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Research Article

# TO STUDY PORTAL HAEMODYNAMICS AS A PREDICTOR OF OESOPHAGEAL VARICES IN CIRRHOTIC PATIENTS IN TAMILNADU

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**ABSTRACT**

To evaluate the relationship of Doppler parameters of hepatic and portal vasculature, hepatic vein wave forms with presence and size of oesophageal varices in cirrhotic patients. A cohort of cirrhotic patients identified by clinical, laboratory and radiological parameters were evaluated. They were investigated for oesophageal varices by oesophagoduodenoscopy and by Doppler ultrasound. The relation between the presence and size of oesophageal varices and Doppler parameters were studied. Fifty two patients were enrolled in this prospective study. There were 44 male and 8 female patients. 23 patients were in Child Pugh Class C , 16 in Child Pugh Class B and 13 in Child Pugh Class A. Small oesophageal varices were associated with monophasic wave forms in 19.4%. biphasic wave forms in 38.7%, triphasic wave forms in 41.9%. Large oesophageal varices were associated with monophasic wave forms in 42.1%. biphasic wave forms in 52.6%, triphasic wave forms in 6.3%. The p- value was statistically significant with 0.013 [p <0.05]. Spleen size greater than 15.3 was associated with large oesophageal varices which was statistically significant, none of the Doppler parameters were statistically significant for large oesophageal varices. Our study data suggested that monophasic hepatic vein wave forms, splenic artery resistivity index, and spleen size greater than 15. 3 cms were related with presence of large oesophageal varices. These parameters may help in targeted identification of patients with large oesophageal varices and aid in their management.

**Keywords :-** Cirrhosis, Doppler ultrasound, Hepatic vein wave forms, Oesophageal varices.

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## INTRODUCTION

Portal hypertension is the most common and dreaded complication of chronic liver disease. It is responsible for the development of gastroesophageal varices, variceal hemorrhage, ascites, renal dysfunction, portosystemic encephalopathy, hypersplenism, and hepatopulmonary syndrome. Bleeding from ruptured oesophagogastric varices is a major complication of portal hypertension and a frequent cause of death. Only 40% of Child pugh class A patients have varices, while they are present in 85% of Child pugh class C patients[1].

In the cirrhotic patients without varices at first endoscopy, the annual incidence of new varices is at mean of 7%, ranging from 5 to 10%[2]. A hepatic venous pressure gradient over 10 mm Hg is the strong predictor for the development of oesophageal varices. Once developed, varices progress in size from small to large before they eventually rupture and bleed. Studies assessing the progression from small to large varices have showed rates of progression of varices ranging from 5 to 30% per year.[3]

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Changes in hepatic vein pressure gradient (either spontaneous or caused by drug therapy or transjugular intrahepatic porto systemic shunts) are usually accompanied by parallel variations in the size of the oesophageal varices, which is significantly reduced when HVPG decreases below 12 mm Hg.[4]

Oesophageal variceal bleeding related to portal hypertension is the second most common cause of severe upper gastrointestinal bleeding (after peptic ulcer disease). The acute mortality rate with each bleed is approximately 30%, and the long-term survival rate is less than 40% after one year with medical management alone. Despite advances in medical therapy, endoscopic hemostasis and portosystemic shunt procedures overall long-term survival rates have not improved for patients with variceal bleeding. Liver transplantation, however can improve the survival in selected patients. Survival in nontransplanted patients with variceal bleeding is heavily influenced by the severity of underlying liver disease, with poorer survival rates for patients with Child-Pugh class C cirrhosis than for those with Child class A or B cirrhosis.

A combination of beta blockers and variceal band ligation is probably the best treatment option, especially in patients who have bleeding. Patients who rebleed despite combined endoscopic and pharmacologic treatment may be treated by transjugular intraheptic or surgical portosystemic shunting. TIPS is the only option in nonsurgical candidates. All Child–Pugh class C patients should be considered for liver transplantation.

The role of non invasive markers in prediction of oesophageal varices in patients with cirrhosis was evaluated in various studies[5]. However, the usefulness of these markers in clinical use is still unclear. Doppler ultrasonography allows us to examine haemodynamics of abdominal vessels including the hepatic and portal system. Thus, many investigators have attempted to confirm the usefulness of Doppler ultrasound in assessing portal hypertension in cirrhotic patients. In particular, it would be highly desirable to have any Doppler parameter be a suitable substitute for the invasive current gold standard of measuring hepatic venous pressure gradient for assessing portal hypertension.

Predicting the grade of varices by noninvasive methods at the time of diagnosis is likely to predict the need for prophylactic beta blockers and band ligation as treatment for the varices. Therefore the present study has been undertaken to determine the appropriateness of Doppler parameters of portal vasculature and hepatic vein wave forms in predicting the existence and grading of the oesophageal varices.

**Aim and Objectives of The Study**

To evaluate the relationship of Doppler parameters of hepatic and portal vasculature, hepatic vein wave forms with presence and size of oesophageal varices in cirrhotic patients.

## MATERIALS AND METHODS

The study included consecutive patients with liver cirrhosis admitted in our institution Department of General Surgery, Melmaruvathur Adhiparasakthi Institute of Medical sciences and Research, Melmaruvathur.

Patients were included in this study after their willingness to undergo necessary investigations. Informed written consent was taken before the enrollment in this study. Ethical committee approval was obtained for the study purpose.

**Inclusion Criteria:**

Patients aged between 18 and 80 years with clinical, laboratory and radiological features of cirrhosis and portal hypertension.

**Exclusion criteria**:

1. Patients on diuretics, beta blockers.
2. Previous surgical interventions for portal hypertension.
3.Previous Endoscopic sclerotherapy/ Endoscopic variceal band ligation therapy / TIPS.
4. Presence of portal vein thrombosis.
5. Presence of hepatocellular carcinoma.
6. Active gastrointestinal bleed on admission.
7. Advanced comorbidity for endoscopy.

**Clinical evaluation:**

In the study group, diagnosis of cirrhosis was done on the basis of clinical, laboratory and radiological parameters. The grading of ascites was done as mild, moderate and severe and the grading of hepatic encephalopathy was done by applying West Haven criteria.

**Laboratory Investigations**:

Haematological investigations like haemoglobin, WBC count, platelet count, prothrombin time , bilirubin (total , direct, indirect ), total protein albumin and globulin , alanine aminotransferase , aspartate aminotransferase, HBsAg and Anti HCV were performed for all patients. Tests for autoimmune liver disease, haemochromatosis and Wilson disease were done only if clinical situation warranted the study. Ascitic fluid analysis was done for estimation of serum ascites albumin gradient. Child pugh score was calculated using the clinical and laboratory parameters.

**Doppler Ultrasound**

The patients in the study group were kept under overnight fasting . The Doppler ultrasound was done with the patient in the supine position during quiet respiration.

The following Doppler factors were recorded by the same equipment (with a 3 - 5 MHz curvilinear linear - array transducer) and by the same operator for all patients.

(1) Portal vein flow velocity as time average maximal velocity in cm/s [6]

(2)Portal vein diameter

(3) Portal vein cross sectional area

(4)Hepatic artery resistance index (systolic velocity - end diastolic velocity)/systolic velocity];

(5) Splenic artery RI measured (systolic velocity - end diastolic velocity)/systolic velocity

(6) Hepatic artery pulsatility index - (systolic velocity - end diastolic velocity)/ mean systolic velocity[7]

(7) spleen size as length in its longest axis

The following indices were calculated:

(1) Liver vascular index as the ratio of portal venous velocity to hepatic arterial pulsatility index[8]

(2) Congestion index (CI) of the portal vein was calculated by dividing portal vein cross-sectional area by mean portal vein velocity[9]. Mean velocity was calculated as the time-averaged maximal velocity multiplied by 0.57.

The hepatic vein wave forms were measured in the right hepatic vein (RHV) since it drains into inferior venacava in about 85% cases[10]. Doppler waveforms were divided into three types namely triphasic, biphasic and monophasic.

**Ascites**

Presence of ascites was determined clinically as well as by ultrasound.

**Endoscopic features**

All the patients were subjected to oesophagoduodenoscopy after an overnight fasting. Oesophageal varices were graded as small if they are less than 5 mm and large if they are greater than 5 mm[1]. Red signs if present were noted over the oesophageal varices.

Gastric varices if present, were typed according to their position and graded as small if less than 10 mm, medium if size is between 10 to 20 mm and large if greater than 20 mm. Portal hypertensive gastropathy was graded as mild and severe.

**Statistical Analysis**

Data were analyzed with SPSS version 15. Descriptive statistics including means, standard deviations, and frequencies were analysed. The chi square test was used to compare differences.Values were considered significant if $P < 0.05$ (95% CI). Presence and grade of oesophageal varices was predicted using the logistic regression equation

**RESULTS**

A total number of fifty two (52) patients were included in the study. Of those, 44(84.6%) were male and 8 (15.4%) were female. The preponderance of male in this study group was attributed to the etiology of the cirrhosis the most common being ethanol induced.

The symptom duration in the patients varies between 15 to 90 days. Ascites was clinically present in 41 of patients and jaundice was present in 30 of patients. About 37 patients had hepatic encephalopathy at presentation.

The majority of the patients were Child Pugh class C 23 (44.2%). Patients with Child Pugh A were 13 (25%) and Child Pugh B constituted 16 (30.8%) the rest of the study group.

Oesophageal varices were present in 50 patients of which 31 had small varices (59.6%) and 19 (36.5%) had large varices . Gastric varices was present only in 3 (5.8%)patients.

34 patients had portal hypertensive gastropathy, among which 4(7.7%) had severe and the rest of 30(57.7%) had mild grade.The majority of patients in this study were belong to alcoholic cirrhosis which constitutes of about 63.4%, Hepatitis B - 19.2%, Hepatitis C - 1.9% .

Small oesophageal varices were associated with monophasic wave forms in 19.4%. biphasic wave forms in 38.7%, triphasic wave forms in 41.9%. Large oesophageal varices were associated with monophasic wave forms in 42.1%. biphasic wave forms in 52.6%, triphasic wave forms in 6.3%. The p- value was statistically significant with 0.013 [p <0.05]Table -1.

**Correlation of hepatic vein wave forms with gastric varices:**

Gastric varices absent were associated with monophasic wave forms in 26.1%. biphasic wave forms in 42.9%triphasic wave forms in 30.6%.Gastric varices present were associated with all the three forms are equal (33.3%).p-value was not significant for gastric varices and portal hypertensive gastropathy.

**Correlation Of Hepatic Vein Wave Forms With Portal Hypertensive Gastropathy :**

Mild Portal Hypertensive Gastropathy were associated with monophasic wave forms in 33.3%. biphasic wave forms in 40%, triphasic wave forms in 26.7%. Severe Portal Hypertensive Gastropathy were associated with monophasic wave forms in 50%. biphasic wave forms in 25%, triphasic wave forms in 25%.

**Correlation Of Hepatic Vein Wave Forms With Child Pugh Score :**

CTP-A were associated with monophasic wave forms in 21.4%. biphasic wave forms in 13.6%, triphasic wave forms in 43.8%. CTP-B were associated with monophasic wave forms in 35.7%. biphasic wave forms

in 27.3%, triphasic wave forms in 31.3%. CTP-C were associated with monophasic wave forms in 42.9%. biphasic wave forms in 59.1%, triphasic wave forms in 25%.

Spleen size greater than 15.3 was associated with large oesophageal varices which was statistically significant. P<0.05.

## DISCUSSION

Variceal bleeding due to portal hypertension develops in 30 – 40% of patients with cirrhotics. With the growing number of chronic liver disease in the world , the likelihood of patients presenting with gastrointestinal bleeding will increase associated with the concurrent increase in the screening procedures for varices. Non invasive screening for identifying patients with high risk varices will definitely of help by means of reducing the cost and improve patient's tolerability.

Studies conducted on noninvasive predictor of varices[11] lack uniformity in their structure . Moreover the accuracy of prediction of oesophageal varices by these non invasive markers is found to be unsatisfactory and hence lack clinical applicability.

It was estimated that 100 screening endoscopy need to be preformed to prevent 1-2 cases of variceal bleeding .Therefore, identification of clinical features that can accurately predict esophageal varices and help identifying patients at greatest risk is important to improve the yield and cost- effectiveness of endoscopic screening.

In the present study, there was preponderance of male as compared to female. This was expected as the most common cause of cirrhosis was ethanol [63.4%] and ethanol consumption was common in males. Most of the patients were in the Child Pugh class B & C. This was also to be expected as our unit is a tertiary care centre catering treatment to advanced liver diseases.

In this study, the relationship of the hepatic vein wave forms with oesophageal varices was studied. Small oesophageal varices were associated with monophasic wave forms in 19.4%. biphasic wave forms in 38.7%,triphasic wave forms in 41.9%.

Large oesophageal varices were associated with monophasic wave forms in 42.1%. biphasic wave forms in 52.6%,triphasic wave forms in 6.3%. This had a statistical significance p<0.05 and the monophasic waves were associated with large varices. This is in concurrence to previous studies.

Baik et al[12] prospectively examined the relationship between waveforms and the severity of portal hypertension measured by HVPG in 78 cirrhotic patients who experienced variceal bleeding. A correlation was found between abnormalities in HV waveforms and HVPG, i.e. with increasing HVPG, the HV waveform tended to flatten. Furthermore, the monophasic waveform was associated with severe portal hypertension (HVPG 415 mmHg) with relatively high sensitivity and specificity in that study population. Hence, flattening of the HV waveform observed in the cirrhotic patients indicates a high likelihood of severe portal hypertension.

In a study from South India conducted by Thomas Joseph et al.[13] it was shown that a loss of triphasic wave pattern was associated with large oesophageal varices. The sensitivity of loss of the triphasic wave pattern in detecting significant large varices was very high (95.23%) and negative predictive value was also high (75%). Severity of liver disease as indicated by Child-Pugh and MELD scores did not correlate with changes in hepatic venous waveforms.

Gorka et al[14] in a study found sensitivity for the detection of largevarices was 92% for monophasic waves, 76% for waves with loss of the reverse flow component, and 62% for biphasic waves. Overall specificity was 100%.

Kim and colleagues[15] prospectively evaluated the correlation between the extent of abnormal Doppler HV waveforms, expressed as Damping Index [DI], and the HVPG, and response to propranolol in patients with cirrhosis. DI is calculated by dividing the minimum velocity over the maximum velocity of the HV waveform.Abnormal HV waveforms were seen in 66 out of 76 patients (86.8%).

Flattening of the HV wave can be attributed to an increase in HV inflow from intrahepatic shunts implicated in portal hypertension[15]. This results in haemodynamic blunting of the effect of variations in central venous pressure during the cardiac cycle, rather than lack of liver compliance. There was no stastical significant correlation of the hepatic vein wave forms with gastric varices, portal hypertensive gastropathy and red signs on oesophageal varices.

Splenomegaly is the cardinal sign of hypertension in cirrhotic patients. Our data showed that spleen size measured by ultrasonography was an independent predictor for the presence of varices.

In our study, spleen size of greater than 15.3 cms was associated with presence of large varices, with a p value of 0.029. [ p<0.05]

Dib N, et al[16] identified non invasive diagnosis of large oesophageal varices because of prognostic and economic issues. He concluded that Indirect echographic markers of portal hypertension and oesophageal varices (ascites, portal vein diameter > or = 13 mm, spleen length, maximal and mean velocimetry of portal vein flow, respectively < 20 cm/s and < 12 cm/s) could be useful. Among this parameters, spleen length is an independent predictive marker of oesophageal varices.

**Table 1: Correlation of hepatic vein wave forms with o. Varices**

| ESOVARIX | | HEP.VEIN WAVEFORMS | | | Total |
|---|---|---|---|---|---|
| | | Mono | Bi | Tri | |
| NO VARIX | Count | 0 | 0 | 2 | 2 |
| | % within ESO VARIX | .0% | .0% | 100.0% | 100.0% |
| | % within HEP.VEIN WAVEFORMS | .0% | .0% | 12.5% | 3.8% |
| SMALL VARIX | Count | 6 | 12 | 13 | 31 |
| | % within ESO VARIX | 19.4% | 38.7% | 41.9% | 100.0% |
| | % within HEP.VEIN WAVEFORMS | 42.9% | 54.5% | 81.3% | 59.6% |
| LARGE VARIX | Count | 8 | 10 | 1 | 19 |
| | % within ESO VARIX | 42.1% | 52.6% | 5.3% | 100.0% |
| | % within HEP.VEIN WAVEFORMS | 57.1% | 45.5% | 6.3% | 36.5% |
| TOTAL | Count | 14 | 22 | 16 | 52 |
| | % within ESO VARIX | 26.9% | 42.3% | 30.8% | 100.0% |
| | % within HEP.VEIN WAVEFORMS | 100.0% | 100.0% | 100.0% | 100.0% |

## CONCLUSION

This study shows spleen size correlate clearly with the presence of large esophageal varices . However these parameters did not predict the presence of gastric or other porto systemic collaterals. Presence of hepatic vein monophasic wave forms predicts the incidence of large oesophageal varices. This would encourage the use of endoscopy screening in patients with large oesophageal varices and this would help to reduce the burden and cost for the patients and health care providers .Studies on large scale are needed before applying these parameters as predictors of oesophageal varices. If so it will enable us to start primary prophylaxis without subjecting patients to oesophagoduodenoscopy.

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