Original Article

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Correlation between Portal Vein Diameter and Clinical Prognostic Scores in Patients with Liver Cirrhosis: A Cross-sectional Study

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ABSTRACT

Introduction: Patients with liver cirrhosis develop portal hypertension, which leads to complications like splenomegaly, ascites, and oesophageal varices. Hepatic Venous Pressure Gradient (HVPG) measurement, the best available method to measure portal hypertension, is invasive and difficult to perform. Some studies suggest that an increased Portal Vein Diameter (PVD) on ultrasonography indicates portal hypertension and correlates with oesophageal varices. Studies correlating PVD with other complications of portal hypertension, like ascites and spleen size, are scarce.

Aim: To correlate ultrasonographically measured PVD with clinical prognostic models: Child-Turcotte-Pugh (CTP) score and Model for End-stage Liver Disease (MELD), as well as with platelet count, in patients with liver cirrhosis.

Materials and Methods: This was a cross-sectional study conducted in the Department of General Medicine in collaboration with the Department of Radiodiagnosis at Government Medical Collge, Chandigarh, India, from February 2021 to September 2021 over an eight-month period in a tertiary healthcare centre in North India. A total of 97 patients with cirrhosis, identified based on clinical features supported by laboratory tests and ultrasonography, were included in the study. A 6 mL of blood sample was collected from each patient for haemogram, biochemical tests (liver function tests and renal function tests), and coagulogram. Ultrasonography assessment of PVD, spleen size, and ascites was performed in a supine position using a right subcostal approach, after overnight fasting. The collected data were analysed using relevant statistical tests.

Results: The mean age of the study population was 47.39±12.64 years, with 73 (75.3%) males and 24 (24.7%) females. The most common aetiological factor for liver cirrhosis was alcohol, present in 52 (53.6%) patients. The mean PVD was found to be 12.31±2.71 mm. The correlation coefficient of PVD with spleen size was 0.3 with a p-value of 0.004, suggesting a positive correlation. The correlation coefficient of parameters like thrombocytopenia, CTP score, and MELD score was -0.2 (p-value=0.066), 0.1 (p-value=0.463), and 0.0 (p-value=0.725), respectively.

Conclusion: Sonographically measured PVD cannot be used as a substitute for the clinical grading and staging of cirrhosis. Only a weak positive correlation was found between PVD and spleen size.

Keywords: Child-Turcotte-Pugh score, Model for end-stage liver disease, Portal hypertension, Thrombocytopenia, Ultrasonography

INTRODUCTION

Liver disease is a major cause of mortality and morbidity, with two million deaths per year worldwide, out of which one million deaths are due to complications of cirrhosis [1]. Cirrhosis is histopathologically characterised by the formation of regenerative nodules because of the development of fibrosis in response to chronic insult. This results in decreased hepatocellular mass and alteration of blood flow, which are responsible for various clinical features of cirrhosis and reflect the severity of the liver disease [2,3]. The loss of hepatocellular functions results in jaundice, coagulation disorders, and hypoalbuminaemia. Portal hypertension is responsible for the development of ascites, splenomegaly, thrombocytopenia, and bleeding from oesophageal varices [3,4].

The portal vein transmits blood from the capillaries of the intestinal wall and spleen via the superior mesenteric vein and splenic vein, respectively, to the hepatic sinusoids. With an increase in venous resistance and venous flow, there is dilatation of the portal vein in liver cirrhosis. Hence, portal vein dilatation may be an indicator of portal hypertension [5]. Some studies have correlated PVD with the presence and grading of oesophageal varices and found a significant correlation [6]. A PVD greater than 13 mm is considered to be the cut-off value for portal hypertension [7].

Ultrasonography is a non ionising, non invasive, easily available, and cost-effective modality. It has better compliance with the patients and can be used to assess liver size and echotexture, spleen size, peritoneal fluid, and PVD. Therefore, ultrasonography can be a potential tool to identify portal hypertension and its complications non invasively [5,8]. PVD may correlate with the complications of portal hypertension and may be an indicator of the disease's prognosis.

Thus, finding a correlation between PVD with clinical aspects (ascites, splenomegaly), laboratory parameters (thrombocytopenia), and prognostic scores (CTP score and MELD score) can help us identify portal hypertension complications and the disease's prognosis early and non invasively. This may also aid in guiding therapy early in the course of the disease.

MATERIALS AND METHODS

This cross-sectional study was conducted from February 2021 to September 2021 in the Department of General Medicine in collaboration with the Department of Radiodiagnosis at Government Medical College, Chandigarh, India. The study received approval from the Institutional Ethics Committee (IEC) (GMCH/IEC/2020/480R/80).

Inclusion criteria:

- Patients with liver cirrhosis based-on clinical, biochemical, and ultrasonographic findings.
- Age greater than 18 years.

Exclusion criteria:

- Patients previously or currently on treatment with beta-blockers.
- Patients with a history of sclerotherapy or banding for oesophageal varices.
- Patients with bleeding disorders unrelated to liver disease.
- Patients with any evidence of hepatocellular carcinoma.
- Patients with a recent history of upper gastrointestinal bleeding.
- Patients with other causes of portal hypertension, such as Budd-Chiari Syndrome, extrahepatic portal vein obstruction, non cirrhotic portal fibrosis.

Sample size calculation: The optimum sample size was calculated based on 90% specificity for prediction of oesophageal varices when the cut-off value {determined by Receiver Operating Characteristics (ROC) curve analysis} for PVD was 12.25 mm [6]. Assuming a 90% confidence level and 5% absolute precision, the optimum sample size was determined to be 97.

Study Procedure

Clinical history and physical examination findings were recorded with particular attention to recent gastrointestinal bleeding (within the last 6 weeks), bleeding disorders, alcoholism, blood transfusions, tuberculosis, intake of hepatotoxic drugs, exposure to sexually transmitted diseases, intravenous drug abuse, jaundice, anaemia, oedema, stigmata of chronic liver disease, dilated abdominal veins, ascites, splenomegaly, and encephalopathy.

Blood tests: A 6 mL blood sample was collected, with 2 mL for a haemogram (haemoglobin, platelet count, total leucocyte count, differential leukocyte count), 2 mL for biochemical tests including serum electrolytes (sodium, potassium, chloride), renal function tests (urea, creatinine), and liver function tests (serum bilirubin levels, total protein, albumin, alanine aminotransferase, and aspartate aminotransferase), and 2 mL for a coagulogram {International Normalised Ratio (INR), Prothrombin Time (PT), activated Partial Thromboplastin Time (aPTT), Prothrombin Time Index (PTI)}.

Ultrasound: All patients were kept fasting overnight prior to the procedure and were scanned in the supine position using a right subcostal approach. Sonographic measurements were conducted by the same examiner and repeated three times to gain PVD and standardised by examining the patient in the supine position with quiet respiration. PVD was measured at the porta hepatis. Other parameters, such as the echotexture of the liver, liver size, cranio-caudal spleen size, and the presence and grading of ascites, were also assessed. Ascites was graded as none, mild (detectable only on ultrasound), moderate (visible moderate abdominal distension), or severe (marked abdominal distension).

CTP and MELD score: Based on the admission data, the CTP score (range: 5-15) and Child class were calculated [9]. The MELD score (range: 6-40) was calculated according to the formula proposed by Kamath PS and Kim VR [10]:

9.57×Loge (creatinine mg/dL)+3.78×Loge (total bilirubin mg/dL)+ 11.2×Loge (INR)+6.43

Outcome measures: All data were recorded, and PVD was correlated with parameters like spleen size, ascites, platelet count, CTP score, and MELD score.

STATISTICAL ANALYSIS

Mean, along with standard deviation, was used for quantitative parameters like PVD and spleen size, while proportions and percentages were used for qualitative outcome parameters. The Chi-square test was utilised to test the significance of the association between outcome parameters and characteristics of patients with Chronic Liver Disease (CLD). The correlation coefficient of PVD with spleen size, platelet count, CTP score, and MELD score was calculated using Spearman's correlation coefficient. The strength of association (Point Biserial Correlation) was calculated for PVD and the presence of ascites. The parametric Student's t-test was used to determine the correlation between PVD and platelet count. Data analysis was conducted using Statistical Packages for Social Sciences (SPSS) software.

RESULTS

A total of 97 patients with liver cirrhosis participated in present study, comprising 73 males and 24 females, with a mean age of 47.39±12.64 years. The baseline characteristics of the study population are summarised in [Table/Fig-1]. A history of alcohol intake in cirrhogenic doses was present in 52 (53.6%) patients. Other causes of cirrhosis included Hepatitis C Virus (HCV) infection in 13 (13.4%) patients, Hepatitis B Virus (HBV) infection in 8 (8.2%) patients, Non Alcoholic Steatohepatitis (NASH) in 6 (6.2%) patients, and autoimmune hepatitis in 3 (3.1%) patients. In 15 (15.5%) patients, no cause of cirrhosis could be ascertained. The common presenting complaints were abdominal pain and abdominal distension, accounting for 46 (47.4%) patients, followed by altered sensorium in 19 patients (19.9%). Other clinical features included fever, jaundice, generalised body swelling, cough, shortness of breath, bleeding nose, decreased urine output, and vomiting [Table/Fig-2].

Parameters	Mean	Minimum	Maximum
Age (in years)	47.39±12.64	20	91
Laboratory parameters	**Mean**	**Minimum**	**Maximum**
Haemoglobin (gm/dL)	9.26±2.86	2.3	16.1
Platelet count ($1\times10^9/mm^3$)	120.82±99.02	11	623
Bilirubin (T) (mg/dL)	6.74±15.38	0.2	31.2
SGPT/ALT (U/L)	55.45±75.64	8	601
Albumin (g/dL)	2.73±0.64	1.7	4.34
INR	1.47±0.43	1.0	2.76
Ultrasonography parameters	**Mean**	**Minimum**	**Maximum**
Liver size	14.04±2.36	10	19.2
Spleen size	12.99±3.21	6	19
Portal Vein Diameter (PVD)	12.31±2.71	6	19
Ascites severity	**Number**	**Percentage**	**95% CI**
Absent	24	24.7%	16.8%-34.7%
Mild	20	20.6%	13.3%-30.3%
Moderate	24	24.7%	16.8%-34.7%
Severe	29	29.9%	21.2%-40.2%
CTP class	**Number**	**Percentage**	**95% CI**
A	8	8.2%	3.9%-16.1%
B	31	32.0%	23.1%-42.3%
C	58	59.8%	49.3%-69.5%
MELD score	**Mean**	**Minimum**	**Maximum**
MELD score	17.88±7.53	6	40

[Table/Fig-1]: Baseline characteristics of the study population.
SGPT: Serum glutamic pyruvic tranaminase; ALT: Alanine transaminase; INR: International normalised ratio

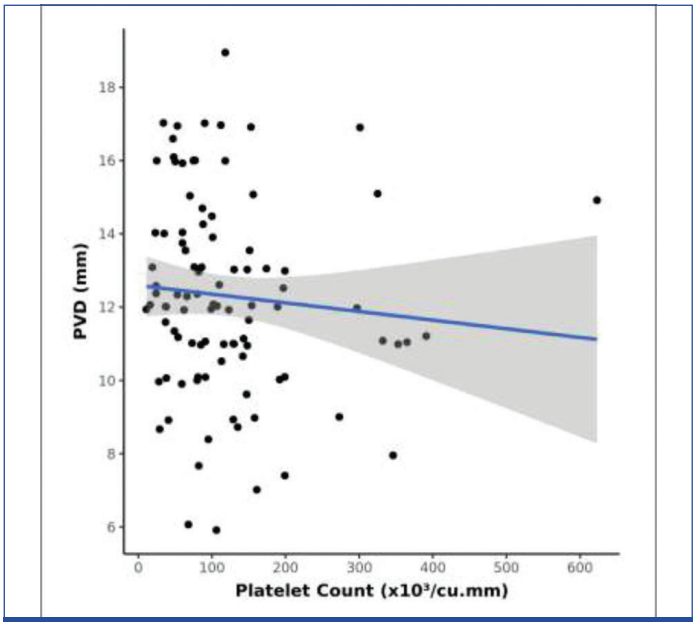
Main presenting complaint	Frequency	Percentage	95% CI
Abdominal distension	23	23.7%	15.9%-33.6%
Abdominal pain	23	23.7%	15.9%-33.6%
Altered sensorium	19	19.6%	12.5%-29.1%
Fever	10	10.3%	5.3%-18.6%
Jaundice	9	9.2%	3.2%-14.8%
Generalised body swelling	6	6.2%	2.5%-13.5%
Cough	2	2.1%	0.4%-8.0%
Shortness of breath	2	2.1%	0.4%-8.0%

Bleeding nose	1	1.0%	0.1%-6.4%
Decease urine output	1	1.0%	0.1%-6.4%
Vomiting	1	1.0%	0.1%-6.4%

[Table/Fig-2]: Distribution of the presenting complaint (N=97).

On ultrasound, the mean PVD was 12.31±2.71 mm, with a range from 6 mm to 19 mm. No correlation was found between PVD and the age of the patients, as the Spearman's correlation coefficient was 0.09 with a p-value of 0.382. The mean PVD in males and females was found to be 12.05±2.64 mm and 13.10±2.83 mm, respectively, with a p-value of 0.117, showing no correlation.

A non parametric test (Spearman's correlation) was used to explore the correlation between PVD and platelet count. There was a weak negative correlation between platelet count (×10³/cu.mm) and PVD (mm), and this correlation was not statistically significant (rho=-0.2, p=0.066) [Table/Fig-3].



[Table/Fig-3]: Scatterplot depicting the correlation between PVD (mm) and platelet count (×10³/mm³).

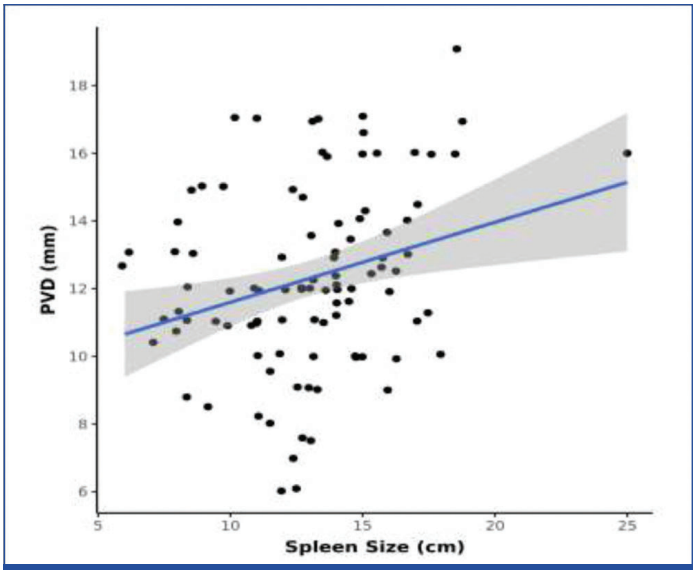
Thrombocytopenia (platelet count <150×10³ cu.mm) was present in 73 (75.3%) patients, while a normal platelet count (platelet count >150×10³ cu.mm) was seen in 24 (24.7%) patients. The mean PVD in patients with thrombocytopenia was 12.47±2.70 mm, and in patients with a normal platelet count, it was 11.82±2.73 mm. There was no significant difference between the groups in terms of PVD (mm) (t=1.012, p=0.318). The strength of association (Point-Biserial Correlation) was 0.1.

A non parametric test (Spearman's correlation) was used to correlate between the PVD and spleen size. There was a weak positive correlation between spleen size (cm) and PVD (mm), and this correlation was statistically significant (rho=0.29, p=0.004) [Table/Fig-4].

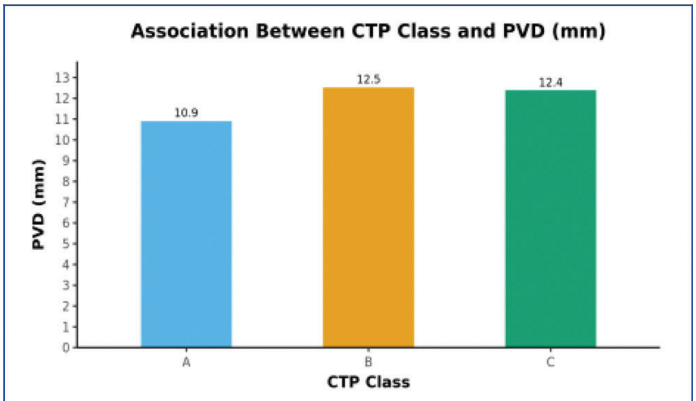
There was a weak positive correlation between CTP and PVD (mm), and this correlation was not statistically significant (rho=0.08, p=0.463).

The mean PVD in CTP class A, CTP class B, and CTP class C was 10.90±1.99 mm, 12.53±2.33 mm, and 12.38±2.96 mm, respectively. The range of PVD (mm) in CTP class A, CTP class B, and CTP class C was 7.6-14, 8-17, and 6-19, respectively. There was no significant difference between the groups in terms of PVD (mm) (χ^2=2.881, p=0.237). The strength of association (Kendall's Tau) was 0.06, indicating little or no association [Table/Fig-5].

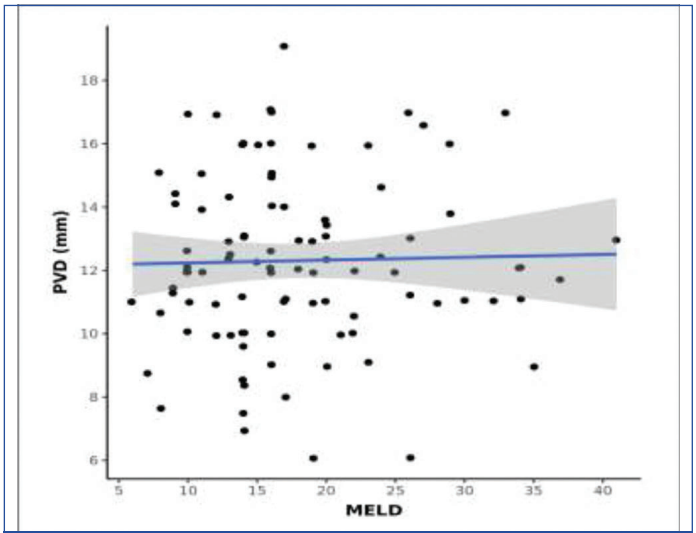
There was a weak positive or no correlation between MELD and PVD (mm), and this correlation was not statistically significant (rho=0.04, p=0.725) [Table/Fig-6].



[Table/Fig-4]: Scatterplot depicting the correlation between PVD (mm) and spleen size (cm).



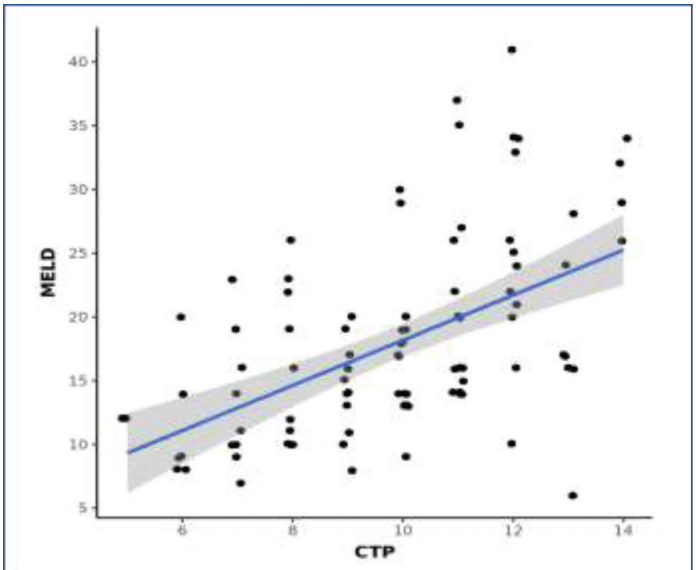
[Table/Fig-5]: Comparison of mean PVD with CTP class (p-value=0.23).



[Table/Fig-6]: Scatterplot depicting the correlation between PVD (mm) and MELD score.

The CTP score was also correlated with the MELD score, and a moderate positive correlation between CTP and MELD score was found. This correlation was statistically significant (rho=0.54, p≤0.001) [Table/Fig-7].

The mean PVD in patients with ascites was 12.43±2.72 mm, and in patients without ascites, it was 11.92±2.70 mm. The range of PVD in patients with ascites was 6 to 19 mm, and without ascites was 6 to 16.6 mm. There was no significant difference between the PVD (mm) and the presence of ascites (t=0.803, p=0.427). The strength of association (Point-Biserial Correlation) was 0.08, indicating little or no association.



[Table/Fig-7]: Scatterplot depicting the correlation between CTP score and MELD score.

The mean PVD in patients with mild ascites was 11.97±2.85 mm, with moderate ascites was 13.02±2.23 mm, and with severe ascites was 12.26±3.00 mm. The PVD range in the no ascites group was from 6-16.6, in the mild ascites group ranged from 7-17, moderate ascites group ranged from 10-17, and severe ascites group ranged from 6-19. There was no significant difference between the grade of ascites and PVD (mm) (χ^2=2.226, p=0.527). The strength of association (Kendall's Tau) was 0.05, indicating little or no association.

The correlation of different parameters using statistical tests and p-values has been depicted in [Table/Fig-8]. There was a weak positive correlation between spleen size and PVD and between CTP and MELD scores, both statistically significant. However, no significant correlation was found between PVD and platelet count, ascites, CTP score, and MELD score.

Correlation parameters	Spearman's correlation coefficient	p-value
PVD vs Platelet count	-0.2	0.666
PVD vs Spleen size	**0.3**	**0.004**
PVD vs Ascites	0.803	0.427
PVD vs CTP score	(Kruskal test) 0.1	0.463
PVD vs CTP class	2.881	0.237
PVD vs MELD score	0	0.725
CTP score vs MELD score	**0.5**	**<0.001**

[Table/Fig-8]: Correlation of different parameters using statistical test and p-value.

DISCUSSION

In the present study, the mean PVD in patients with liver cirrhosis was 12.31±2.71 mm. PVD positively correlated with spleen size. Bhattarai S et al., studied 150 patients (117 males and 33 females) with liver cirrhosis. The average spleen size of patients without varices was 12.67±2.35 cm and with varices was 15.367±1.210 cm. Patients with small varices and large varices had mean spleen sizes of 14.98±1.55 cm and 15.50±1.04 cm, respectively. This difference was statistically significant and suggested that spleen size correlated with oesophageal varices and portal hypertension [6]. A similar study by Shanker R et al., found a larger spleen size in the variceal group than the non variceal group (14.69±1.08 cm vs 12.45±0.65 cm, p<0.01) [11]. According to Chalasani N et al., spleen size is an independent factor in determining the risk of varices [12], and Thomopoulos KC et al., described spleen size >13.5 cm as being associated with varices [13].

Zaman S et al., conducted a study to determine the correlation between PVD and spleen size (craniocaudal). The study enrolled 1000 patients (369 females, 631 males) with a mean PVD of 10.27±1.78 mm and found an R-value of 0.98, suggesting a strong correlation between them. The present study is consistent with Zaman S et al.,'s study, although not showing the correlation to the same extent [14].

In present study, the mean PVD in patients with thrombocytopenia (platelet count <150×103/mm^3) was 12.47±2.70 mm, and in patients with a normal platelet count (platelet count >150×103/mm^3) was 11.82±2.73 mm. There was a weak negative correlation between platelet count and PVD (mm), but this correlation was not statistically significant (rho=-0.2, p=0.066). Gue CS et al., conducted a study to determine the correlation between thrombocytopenia and the presence of varices in cirrhotic patients. The results of above study showed that grade 2 and 3 varices were present in 6.3% of patients with a platelet count >150,000/mm^3, in 25% if the platelet count was 100,000-150,000/mm^3, in 38.9% of patients if the platelet count was 50,000-99,000/mm^3, and 100% if the platelet count was <50,000/mm^3. According to the above study, thrombocytopenia could be used to stratify the risk for the presence of oesophageal varices in patients with cirrhosis, and endoscopy would have a high yield of varices in patients with a platelet count <150,000/mm^3 [15]. A similar study conducted in Egypt enrolled 110 patients with cirrhosis, out of which 87 patients had oesophageal varices. They found that out of the total 77 patients with thrombocytopenia, 20 (25.97%) patients had grade II varices, and 21 (27.27%) patients had grade III or grade IV varices. Whereas in patients without thrombocytopenia (33 patients), 7 (21.21%) patients had grade II oesophageal varices, and only 3 (9.09%) patients had grade III or grade IV. This study concluded that a platelet count cutoff of 149,000/mm^3 has 82% specificity and 39% sensitivity for the occurrence of varices [16].

A study by Liu J et al., showed that the mean portal pressure gradient before and after Transjugular Intrahepatic Portosystemic Shunt (TIPS) placement was 28.3±4.6 mmHg and 11.3±4.5 mmHg (p<0.001). The mean spleen volume before and after 1-2 months of TIPS placement was 868±409 cm^3 and 710±336 cm^3 (p<0.001). In parallel to this, the number of patients with severe thrombocytopenia reduced from 25 (35.7%) to 11 patients (15.7%) in 6-12 months after TIPS placement. This study concluded that decreased portal pressure leads to a reduction in spleen volume and an increase in platelet count [17]. The above studies show a significant correlation between thrombocytopenia and portal hypertension. In contrast, the present study did not show any significant correlation of thrombocytopenia with PVD.

In the present study, the mean PVD in patients with ascites was 12.43±2.72 mm, and without ascites was 11.92±2.70 mm. There was no significant correlation between PVD and the presence or grade of ascites (t=0.803, p=0.427). The strength of association (Point-Biserial Correlation) was 0.08 (indicating little to no association). Other studies directly correlating PVD with ascites are limited, but various studies are available that correlate the presence of ascites with other portal hypertension markers.

Wadhawan M et al., studied the correlation of HVPG with ascites and found that the baseline Hepatic Venous Pressure Gradient (HVPG) in patients with ascites (18.5±5.6) was significantly higher than in those without ascites (16.6±7.6) (p-value=0.03). There was a significant correlation between higher HVPG and the presence of ascites (r=0.2, p-value=0.03) [18]. Torres E et al., found that high Serum Ascites Albumin Gradient (SAAG) (>1.1) ascites was associated with oesophageal varices [19]. Consistent with Torres E et al., study, Suresh I and Jagini SP concluded that the sensitivity of SAAG in predicting the presence of varices is 81%, and the specificity and positive predictive value are 100% [19,20]. The correlation of ascites with oesophageal varices and HVPG, which are indicators of portal hypertension, indirectly indicates a correlation of ascites with portal hypertension.

In present study, there was a weak positive correlation between CTP and PVD (mm), and this correlation was not statistically significant (rho=0.08, p=0.463). Similarly, no correlation was found between PVD and MELD score (rho=0.04, p=0.725).

A study by Ramanathan S et al., correlated CTP score and MELD score with HVPG and found that the mean HVPG was higher in patients with CTP class C (21.8±5.5 mmHg) than CTP class B (16.9±2.9 mmHg) and CTP class A (10.5±4.1 mmHg, p≤0.001). The Spearman's ratio for the MELD score was 0.504, suggesting a positive correlation with HVPG with a p-value of 0.002 [21]. Similarly, a study by Wadhawan M et al., showed that the mean HVPG was significantly higher in CTP class B (n=97, 17.4±6.9 mmHg) and class C (n=56, 19.0±5.7 mmHg) compared to class A cirrhosis (n=23, 12.2±5.9 mmHg, p <0.01). The mean HVPG was higher in CTP class C than class B [18].

Shateri K et al., conducted a cross-sectional study in Iran to find a correlation of PVD with CTP score and MELD score, the results of which showed little to no positive correlation of PVD with the CTP score and MELD score (r=0.241, p=0.05) and (r=0.216, p=0.05), respectively [22]. The present study is consistent with the study by Shateri K et al., as both do not show any significant correlation of PVD with either CTP score or MELD score.

Data available on this subject from India is very limited, so larger multicentric prospective studies are required to confirm the correlation of PVD with clinical, laboratory parameters, and prognostic scores. Furthermore, other non invasive parameters like liver and spleen stiffness may be evaluated to predict portal hypertension and its complications in chronic liver disease.

Limitation(s)

There are a few limitations to present study. The sample size was small, and larger sample studies are needed to generalise the results of present study to the general population. It was a single-centric study with most patients from three states in North India, namely Punjab, Haryana, and Himachal Pradesh. Whether present findings can be applied to the general population remains in doubt. The present study used ultrasonography, which is an observer-dependent technique. Patients with a history of upper gastrointestinal bleeding were excluded, but the possibility of including patients with occult upper gastrointestinal bleeding could not be ruled out.

CONCLUSION(S)

According to the results of present study, PVD does not correlate with the grading of ascites, the severity of thrombocytopenia, or prognostic scores like CTP score and MELD score. The present study suggests that sonographic PVD cannot be used as a substitute for the clinical grading and staging of cirrhosis. Only a weak positive correlation was found between PVD and spleen size.

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