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ORIGINAL ARTICLES

Diagnostic predictability of using portal vein diameter in confirming portal hypertension

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Abstract: *Objective: To find an accurate cut-off value for Portal vein diameter as a diagnostic tool for Portal hypertension on comparing with Doppler ultrasound, CT abdomen, or endoscopy of collateral circulation.*

Materials and methods: This was a cross-sectional study, with a sample size of 100 patients. Inclusion criteria were patients above the age of 15 years who underwent routine USG abdomen for various conditions in the Radiology Department of a tertiary hospital. Distribution of age, gender, chronic liver disease, portal hypertension, and portal vein diameter of all the patients were measured and recorded. Portal hypertension was diagnosed using the presence of collaterals either on upper endoscopy (if done), Doppler USG, or CT multiphase abdomen.

Results: Portal vein diameter of 10.5mm was found to have a sensitivity of 61.22%, specificity of 60.78%, a positive predictive value of 60.00%, a negative predictive value of 62.00%, and accuracy of 61.00%. 45 out of 59 (76.3%) and 37 out of 49 (75.5%) patients of the 50-70 years age group had chronic liver disease and portal hypertension respectively. 45 out of 65 (69.2%) males 37 out of 65 (56.9%) males developed CLD and PHTN.

Conclusion: Using portal vein diameter as a diagnostic indicator of PHTN has an accuracy of %, so it can be used in routine USG abdomen of CLD patients to prevent the dreaded complications of PHTN and treat it early. Male gender and patients above the fifth decade were increasingly predisposed to both CLD and PHTN.

Keywords: *portal hypertension, chronic liver disease, portal vein diameter, collateral circulation*

INTRODUCTION

Portal hypertension is defined as portal venous pressure more than 12 mm of Hg [1]. It is measured as the hepatic venous pressure gradient (difference of wedge hepatic pressure and free hepatic venous pressure), which is normally 4-6 mm Hg. More than 10 mmHg is portal hypertension, and more than 12 mmHg causes clinically significant variceal bleeding. [2]

Portal hypertension is major pathophysiology in patients with cirrhosis. Portal pressure is the gold standard to evaluate the severity of portal hypertension, and radiological intervention is the only procedure for pressure measurement [3].

Causes of portal hypertension are pre-sinusoidal (portal vein thrombosis, extrinsic compression of portal vein, Schistosomiasis (S. mansoni or S. japonicum)), sinusoidal (cirrhosis) and post-sinusoidal (Budd-Chiari syndrome, hepatic venous-occlusive disease, congestive cardiac failure) [1]

Portal hypertension leads to dilatation of portal vein,

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splenomegaly, and formation of portal-systemic collaterals at different sites. The portal system and the systemic venous circulation are connected at several locations. Collaterals develop in areas where anatomic connections exist between the portal venous and systemic circulation. These are vascular channels that are functionally closed in normal conditions but become dilated in portal hypertension as a consequence of increased intravascular pressure and blood flow. These gastro-oesophageal varices are responsible for the main complications of portal hypertension and massive upper GI bleeding [4].

The complications of portal hypertension (gastrointestinal hemorrhage, hepatic encephalopathy, hepatorenal syndrome, and ascites) continue to be the cause of significant morbidity and mortality. Portal hypertension remains one of the most serious sequelae of chronic liver disease [2].

To prevent bleeding from occurring, an appropriate early diagnosis of oesophageal varices at risk of bleeding should be achieved. Upper digestive tract endoscopy is the gold-standard method for diagnosing the presence of gastroesophageal varices and identifying signs of risk of bleeding. Universal endoscopic screening of oesophageal varices was recommended in all patients newly diagnosed with cirrhosis until 2015. However, due to the increase in the proportion of patients with early cirrhosis achieved by non-invasive diagnostic methods, this strategy proved to lead to a large number of unnecessary endoscopies that would eventually decrease the patient's compliance and increase the health system costs. In the last 10 years, increasing evidence regarding non-invasive methods accumulated and proved useful for stratifying the risk of carrying varices and varices requiring treatment in patients with CLD [5].

60-80% of patients with cirrhosis of the liver are associated with esophageal varices and 25-30% of them have got a risk of bleeding. Every year incidence of esophageal varices increases by 5% and 5-10% of patients with small varices progress to large varices [6].

Endoscopy is the standard diagnostic approach in patients with portal hypertension. But it is an invasive technique. Noninvasive imaging techniques are preferred for early diagnosis and prevention of complications. Portal vein diameter, peak systolic velocity, presence of collaterals, splenomegaly, etc can be found using Doppler USG and CT multiphase abdomen. The overall sensitivity for detection of coronary-gastroesophageal collaterals using USG was 80% and 64%, respectively, in a study done by Subramanyam et al. [7]. Doppler USG is frequently obtained in patients with chronic liver disease, suspected portal hypertension, and known portal hypertension. Among the many findings of portal hypertension is the enlargement of the portal vein, decreased portal vein velocity, increased congestion index, development of portosystemic collaterals, and reversal of portal vein flow. Using all of these, it is possible to diagnose portal hypertension with a high degree of sensitivity and specificity non-invasively [8].

It is possible to rule out clinically significant portal hypertension in patients with cirrhosis (i.e., hepatic venous pressure gradient ≥ 10 mmHg) with a level of clinically acceptable accuracy by combining measurements of liver stiffness, spleen stiffness, and Doppler ultrasound evaluation. The combination of these methods allows for the identification of patients with the most serious degree of portal hypertension as seen by Bolognesi M et al. [9].

Despite the safety, affordability, and widespread use of ultrasound, there is a paucity of literature on ultrasonographic assessment of the portal vein (PV) diameter in adult patients in our local environment [10]. According to a study done by Nestaĭko OV [11], real-time ultrasound investigation could provide important information and should be used as a non-invasive and harmless investigation in hepatological studies. Splenic Stiffness can also be used for assessing and monitoring PHTN in hepatitis C induced cirrhosis according to the study by Colecchia et al. [12].

The cutoff point for portal vein diameter differs from study to study despite the existence of a normal mean portal vein diameter between 10–15 mm in different settings. This implies the existence of limited evidence on the exact portal vein diameter which can help diagnose portal hypertension early [13].

This study will help find an exact cutoff point of portal vein diameter which can help rule out false positives and find out false negatives and provide prompt detection and treatment and prevention of progression of a chronic liver disease patient to portal hypertension, using a routine non- invasive USG scan.

MATERIALS AND METHODS

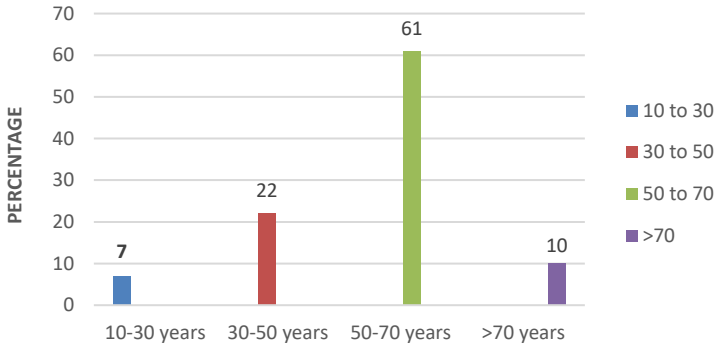
This is a cross-sectional study carried out in the Radiology department of AIMS, Kochi. Since this is a pilot study, a total of 100 patients above the age of 15, referred from various other departments to the Department of Radiology for routine USG of the abdomen are included. Those patients who underwent imaging studies elsewhere, patients having thromboembolic problems, etc are excluded. Portal vein diameter and Doppler study of spleen for splenic collaterals are carried out. MRD numbers of these patients are collected

to note down their age, history of CLD, Portal hypertension, and the presence of collaterals in the CT scan of the abdomen or upper endoscopy (if taken previously) is tabulated in MS Excel. The cut-off for Portal vein Diameter is found using Receiver Operator Characteristic Curve in SPSS. Qualitative variables are expressed as percentages and quantitative variables are expressed as mean with standard deviation. Informed consent was obtained from all the patients. The Ethics Committee of Amrita Institute of Medical Sciences has approved the study.

RESULTS

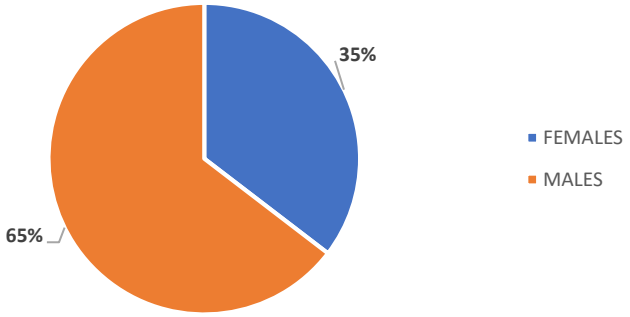
The mean age of the sample population was 54.9 +/- 13.89 years, median age 56 years (range of 17-84 years), and mode 67 years. The majority (61, 61%) of the patients were of the 50-70 years age group (Figure 1).

Figure 1: Distribution of age



35 (35%) of patients were females and 65 (65%) were males (Figure 2).

Figure 2: Distribution of gender



Of the 100 patients who underwent USG abdomen, 59 (59%) patients were found to have ultrasonographic features of chronic liver disease, while 41 (41%) patients did not have CLD.

49 (49%) patients were found to have collateral circulation, seen either on Doppler USG, or CT Multiphase abdomen, or Upper endoscopy (if done previously for any other indications), and 51 (51%) patients did not have any collaterals (Table 1).

Thus 49 (49%) patients had portal hypertension. Among the 50-70 years age group, 60.6% (37) of patients had PHTN, while 0 patients had PHTN in the 10-30 years age group, which clearly showed that PHTN was more common after the fifth decade of life (Figure 3).

Table 1: Distribution of portal hypertension (PHTN)

Collaterals	Frequency	Percent
Present	49	49.0
Absent	51	51.0
Total	100	100.0

More Males (56.9%) had PHTN as compared to females (34.2%), which was statistically significant, p-value <0.05 (Figure 4).

ROC curve to find out the portal vein diameter cut off had an Area 0.676, diameter 10.5 mm, with a sensitivity of 61.2%

and specificity of 60.8%, p-value of 0.002 (Figure 5).

Figure 3: Comparison of age and PHTN

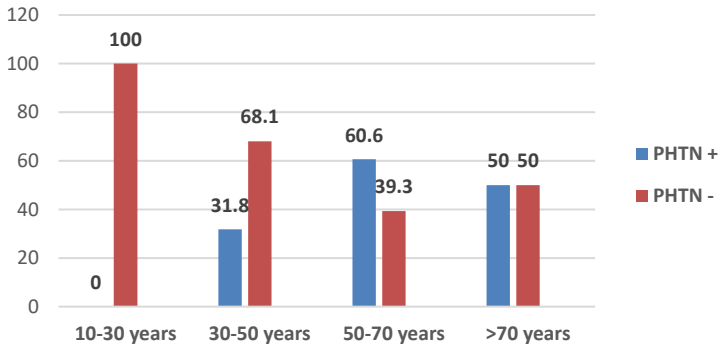


Figure 4: Comparison of gender and PHTN

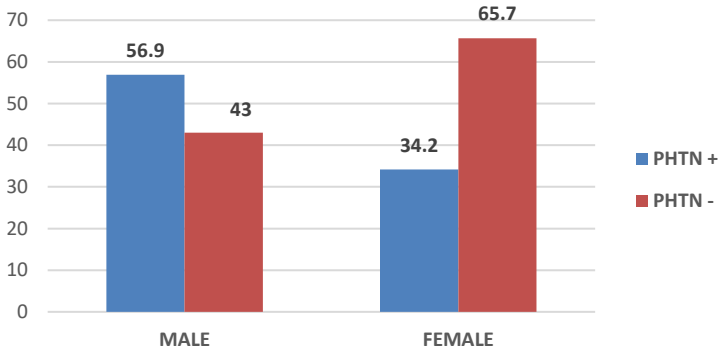
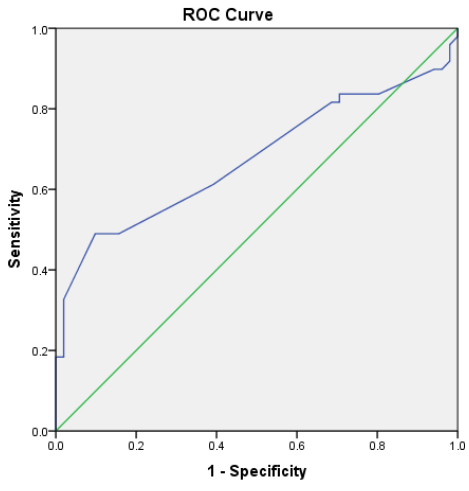


Figure 5: ROC curve of portal vein diameter and PHTN



AUROC	0.676
PVD	10.5 mm
p value	*0.002*
Sensitivity	61.2%
Specificity	60.8%

DISCUSSION

Portal hypertension is the most common complication and also one of the important causes of death in chronic liver diseases. Increased resistance to portal blood flow due to alteration of the hepatic architecture leads to dilatation of portal vein, splenomegaly, and formation of oesophageal and gastric varices, variceal hemorrhage, ascites, hypersplenism, encephalopathy, etc [4]. The overall sensitivity for detection of coronary-gastroesophageal collaterals using USG was 80% and 64%, respectively, in a

study done by Subramanyam et al. [7].

The mean age of the sample population was 54.9 +/- 13.89 years, the median age was 56 years (range of 17-84 years) and the mode of the age was 67 years. Most of the patients (61.0%) were in the 50-70 years age group, similar to the findings of Gaduputi et al. [14]. In a similar study done by Gunda et al. [15], it was found that the odds of having esophageal varices were independently higher among patients with increased age (Median age of 51 vs. 45 years; OR:1.02; 95%CI: 1.0–1.04; p = 0.030)

Out of the 100, 35 (35.0%) of patients were females and 65 (65.0%) were males. This pattern of male predominance is comparable to the study done by Sharma et al. [16] which had 86.1% of male patients. In the study by Akande et al. [17], 81% of males and a mean age of 44+12.6 years were seen among the cases.

More patients, i.e 73.7% and 60.6% of patients in the 50-70 years age group was found to have chronic liver disease and portal hypertension respectively; which clearly shows that both CLD and subsequently the development of PHTN is more common after the fifth decade of life. The same was the case with the male gender, i.e, 69.2% and 56.9% of males had CLD and PHTN respectively; which again shows that males have an increased association with CLD and subsequent development of PHTN. In a similar study done by Manik Mahajan et al. [18], it was found that the majority of the patients (32%) belonged to the 6th decade, with 66% of male and 34% of female patients having CLD and PHTN.

10.5 mm was found to be the cut off for Portal vein diameter in our study using ROC curve, with an area of 0.676, p-value 0.002, comparable to that found by Bhattarai S et al. [19], Sarwar et al. [20], and Guo et al. [21]. Collateral circulation was found in 49% of patients, which was seen either during Doppler ultrasound, CT multiphase abdomen, or endoscopy. Out of these 30, (60.0%) of the patients had a portal vein diameter of more than 10.5 mm. These were the true positive cases. Among these, 72% were in the 50-70 years age group. A similar study done by Gemecho et al. [13] revealed a normal mean portal vein diameter of 10.6 mm ±1.8 SD with a respirophasic variation of 25.6% and in the study by Ekta Gupta et al. [22] in North India, mean normal portal vein diameter measurement in male subjects was 10.33 mm ± 1.004 & in female 9.41 mm ± 1.16.

PVD > 10.5 mm was found in 20 (40.0%) of patients, but they did not have any collaterals; i.e these were the false-positive cases. CLD was found in 6 (30%) of these patients while 1 (0.5%) patient had portal vein thrombosis which explains the increase in diameter. Higher portal vein diameter was seen in those patients with esophageal varices in the study by Sangma MA [23] and Li FH et al. [24], as well as in cirrhotic children by Riahinezhad et al [25]. Diameter tended to increase in Child-Pugh scoring initially, before getting stenosed at higher scores, as found by Zhoy et al. [26].

The true negative cases were 31 (62.0%) of patients, who had a portal vein diameter less than 10.5 mm. The false-negative cases, i.e, 19 (38.0%) patients with PVD<10.5 mm but with the presence of collaterals, were long-standing chronic liver disease patients with well-established collateral circulation and a grossly shrunken liver, which led to the stenosis of the portal vein, leading to the decrease in PVD. Normal mean portal vein diameter in Ethiopian patients was found to be 10.0+1.8 mm (range 8.2 to 11.8) with a 21.5% reduction with the phases of respiration, in a similar study done by Scabel et al. [27]

In another study done by Lopamudra et al. [4], the average portal vein diameter of patients without gastro-oesophageal varices was 11.545 ± 1.514 mm and of patients with varices 13.998 ± 1.123 mm, which was slightly higher than the PVD found in our study, so was the normal mean portal vein diameter found in a study by Jefferey W et al. [28], which was 11 + 2 mm in patients aged between 21-40 years.

Thus, portal vein diameter of 10.5 mm had a sensitivity of 61.22%, specificity of 60.78%, the positive predictive value of 60.00%, the negative predictive value of 62.00%, and accuracy of 61.00%, in correctly diagnosing portal hypertension. In a similar study done by Johana Prihartini et al. [29], PVD of 11.5 mm had a sensitivity of 75% and a specificity of 54.5%. The sensitivity of using portal vein calibrations was found to be only 41.8% in the study done by Bolondi L et al. [30].

Limitations of our study: Portal vein diameter can vary in various physiological conditions, like inspiration, expiration, duration of liver pathology, etc. In our study, the diameter was taken at the hilum without taking into account the respiratory changes or the duration of CLD. Portal vein diameter can also vary according to age. There are wide variations in the age groups of the patients. More specific indicators like splenic size, peak systolic velocity, the direction of flow in the portal vein could have been included in the study.

CONCLUSIONS

Thus, portal vein diameter of 10.5 mm was the cut-off above which portal hypertension could develop, as was shown in the study. Though of limited sensitivity and specificity, it can be used to lookout for portal hypertension in chronic liver disease patients and prevent it as early as possible, before its dreaded complications set in. Both CLD and PHTN had a

high incidence after the fifth decade of life, so screening for either disease is recommended after 50 years of age.

Contribution of authors

Varsha Somakumar, Cibi Manoharan, V S Srikanth – Concepts, design, the definition of intellectual content, literature search, data acquisition, data analysis, Manuscript preparation, editing, and review

Rajesh Kannan, Chitra Tomy, Anagha Vasant, Vishal Marwaha, VR Mujeeb - Concepts, design, the definition of intellectual content, Manuscript editing and review.

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