1985; 5:419-424.

60. Pagliaro L, D'Amico G, Pasta L, et al. Portal hypertension in cirrhosis: Natural history. In: Bosch J, Groszmann RJ. Eds. Portal Hypertension: Pathophysiology and treatment. Oxford: Blackwell Scientific;1994:72-92.

61. Lebrec D, De Fleury P, Rueff B et al. Portal hypertension, size of esophageal varices, and risk of gastrointestinal bleeding in alcoholic cirrhosis. Gastroenterology. 1980; 79:1139-44.

62. Garcia-Tsao G, Sanyal AJ, Grace ND, et al. The Practice Guidelines Committee of the American Association for the Study of Liver Diseases and the Practice Parameters Committee of American College of Gastroenterology. Am J Gastrenterol.2007; 102:2086-102.

63. Bosch J, Garcia-Pagan JC, Prevention of variceal rebleeding. Lancet 2003; 361:952-954.

64. Paquet KJ. Prophylactic endoscopic sclerosing treatment of esophageal wall in varices: A prospective controlled trial. Endoscopy 1982;14:4-5

65. Perello A, Escorsell A, Bru C, et al. Wedged hepatic venous pressure adequately reflects portal pressure in hepatitis C virus related cirrhosis. Hepatology. 1999; 30:1393-7.

66. Turnes J, Garcia-Pagan JC, Abraldes JG, et al. Pharmacological reduction of portal pressure and long term risk of first variceal bleeding in patients with cirrhosis. Am J Gastroenterol. 2006; 101:506-12.

67. De Francis R. Evolving consenses in portal hypertension. Report of the Baveno IV consensus workshop on methodology of diagnosis and therapy in portal hypertension. J Hepatol. 2005;43:167-76

68. **Piscaglia F**, Donati G, Serra C, Muratori R, Solmi L, Gaiani S, Gramantieri L, Bolondi L. Value of splanchnic Doppler ultrasound in the diagnosis of portal hypertension. *Ultrasound Med Biol* 2001; **27**: 893-899

69. **Li FH**, Hao J, Xia JG, Li HL, Fang H. Hemodynamic analysis of esophageal varices in patients with liver cirrhosis using color Doppler ultrasound. *World J Gastroenterol* 2005; **11**: 4560-4565

70. Dick R, Watkinson A, Olliff JFC, Liver and spleen: In David Sutton (Ed). Text book of Radiology and imaging 7th ed. Churchill Livingstone.

71. Zwiebel WJ. Vascular disorders of the liver. In Zwiebel (Ed):Introduction to vascular Ultrasonography 4th ed. WB Saunders Company, 2000.

72. Freeny PC, ItaiY. Portal venous system. In Margulis and Burhenne's (Eds): Alimentary Tract Radiology 5th ed, 1994.

73. Baker EL, Kerlan RK Jr. In Gazelle Saini Mueller (Eds) Hepatobiliary and pancreatic radiology imaging and intervention. Imaging of portal hypertension, 1998.

74. Koda M, Murawaki Y, Kawasaki H, Ikawa S. Portal blood velocity and portal blood flow in patients with chronic viral hepatitis: relation to histological liver fibrosis. Hepatogastroenterology 1996; 43:199–202.

75. Paulson EK, Klicwer MA, Frederich MG, et al. Hepatic artery: Variability in measurement of resistive index and systolic acceleration time in healthy volunteers. Radiology 1996; 200:725-29.

76. Zhang Li, Duan, Jin-Mao-Li, Ji-Kai yin, Hemodynamic Features of Doppler Ultrasonography in Patients with Portal Hypertension. J Ultrasound Med 2007; 26:1689–1696.

77. Noguera AM, Montserrat Em Torrubis S etal. Doppler in hepatic cirrhosis and chronic hepatitits. Seminars in Ultrasound CT and MRI 2002;23(1):19-36

78. Haag K, Rossle M, Ochs A, et al. Correlation of duplex sonography findings and portal pressure in 375 patients with PHT. AJR Am J Roentgenol 1999; 172:631–635.

79. Piscaglia F, Donati G, Serra C, et al. Value of splanchnic Doppler ultrasound in the diagnosis of portal hypertension. Ultrasound Med Biol 2001; 27:893–899.

80. Sabba C, Merkel C, Zoli M, Ferraioli G, Gaiani S, Sacerdoti D, Bolondi L. Interobserver and interequipment variability of echo-Doppler examination of the portal vein: effect of a cooperative training program. *Hepatology* 1995; 21: 428-433.

81. Piscaglia F, Gaiani S, Zironi G, Gramantieri L, Casali A, Siringo S, Serra C, Bolondi L. Intra- and extrahepatic arterial resistances in chronic hepatitis and liver cirrhosis. *Ultrasound Med Biol* 1997; 23: 675-682.

82. Sacerdoti D, Gaiani S, Buonamico P, Merkel C, Zoli M, Bolondi L, Sabba C. Interobserver and interequipment variability of hepatic, splenic, and renal arterial Doppler resistance indices in normal subjects and patients with cirrhosis. *J Hepatol* 1997; 27: 986-992.

83. Tarzamni MK, Somi MH, Farhang S, Jalilvand M, Portal hemodynamics as predictors of high risk esophageal varices in cirrhotic patients. *World J Gastroenterol* 2008 March 28; 14(12): 1898-1902.

84. Moriyasu F, Nishida O, Ban N, Nakamura T, Sakai M, Miyake T, Uchino H. "Congestion index" of the portal vein. *AJR Am Roentgenol* 1986; 146: 735-739.

85. Shabestari A, Nikoukar E, Bakhshandeh H et al. Hepatic Doppler Ultrasound in Assessment of the Severity of Esophageal Varices in Cirrhotic Patients. Iran. J. Radiol. Spring 2007;4(3):151-158

86. Haktanir A, Cihan BS, Çelenk C, Cihan S, Value of Doppler Sonography in Assessing the Progression of Chronic Viral Hepatitis and in the Diagnosis and Grading of Cirrhosis. J Ultrasound Med 2005; 24:311–321.

87. Sharma SK, Aggarwal R. Prediction of large esophageal varices in patients with cirrhosis of the liver using clinical, laboratory and imaging parameters. *J Gastroenterol Hepatol* 2007; 22: 1909-1915

88. Thomopoulos KC, Labropoulou-Karatza C, Mimidis KP, Katsakoulis EC, Iconomou G, Nikolopoulou VN. Non-invasive predictors of the presence of large oesophageal varices in patients with cirrhosis. *Dig Liver Dis* 2003; 35: 473-478.

89. 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; 52: 1200-1205.

90. Bressler B, Pinto R, El-Ashry D, Heathcote EJ. Which patients with primary biliary cirrhosis or primary sclerosing cholangitis should undergo

endoscopic screening for oesophageal varices detection? *Gut* 2005; 54: 407-410.

91. Tafarel JR, Tolentino LH, Correa LH, Bonilha DR, et al. Prediction of esophageal varices in hepatic cirrhosis by noninvasive markers. European Journal of /gastroenterology & Hepatology. 2011;23:754-758.

92. Ismail FW, Shah HA, Hamid S, Abbas Z, Abid S, Mumtaz K, Jafri W et al. Noninvasive predictors of large varices in patients hospitalized with gastroesophageal variceal hemorrhage. Hepatol Int (2008) 2:124–128.

93. Sanyal AJ, Fontana RJ, Di Bisceglie AM, Everhart JE, Doherty MC, et al. HALT-C Trial group. The prevalence and risk factors associated with esophageal varices in subjects with hepatitis C and advanced fibrosis. Gastrointest Endosc 2006;64:855-864.

94. Sebastiani G, Tempesta D, Fattovicb G, Casterz L, Halfon P, Bourliere M et al. Prediction of esophageal varices in hepatic cirrhosis by simple serum non-invasice markers-results of a multicenter, large-scale study. J Hepatol 2010; 53:630-638.

95. Baik SK, Jeong PH, Ji SW, et al. Acute hemodynamic effects of octreotide and terlipressin in patients with cirrhosis: a randomized comparison. Am J Gastroenterol. 2005; 100:631-635.

96. Garcia-Pagan JC, Bosch J. Endoscopic band ligation in the treatment of portal hypertension. Nat Clin Pract Gastroenterol Hepatol. 2005; 2:526-535.

97. Avgerinos A, Armonis A, Stefanidis G, et al. Sustained rise of portal pressure after sclerotherapy,but not band ligation, in acute variceal bleeding cirrhosis. Hepatology. 2004; 39:1623-30.

98. Lo GH, Lai KH, Cheng JS, Chen MH, Huang HU, et al. Endoscopic variceal ligation plus nadolol and sucralfate compared with ligation alone for the prevention of variceal rebleeding: a prospective, randomized trial. Hepatology. 2000; 32:461-465.

99. De la Pena J, Brullet E, Sanchez-Hernandez E, Rivero M, Vergara M, et al. Variceal ligation plus nadolol compared with ligation for prophylaxis of variceal rebleeding: a amulticenter trial. Hepatology 2005; 41:572-578.

100. Burton JR, Liangpunsakul S, Lapidus J, Giannini E, Chalasani N, Zaman A. Validation of a multivariate model predicting presence and size of varices. J Clin Gastroenterol. 2007; 41:609-615.

101. Levy C, Zein CO, Gomez J, et al. Prevalence and predictors of esophageal varices in patients with primary biliary cirrhosis. Clin Gastroenterol Hepatol 2007; 5:803-808.

102. Liu CH, Hsu S, and Liang, Tsai F et al. Esophageal varices: Noninvasive diagnosis with Duplex Doppler US in patients with compensated cirrhosis. Radiology 2008; 248:132-139.

ABBREVIATIONS

PHT	- Portal Hypertension
EV	- Esophageal varices
SEV	- Small esophageal varices
LEV	- Large esophageal varices
PPG	- Portal Pressure Gradient
CTP	- Child Turcot Pugh
MELD	- Model for End stage liver disease
HVPG	- Hepatic vein pressure gradient
WHVP	- Wedged Hepatic vein pressure
FHVP	- Free hepatic vein pressure
PVV	-Portal Vein Velocity
PV DM	-Portal vein diameter
PV CSA	-Portal vein cross sectional surface area
HARI	- Hepatic Artery Resistance Index
HAPI	- Hepatic Artery Pulsatility Index
SARI	- Splenic Artery Resistive Index
LVI	- Liver Vascular Index
CI	- Congestive Index
PHI	- Portal Hypertension Index
P/S	- Platelet count / Spleen diameter ratio
APRI	- AST/Platelet Ratio Index
CVH	- Chronic Viral Hepatitis

TIPS	-Transjugular intrahepatic portosystemic shunt
EVL	-Endoscopic Variceal band Ligation
HBsAg	-Hepatitis B surface Antigen
HBV DNA	-Hepatits B Virus Deoxyribonucleic acid
Anti HCV	-Anti Hepatits C virus
HCV RNA	-Hepatitis C virus Ribonucleic Acid

CODING FOR MASTER CHART

1. Sex - 1- Male, 2- Female
2. Literacy - 1- Literate, 2-Illiterate
3. Alcohol consumption – 1-Yes, 2- No
4. Smoking - 1-Yes, 2- No
5. CTP class - 1-Class A, 2- Class B, 3-Class C
6. Ascites - 1-Present, 2- Absent
7. Hepatic Encephalopathy - 1-Present, 2- Absent
8. HRS - 1-Present, 2- Absent
9. SBP - 1-Present, 2- Absent
10. Coagulopathy - 1-Present, 2- Absent

S.No.	Name	Age	Sex	MGE no	Etiology	Literacy	Alcohol	Smoking	CTP score	CTP grade	MELD	Ascites	HE	HRS	SBP	COAGULOPATHY	Previous H/o bleed	Hb(gms%)	Platelet count	PT	INR	Creatinine	TB	AST	Albumin
1	Jayaraman	56	1	579/12	Alcohol	1	1	1	9	2	16	1	2	2	2	2	1	7.6	105000	19	1.4	1.69	1.2	63	2
2	Noor basha	55	1	428/12	Alcohol	1	1	1	8	2	6	1	2	2	2	2	2	9.9	217000	14	1	0.65	0.9	60	2
3	Nagendran	45	1	640/12	Alcohol	1	1	1	14	3	26	1	1	2	2	1	1	5.9	138000	31	2.4	0.84	14.3	65	1.6
4	Ramadass	44	1	3573/10	Hepatitis B	1	2	2	6	1	8	2	2	2	2	2	1	9.6	81000	15	1.1	0.98	1.3	130	3.2
5	perumal	56	1	7035/11	Hepatitis B	1	2	2	5	1	11	2	2	2	2	1	2	6.9	85000	20	1.5	0.85	1.03	21	3.7
6	Ravi	45	1	6052/11	Alcohol+Hepatits B	1	1	2	10	3	17	1	1	2	2	2	1	12.8	85000	16	1.3	1.07	6.1	128	2.8
7	Chokkalingam	32	1	6642/11	cryptogenic	1	2	2	9	2	10	1	1	2	2	1	2	10.4	49000	19	1.4	0.59	0.68	45	2.3
8	Sathish	33	1	204/12	Alcohol	1	1	2	11	3	22	1	2	2	1	1	2	10	96000	27	1.8	0.69	10	297	2
9	Jaganathan	43	1	707/12	Hepatitis B	1	2	2	9	2	18	1	2	1	2	2	2	8.5	72000	18	1.3	1.86	2.1	91	2
10	Kali	48	1	275/10	Hepatitis B	2	2	1	10	3	16	1	2	2	2	1	1	10.9	99000	22	1.6	0.65	3	123	2
11	Sekar	35	1	4804/10	Alcohol	1	1	1	5	1	11	2	2	2	2	2	1	14.6	50000	19	1.4	0.62	1.2	36	3.6
12	Dhivakar	28	1	6840/10	Alcohol	1	1	2	6	1	15	2	2	2	2	2	1	10.7	192000	17	1.2	0.3	5.09	137	3.5
13	Sivalingam	53	1	6700/11	Hepatitis C	1	1	2	7	2	10	1	2	2	2	2	1	10.3	60000	19	1.4	1	1	73	2.5
14	Visalatchi	70	2	5010/10	cryptogenic	2	2	2	8	2	10	1	2	2	2	2	1	7.1	155000	15	1.1	0.7	1.9	51	2.2
15	Panch peer	42	1	6538/04	cryptogenic	1	2	2	6	1	9	1	2	2	2	2	1	11.2	99000	16	1.2	0.8	1.2	49	3.9
16	Sakkiya banu	18	2	3515/09	cryptogenic	1	2	2	8	2	9	1	2	2	2	2	2	7.4	29000	15	1.1	0.7	1.5	27	2.3
17	Vasudevan	51	1	4439/10	Hepatitis B	1	2	2	6	1	11	1	2	2	2	2	1	9.3	162000	16	1.1	1.02	0.9	51	3.6
18	Srinivasan	18	1	3678/10	cryptogenic	1	2	2	9	2	16	1	2	2	2	2	1	6.3	90000	20	1.5	0.5	3.7	72	3.1
19	Rosy	47	2	4196/01	cryptogenic	1	2	2	6	1	8	2	2	2	2	2	1	5.8	70000	18	1.2	0.7	0.6	40	2.4
20	Palani	55	1	312/11	alcohol	2	1	1	8	2	7	2	2	2	2	2	2	7.2	79000	20	1.9	0.9	1.2	65	2.1
21	Kokila	28	2	474/11	cryptogenic	1	2	2	5	1	9	2	2	2	2	2	1	10	100000	18	1.3	0.53	0.8	36	3.7
22	Stellamary	43	2	6249/11	cryptogenic	1	2	2	8	2	7	1	2	2	2	2	1	2.6	24000	16	1.1	0.6	0.2	24	1.2
23	Gunasekar	54	1	2609/11	Hepatitis B	1	1	1	7	2	10	2	2	2	2	2	2	12.8	50000	17	1.2	0.95	1.6	82	2.8
24	Ramani	22	2	5650/11	cryptogenic	1	2	2	6	1	6	1	2	2	2	2	2	12.1	26000	14	1	0.6	0.4	32	4.3
25	Vargese	50	1	259/10	Hepatitis B	1	2	2	9	2	12	1	1	2	1	1	1	10.6	91000	19	1.4	0.6	1.6	122	2.4
26	Rajasekar	60	1	3089/11	Hepatitis B	1	1	1	6	1	8	2	2	2	2	2	1	12.6	88000	14	1	1.19	0.5	74	2.8
27	Malligabegam	46	2	285/11	Hepatitis B	2	2	2	7	2	9	1	2	2	2	2	2	10.1	109000	16	1.1	0.9	1.4	23	2.6
28	Chakkarapani	25	1	4869/10	Hepatitis B	1	2	2	7	2	15	2	2	2	2	2	2	8.7	71000	30	2.2	0.75	1	70	2.4
29	Sekar	42	1	6702/11	Alcohol	1	1	1	7	2	13	1	2	2	2	1	1	8.9	88000	19	1.5	0.9	1.8	75	2.9
30	Anusuya	60	2	3403/09	Hepatitis C	2	2	2	10	3	20	2	2	2	2	2	1	4.5	54000	24	2	1.2	2.8	67	2.1
31	Nagaraj	42	1	510/11	Alcohol	2	1	1	10	3	16	1	1	2	2	2	1	12.3	61000	18	1.2	0.98	5.2	121	2.6
32	Venkataraman	49	1	5217/11	Alcohol	2	2	1	7	2	14	1	2	2	2	2	1	7.9	87000	21	1.5	1.37	0.5	69	2.8
33	Sakunthala	45	2	2265/07	cryptogenic	2	2	2	5	1	10	2	2	2	2	2	1	10.9	100000	16	1.4	1	1	35	3.9

Endoscopy	Doppler study												P/S Ratio	APRI
	Liver size (cm)	PV DM(cm)	PV velocity (cm/se	HARI	HAPI	PV cs area(cm2)	spleen size (cm)	SARI	Liver vascular index	Congestive index	PHT INDEX	Collaterals		
3	9.5	0.67	7.5	0.8	1.76	0.2	17.4	0.76	4.26	0.03	0.049	2	603	0.1
1	12.3	0.9	10.8	0.64	1.09	0.36	10.17	0.71	9.91	0.033	0.03	2	213	0
2	12.7	1.6	18.3	0.5	0.8	1.42	12.91	0.7	22.9	0.078	0.011	2	1070	0.1
3	12.6	1.25	10.7	0.69	1.13	0.55	21.5	0.56	9.47	0.05	0.022	1	377	0.2
3	11.8	1.3	8.4	0.64	1.2	1.4	17.2	0.64	7	0.167	0.029	2	494	0
3	11.5	1.43	6.9	0.8	1.55	0.69	14.4	0.57	4.45	0.1	0.04	2	590	0.2
2	10	0.65	9.1	0.75	1.75	0.05	19	0.73	5.2	0.0054	0.036	1	251	0.1
2	11.9	0.7	6	0.8	1.74	0.07	14.9	0.67	3.45	0.012	0.053	1	644	0.3
2	9.8	0.8	12.2	0.65	1.15	0.14	16.8	0.6	10.61	0.011	0.019	1	429	0.1
2	10.2	1.03	13.6	0.78	1.25	0.67	14.2	0.72	10.88	0.049	0.025	1	697	0.1
2	13.5	1.12	8.4	0.56	0.95	1.07	21.8	0.53	8.84	0.127	0.173	2	229	0.1
3	13.9	0.86	14.1	0.78	1.5	1.14	14.4	0.61	9.4	0.99	0.017	1	1333	0.1
1	14.3	1.3	15	0.42	0.57	0.93	18.8	0.9	29	0.062	0.014	2	319	0.1
3	9.8	0.9	17.2	0.6	1.03	0.56	8.2	0.62	16.7	0.032	0.177	2	1890	0.3
2	12.6	1.2	10.3	0.8	2.5	1.58	20	0.69	4.12	0.153	0.032	2	495	0.1
1	12.3	1.9	15.2	0.75	1.5	3.72	23.2	0.94	10.1	0.24	0.028	2	125	0.1
3	14.3	1.1	9	0.5	0.7	0.54	13.7	0.6	12.8	0.06	0.005	2	1182	0
3	12.8	1.05	18.5	0.86	1.21	0.5	19.6	0.7	15	0.027	0.195	2	459	0.1
2	9.8	1.18	14.2	0.88	2.9	0.6	17.9	0.7	4.89	0.21	0.026	2	391	0.1
3	10.2	1.3	19.2	0.6	1.8	1.9	12.3	0.6	10.6	0.098	0.011	2	642	0.1
3	12	1.5	20	0.8	1.6	1.4	18.5	0.7	12.5	0.07	0.0163	2	540	0
3	12.9	1.2	10	0.61	1	1.85	16.5	0.74	10	0.185	0.27	1	570	0
3	11.5	1.15	13.4	0.5	0.67	1.08	20	0.53	20	0.081	0.0118	2	250	0.2
1	12.8	1.4	16.4	0.67	1	1.04	9.6	0.76	16.4	0.063	0.0186	2	2708	0
3	12.2	1.33	15.4	0.71	1.14	0.66	16.2	0.76	13.51	0.043	0.021	2	562	0.1
3	9.9	1.4	7.5	0.68	1.2	0.92	11.9	0.62	6.25	0.123	0.035	2	740	0.1
1	10.5	1.27	14.5	0.75	1.32	0.37	11.8	0.68	10.9	0.026	0.0211	2	924	0
2	10.6	1.19	6.6	0.57	0.92	0.45	16.7	0.6	7.17	0.068	0.0311	2	425	0.1
3	11.5	1	20	0.6	1.1	0.39	10	0.6	18.2	0.0195	0.0104	2	880	0.1
3	12.3	1.03	13.3	0.73	1.5	0.63	9.7	0.75	8.9	0.047	0.024	1	557	0.1
2	14.3	1.24	10.7	0.78	1.9	1.39	13.4	0.72	5.63	0.129	0.032	2	455	0.2
3	13.4	1.3	15	0.68	1.36	1.34	15.7	0.64	11.03	0.091	0.017	2	554	0.1
3	13.8	1.3	7	0.8	2.15	2.5	15.6	0.6	3.25	0.36	0.041	2	641	0

33	Sakunthala	45	2	2265/07	cryptogenic	2	2	2	5	1	10	2	2	2	2	2	1	10.9	100000	16	1.4	1	1	35	3.9
34	Divya	18	2	4413/08	cryptogenic	1	2	2	5	1	9	2	2	2	2	2	1	10.8	100000	14	1.2	0.8	1.2	20	4
35	Thanikaivel	40	1	6955/11	Alcohol	1	1	2	10	3	21	2	2	2	2	2	2	8.6	255000	28	2	135	2.9	88	1.8
36	Pandian	52	1	5113/11	Alcohol	2	1	2	7	2	11	1	2	2	2	2	2	10.2	95000	21	1.5	0.7	0.9	45	3.1
37	Palayam	45	1	1227/11	cryptogenic	1	2	2	7	2	7	2	2	2	2	2	1	6.3	42000	15	1	0.63	1.2	29	3.2
38	Sathiakumar	42	1	362/08	cryptogenic	1	2	2	5	1	10	2	2	2	2	2	1	6	61000	16	1.4	1.1	0.8	46	3.9
39	Sharmila banu	35	2	3165/07	cryptogenic	1	2	2	5	1	9	2	2	2	2	2	1	11.8	61000	14	1.3	0.9	0.8	26	4.4
40	Yasin	22	2	4823/11	wilsons	1	2	2	8	2	12	2	2	2	2	2	2	7.6	90000	16	1.2	0.6	2.6	102	2.7
41	Mumtaj begum	33	2	444/09	cryptogenic	1	2	2	6	1	7	2	2	2	2	2	1	12.2	100000	16	1.1	0.7	1	40	3.2
42	Palani	37	1	622/12	Alcohol	1	1	2	10	3	12	1	2	2	2	2	1	7.7	80000	17	1.7	0.8	2.9	58	2.3
43	Thameem	22	1	3651/11	cryptogenic	1	2	2	6	1	10	2	2	2	2	2	1	12.6	84000	16	1.1	1	2	24	3.9
44	Malleswari	40	2	1103/06	cryptogenic	1	2	2	5	1	19	2	2	2	2	1	1	9.8	78000	37	2.8	0.52	1.2	79	2.8
45	Elumalai	23	1	3848/09	Alcohol	1	1	2	7	2	13	1	2	2	2	2	2	12.3	120000	18	1.6	0.9	1.6	89	3.1
46	Nedunchelian	56	1	7538/10	Hepatitis B	1	1	2	5	1	9	2	2	2	2	2	1	7.2	144000	16	1.2	0.9	1.2	76	3.7
47	Rajendran	65	1	4088/11	Alcohol	1	1	1	7	2	12	1	2	2	2	2	2	11	100000	14	1.2	1.2	1.7	96	2.8
48	Vasantha	42	2	630/06	Hepatits C	2	2	2	6	1	11	2	2	2	2	2	2	5	130000	16	1.4	0.8	1.3	24	3.2
49	Anthonysamy	66	1	n	Alcohol	1	1	2	6	1	13	1	2	2	2	2	1	10.2	130000	14	1.6	1.2	0.6	52	4.2
50	Kamali	35	2	1532/06	cryptogenic	1	2	2	6	1	8	1	2	2	2	2	2	9	110000	14	1.2	0.8	0.8	21	3.6
51	Sampoornam	58	2	5706/09	cryptogenic	2	2	2	10	3	17	1	2	2	2	1	2	8.4	24000	25	1.8	0.7	3	79	2.1
52	Kahimunizha	26	2	4125/11	cryptogenic	1	2	2	8	2	22	2	2	2	2	1	1	6.8	50000	30	2.9	0.7	0.9	27	3.4
53	Prabu	30	1	272/10	Alcohol	1	1	1	9	2	6	1	2	2	2	2	1	16	142000	14	1	0.9	2.5	444	2.1
54	Mangalam	16	2	5687/11	Wilsons	1	2	2	11	3	19	1	2	2	2	1	2	8.1	180000	31	2.3	0.6	2.3	123	2.4
55	Baskar	46	1	3155/09	cryptogenic	1	2	2	6	1	10	1	2	2	2	2	2	9.2	57000	16	1	0.78	1.3	62	4.2
56	Koteeswari	52	2	442/07	Autoimmune	2	2	2	5	1	10	2	2	2	2	2	1	7.4	120000	16	1.3	1	1.3	23	3.6
57	Govardhanam	47	1	6156/10	Alcohol	1	1	2	8	2	10	1	2	2	2	2	1	13.3	97000	15	1	0.87	2.5	77	3.5
58	Zakir	43	1	154/10	Hepatitis B	1	1	1	8	2	11	1	2	2	2	1	2	6.9	100000	20	1.4	1.09	0.9	87	2.4
59	Jaffer	52	1	91/12	Alcohol	1	1	1	10	3	12	1	1	2	2	2	2	10.2	110000	15	1.5	1	1.2	55	1
60	Kumar	54	1	183/12	Hepatitis B	1	2	2	11	3	15	1	1	2	2	1	2	7.9	51000	19	1.4	1.08	2.8	54	2.3
61	Arumugam	38	1	222/12	Alcohol	2	1	1	8	2	23	2	2	2	2	1	1	4.8	64000	27	2.1	0.92	8.3	83	3.8
62	Albert xavier	52	1	5908/11	Alcohol	1	1	2	8	2	30	2	2	2	2	1	1	6.2	69000	24	2.1	2.7	4.6	72	3.7
63	Mohandass	64	1	6470/10	cryptogenic	1	2	2	11	3	18	1	1	2	1	1	1	5.5	59000	20	1.8	0.9	3.8	54	4.1
64	Chandra	40	2	6282/09	cryptogenic	2	2	2	7	2	6	1	2	2	2	2	2	8.9	92000	17	1	0.7	1	46	3.2
65	Gunasekaran	49	1	6642/09	Alcohol	1	1	1	10	3	22	1	2	1	2	1	1	12.4	112000	18	1.3	1.39	11.9	82	2
66	Ethiraj	51	1	5550/09	Hepatitis B	2	2	2	9	2	19	1	2	2	2	1	2	10.2	79000	20	2.1	1.1	2.2	45	3.2

3	12.9	1.02	5.6	0.72	1.37	0.56	20.5	0.54	4.09	0.1	0.041	2	488	0
2	14.2	1.1	16.9	0.74	1.23	0.92	13.7	0.71	13.7	0.054	0.019	2	1861	0
3	12.5	1.13	12.3	0.69	1.24	1.15	13.24	0.76	9.91	0.093	0.0256	2	719	0.1
3	11.75	1.2	9.9	0.7	1.5	1.42	10.8	0.64	6.6	0.143	0.0271	2	202	0.1
3	12.8	1.28	6.1	0.67	1.14	0.91	19.9	0.57	5.4	0.149	0.039	1	306	0.1
3	11.4	1.2	14.5	0.73	1.6	0.63	17.6	0.7	6.13	0.064	0.0313	1	347	0
1	13.11	1.63	19.7	0.57	0.99	2.12	10.5	0.74	19.9	0.108	0.0128	2	857	0.1
3	13	1.6	6.9	0.61	1.06	1.36	21.3	0.66	6.51	0.197	0.035	1	469	0
3	11.4	1.5	15.8	0.9	1.7	0.9	16.4	0.7	9.3	0.057	0.023	1	488	0.1
2	11.4	0.98	9.9	1.41	0.71	0.32	16.13	0.47	13.9	0.032	0.04	1	522	0
3	11.29	0.85	7.9	0.64	0.94	0.49	17	0.78	8.04	0.062	0.037	1	459	0.1
2	11.09	1.9	6.3	0.89	1.9	2.21	16	0.71	3.32	0.35	0.058	2	750	0.1
3	12	1.21	7.5	0.76	1.3	0.64	15.7	0.62	5.77	0.085	0.037	2	917	0.1
1	12.4	0.8	12.6	0.74	1.71	0.58	8.71	0.73	7.4	0.046	0.026	2	1149	0.1
1	10.3	0.9	16.3	0.79	1.9	0.3	12.7	0.67	8.58	0.018	0.019	2	1023	0
3	12.3	1.07	8.7	0.6	1.13	0.9	10.1	0.77	7.69	0.103	0.032	2	1287	0
1	11.7	1.64	23	0.7	1.45	1.58	14.3	0.66	15.9	0.069	0.012	1	769	0
2	10.8	1.34	8.9	0.79	1.8	1.45	20.8	0.56	4.9	0.162	0.029	1	115	0.3
3	10.8	1.43	11.8	0.65	1	2.11	19	0.69	11.8	0.179	0.023	1	263	0.1
3	11.2	1.3	10.6	0.78	1.1	1.5	13.4	0.72	9.64	0.142	0.031	1	1060	0.3
1	10.1	0.7	11	0.68	1.2	0.7	14.5	0.64	9.2	0.063	0.261	2	1241	0.1
3	11.4	1.24	12.6	0.87	2.1	2.14	15	0.66	6	0.169	0.027	1	380	0.1
3	8.6	1.7	12.8	0.7	2.2	1.62	17.8	0.7	5.82	0.127	0.023	1	674	0
3	12.1	0.92	13.9	0.8	2.28	0.41	13.6	0.83	6.1	0.029	0.0286	1	713	0.1
2	11.1	1.2	12.6	0.65	1.1	0.86	12.8	0.76	11.45	0.068	0.024	2	781	0.1
2	13	1.4	15	0.84	2.87	1.2	14.9	0.64	5.23	0.8	0.022	2	738	0.1
2	12.3	1.1	9.9	0.76	1.5	0.47	14.2	0.7	6.6	0.047	0.032	2	359	0.1
2	12.8	1	5.9	0.8	1.4	0.46	15.6	0.54	4.21	0.078	0.079	2	410	0.1
2	11.1	1	12.3	0.84	1.5	0.53	15.8	0.77	8.2	0.043	0.0315	1	437	0.1
3	12.8	1.1	17.5	0.7	1.34	0.89	13.1	0.72	13.06	0.051	0.017	1	450	0.1
2	12.3	0.6	18.3	0.8	1.4	0.67	16.1	0.66	13.07	0.037	0.017	2	571	0.1
3	11.4	1.18	14.9	0.78	1.6	1.6	15.2	0.7	9.3	0.107	0.022	2	737	0.7
3	12.2	1.3	10.1	0.77	1.2	1	13.4	0.56	8.4	0.099	0.026	1	940	0.1

67	Jagadeesan	55	1	224/10	cryptogenic	1	2	2	8	2	15	2	2	2	2	1	2	9.9	130000	18	2	0.71	1.2	52	2.9
68	Jayalakshmi	50	2	5458/09	cryptogenic	1	2	2	7	2	10	1	2	2	2	2	1	8.9	64000	17	1.3	0.4	1.2	65	3
69	Rajendran	55	1	2005/10	Alcohol	2	1	1	7	2	8	1	2	2	2	2	1	11.2	148000	14	1	0.5	1.4	59	3.2
70	raja	39	1	4712/09	Alcohol	1	2	2	9	2	15	2	2	2	2	2	2	13.4	124000	16	1.1	0.8	8.1	142	2.5
71	srinivasan	35	1	6280/07	cryptogenic	1	2	2	6	1	7	2	2	2	2	2	2	14	150000	18	1.1	0.8	1	95	3.1
72	Jothi	55	1	240/10	Hepatitis B	1	2	2	6	1	8	2	2	2	2	2	2	9.1	84000	15	1.1	0.4	1.1	67	2.9
73	rosy	46	2	4916/07	cryptogenic	1	2	2	6	1	7	2	2	2	2	2	1	12.4	100000	16	1.1	0.7	0.9	80	3.2
74	Neelakandan	49	1	5664/10	Alcohol	1	1	1	7	2	9	2	2	2	2	2	1	13.6	99000	14	1	1.2	1.1	52	2.4
75	Murthy	59	1	4585/08	Hepatitis C	2	2	2	5	1	9	2	2	2	2	2	2	13	110000	13	1.1	1.1	1.3	78	3.8
76	Murugan	50	1	3006/09	Hepatitis B+Alcohol	1	1	1	6	1	10	2	2	2	2	2	2	14	140000	14	1.2	0.57	1.2	45	3.1
77	Krishnaveni	58	2	5988/10	Hepatitis B	1	2	2	7	2	8	1	2	2	2	2	2	9.2	105000	15	1.1	1	1.3	52	2.8
78	Dhanasekar	40	1	6037/10	Hepatitis B	1	2	2	7	2	8	1	2	2	2	2	2	11.4	135000	14	1	1	1.2	66	3.1
79	Raja	39	1	4712/10	Alcohol	1	1	1	9	2	19	1	2	2	2	1	2	14	134000	14	1.5	0.8	8.1	174	3.4
80	sivaprasad	46	1	4215/09	Alcohol	1	1	1	6	1	6	2	2	2	2	2	2	13.9	150000	17	1	0.6	0.9	143	3.1
81	srinivasan	35	1	6280/07	cryptogenic	1	1	1	5	1	7	2	2	2	2	2	2	13.8	153000	16	1.1	0.8	1	67	3.7
82	selvi	40	2	5335/09	Hepatitis C	1	2	2	8	2	10	2	2	2	2	1	2	12.4	100000	14	1.7	0.5	1.71	79	2
83	sudhakar	45	1	4941/09	Hepatitis B	1	2	2	8	2	10	2	2	2	2	1	2	8.9	77000	18	1.1	0.8	2	92	2.9
84	Adhinarayanan	50	1	1896/10	Hepatitis C+Alcohol	1	1	1	10	3	19	1	2	2	2	2	2	8.6	47000	18	1.3	0.71	12.4	105	2
85	Bagavathy	48	2	1208/10	Hepatitis B	2	2	2	9	2	16	2	2	2	2	2	2	5.3	64000	18	1.6	0.5	3.2	96	2.2
86	Dharanikkkarasi	42	2	932/10	Hepatitis B	1	2	2	6	1	7	2	2	2	2	2	2	10.2	65000	16	1.1	0.6	0.6	60	3
87	Indira	43	2	228/10	cryptogenic	1	2	2	8	2	14	2	2	2	2	1	2	10.1	45000	24	1.7	0.83	1.6	52	2.4
88	Ethiraj	48	1	7055/10	Hepatitis B	1	2	2	8	2	16	1	2	2	2	2	2	9.6	77000	16	1.6	0.9	3.3	84	3.6
89	Gajendran	52	1	1764/11	Alcohol	1	1	1	10	3	14	1	2	2	2	1	2	8.9	133000	19	1.3	1.23	2.4	43	2
90	Ganesan	55	1	1899/10	Hepatitis B+Alcohol	1	1	1	9	2	12	1	2	2	2	2	2	9.8	97000	19	1.3	0.98	2.2	89	2
91	Gajapathy raja	32	1	3040/10	Hepatitis B+Alcohol	1	1	1	10	3	19	1	2	2	2	1	2	9	89000	22	1.7	0.9	6	39	2.9
92	Kannan	40	1	1287/11	Hepatitis B+Alcohol	1	1	1	10	3	19	1	2	1	2	1	2	5.8	31000	25	1.9	1.31	2.3	57	2
93	Muthu	39	1	825/11	Alcohol	2	1	1	10	3	16	1	2	2	1	1	2	8.5	83000	25	1.8	0.76	2.2	148	2
94	Manokaran	56	1	264/11	Hepatitis B+Alcohol	1	1	1	11	3	18	1	2	2	2	2	2	10	217000	16	1.5	0.5	5.7	314	2
95	Murali	37	1	1468/06	Hepatitis B	1	2	2	8	2	14	2	2	2	2	2	2	8.2	49000	16	1.1	1.2	3.4	55	2.6
96	Noordeen	45	1	5146/10	Alcohol	2	1	1	12	3	18	1	1	2	1	2	2	7.7	149000	17	1.3	0.85	8.6	81	2.3
97	Pushpa	55	2	6429/09	Hepatitis C	1	2	2	9	2	11	1	2	2	2	2	2	10.2	71000	18	1.2	0.5	2.1	48	2
98	Ramu	48	1	4719/10	Alcohol	2	1	1	10	3	19	1	2	2	2	2	2	10.6	78000	23	1.6	0.5	6.7	54	2.4
99	Siva	41	1	1260/11	Hepatitis B+Alcohol	1	1	1	11	3	26	1	2	2	2	2	2	12.2	40000	28	2	1.7	6.7	170	2

1	13.2	0.9	18	0.62	0.8	0.52	21	0.7	22.5	0.029	0.014	2	619	0
3	13	1.6	10	0.68	1.22	2.1	18	0.64	8.2	0.21	0.026	2	356	0.1
2	13.6	1.4	17.8	0.74	1.26	1.6	14.8	0.69	13.8	0.089	0.0166	1	1072	0
2	13	1.1	8	0.7	1.21	0.9	13	0.7	6.61	0.113	0.037	1	954	0.1
2	12	1.2	17.5	0.68	1.15	0.93	17.5	0.64	15.2	0.053	0.015	1	986	0.1
3	12.3	1.35	14	0.66	1.2	0.7	13	0.72	11.7	0.05	0.02	2	646	0.1
2	10.2	1.4	9.1	0.78	1.45	2.1	12.8	0.64	6.3	0.231	0.033	2	781	0.1
3	11.8	1.4	16	0.8	1.02	1.62	22	0.71	15.7	0.101	0.021	2	450	0.1
1	13	1.3	16.2	0.7	1.23	1.5	9.2	0.7	13.2	0.092	0.018	2	1195	0.1
2	13.7	1.21	14.5	0.66	0.78	1.42	13	0.68	18.5	0.097	0.019	2	1076	0
1	12	0.6	15	0.68	0.97	0.87	8.5	0.6	15.5	0.058	0.0163	2	1235	0.1
1	12.6	1.3	14.2	0.7	0.84	0.9	14.2	0.7	16.9	0.063	0.021	2	950	0.1
1	13	1.1	8	0.6	0.58	0.8	13	0.62	13.7	0.1	0.028	2	1030	0.1
1	15.2	1.1	15.4	0.65	1.1	0.85	11.2	0.66	14	0.055	0.0167	2	857	0.1
2	12	1.18	16.1	0.7	0.9	0.96	17.5	0.75	17.8	0.059	0.0195	2	1195	0
1	11.6	1.4	9.4	0.7	1.2	1.12	12.8	0.69	7.8	0.119	0.031	1	781	0.1
3	10.2	1.4	9.1	0.9	1.52	0.94	12.8	0.76	5.9	0.103	1.045	2	602	0.1
3	15.6	1.1	13.4	0.66	0.9	0.89	12.2	0.6	14.8	0.066	0.018	1	385	0.2
2	12.2	0.9	17.4	0.68	1.3	1.42	10.7	0.62	13.4	0.083	0.015	1	598	0.2
3	12.4	1.4	8.3	0.76	1.6	1.3	12.7	0.76	5.18	0.157	0.041	2	512	0.1
1	13.5	1.2	28	0.69	0.93	1.1	18	0.7	19.4	0.61	0.161	2	250	0.1
2	10	1.3	11.3	0.72	0.87	1.4	13.9	0.69	12.99	0.124	0.022	2	554	0.1
2	10.5	1.1	14.2	0.64	0.77	0.9	14.2	0.65	18.4	0.0803	0.0175	2	937	0
2	8	1.2	13.6	0.68	1.2	0.64	12	0.6	11.3	0.047	0.018	2	808	0.1
2	9.3	1.6	15	0.65	1.42	1.2	14.3	0.62	10.56	0.08	0.0161	2	622	0
2	10.9	1.2	12.6	0.7	1.3	1	13	0.67	9.7	0.08	0.022	1	238	0.2
2	13.4	1	17.5	0.6	1.21	0.98	11.6	0.63	14.5	0.056	0.0129	1	716	0.2
1	14.3	0.9	14.6	0.64	1.13	1.2	11.2	0.61	12.9	0.082	0.0161	2	1938	0.1
2	10.6	1.3	11.2	0.7	1.1	1.12	13.9	0.71	10.2	0.1	0.027	1	353	0.1
2	14.1	1.2	31.6	0.6	1.16	1.3	14.6	0.69	17.7	0.063	0.0121	2	1021	0.1
2	10.2	1.2	13.6	0.7	1.2	0.9	13.2	0.66	11.3	0.066	0.0203	2	538	0.1
3	14.4	1.1	12	0.69	0.98	1.2	12.9	0.6	12.3	0.1	0.021	2	605	0.1
3	11.1	1.6	8.7	0.7	1.48	1.27	13.4	0.56	5.87	0.145	0.027	1	299	0.4

PROFORMA

1. Name:
2. Age :
3. Sex :
4. MGE No :
5. Diagnosis : Cirrhotic
6. D.O.R:
7. Resident of: Chennai / other city (specify)
8. Type of house: pucca/hut/semi
9. Per capita income:
10. Literacy status: studied up to: no education/I-V/VI-VIII/IX-XII/college/professional/other courses
11. Occupation (as such):
12. No. of children:
13. No. of adult family members:
14. Religion: Hindu /Muslim /Christian/others (specify):
15. Smoker: - present/past/never

    Duration of smoking in yrs: ≤1/1-≤5/5-≤10/ > 10-≤20/>20 Yrs

    Brand:- Beedi/Cigarette/combined
16. Alcohol:- present/past/never

Clinical details:

1. History of bleed: yes / no
2. Duration of illness (as such):
3. Age at diagnosis (as such):
4. History suggestive of liver disease:

5. Jaundice : Yes / No; if yes for how long……

6. Oedema legs : Yes / No

7. Weight loss : Yes/No

8. Weakness : Yes / No

9. Ascites : Yes / No

10. Hepatomegaly : Yes / No

11. *Splenomegaly* : Yes / No

12. **Investigations:**

Date	
TC	
Hb	
Platelets	
PCV	
PT	
APTT	
INR	
Urea	
Sugar F/PP	
Creatinine	
Chloride	
Bilirubin T	
Bilirubin B	
Albumin	
Globulin	
AST	
ALT	
GGT	
S. Alk Phos	
S. AFP	
HBsAg	
Anti HCV	

13.Ascitic fluid analysis:

14. UGI scopy:

Date			
Grades of varices			
Fundal varices			
PHG			
Endotheraphy			

15. Liver biopsy: as required

16. USG abdomen:

Liver	Shrunken/large/normal size	
	Echotexture: coarse / normal	
	Edges: regular/irregular	
	Nodularity: yes / No	
PV (mm)	Diameter:	
	Direction of blood flow	Hepatopetal
		Hepatofugal
Pv velocity (cm/s)		
Hepatic artery Resistance index		
Liver vascular Index		
Congestion Index		
Portal hypertension index		
Spleen size (cm)		
Splenic artery Resistance index		
Ascities	Present/absent	

17. Doppler indices:

Date	PV dm (cm)	PV Velocity (cm/se)	HARI	HA pulsatility index (HAPI)	PV cs area	Spleen size cm	SARI	Liver vascular index	Congestive index	PHT index

HARI = Systolic velocity – end diastolic velocity/systolic velocity

Liver Vascular Index = Portal venous velocity/ HA pulsatility index

Congestive index = PV cross sectional area/ PV velocity

PHT index =(HARI*0.69)(SARI*0.87)/ PV mean velocity

18. Platelet / Spleen diameter ratio :
19. AST / platelet count ratio index:
20. Treatment:-
21. Follow up:
22. Outcome: