

**ORIGINAL RESEARCH**

# Assessment of the prevalence of portal vein thrombosis symptoms and its clinical relevance

<sup>1</sup>Umesh Kumar Prajapati, <sup>2</sup>Anupam Raj Gaurab, <sup>3</sup>Sanjay Rawat, <sup>4</sup>Bharat Batham

<sup>1</sup>Senior Resident, Department of Endocrinology, All India Institute of Medical Sciences, Bhopal, Madhya Pradesh, India

<sup>2</sup>Senior Resident, Department of Endocrinology, University College of Medical Sciences, New Delhi, India

<sup>3</sup>Senior Resident, Department of Medicine, Government Medical College, Ratlam, Madhya Pradesh, India

<sup>4</sup>Senior Resident, Department of Medicine, Gajra Raja Medical College, Gwalior, Madhya Pradesh, India

**Corresponding author**

Bharat Batham

Senior Resident, Department of Medicine, Gajra Raja Medical College, Gwalior, Madhya Pradesh, India

Email: [bharat.batham1@gmail.com](mailto:bharat.batham1@gmail.com)

Received: 23 December, 2023

Accepted: 12 January, 2024

**ABSTRACT**

**Background:** Portal vein thrombosis in the general population is a rare event, but it occurs relatively frequently in patients with cirrhosis and its prevalence increases with severity of the disease. In this study we are studying the prevalence of PVT symptoms, its clinical relevance. **Materials & Methods:** 100 patients of chronic liver disease were subjected to abdominal ultrasonography, portal vein colour doppler studies and coagulation profile. All patients were subjected to fasting glucose, liver function test and the coagulation profile (Prothrombin time, INR). **Results:** In male participants age group 36-45 years have the highest number of participants; age group 15-25 years has the lowest number of participants while in female participants age group 46-55 years and 26-35 years have the highest and lowest participants respectively. Hepatitis B (20%) was the most common etiology of chronic liver diseases followed by others (18%) and Alcohol (15%). Hepatitis C contributes as an etiology in 4% of chronic liver disease patients. Ascites was the most common clinical feature in the study subjects (60%) followed by Splenomegaly (35%). Encephalopathy and Hematemesis both constitute 29% in participants. 89 participants have above-normal splenic diameter while 29 participants have above-normal portal vein diameter. Portal vein thrombosis was found in 9 participants while 6 of them have portal vein obstruction. According to Child Pugh class majority of participants were in class A (42%) and lowest numbers are in class B (28%). All 9 patients of portal vein thrombosis have splenomegaly and ascites while 7 patients also have encephalopathy and 6 have hematemesis along with portal vein thrombosis. Hematemesis encephalopathy, splenomegaly, ascites are significantly associated with PVT group when compared with non PVT p values of 0.001,0.002,0.001 and 0.002 respectively. **Conclusion:** Portal vein thrombosis is considered to be a frequent complication of liver cirrhosis. Portal vein thrombosis could worsen the rate of hepatic decompensation and survival of cirrhosis. The prognostic value of PVT in cirrhosis remains a gray zone.

**Keywords:** Chronic liver diseases, Hematemesis, Portal vein thrombosis

This is an open access journal, and articles are distributed under the terms of the Creative Commons Attribution- Non Commercial- Share Alike 4.0 License, which allows others to remix, tweak, and build upon the work non- commercially, as long as appropriate credit is given and the new creations are licensed under the identical terms.

**INTRODUCTION**

Portal vein thrombosis in the general population is a rare event, but it occurs relatively frequently in patients with cirrhosis and its prevalence increases with severity of the disease.<sup>1</sup> PVT can develop in the intrahepatic or extrahepatic segments of portal vein and extend to the superior mesenteric vein and or the splenic vein. Moreover, PVT can progress from a partial obstruction of a thrombus in the lumen to a complete blockade of portal venous blood flow. In cirrhotic patients the prevalence of PVT ranges from 0.6% to 26%.<sup>2</sup>

It may be argued that in patients with cirrhosis, prothrombotic disorders, if present might be

counterbalanced by the hypercoagulable state related to the impaired synthesis of procoagulant factors due to liver insufficiency.<sup>3</sup> However, the complex interaction among procoagulant and anticoagulant mechanisms found in cirrhosis rarely results in hypercoagulable states and bleeding but may even lead to hypercoagulable states facilitating portal vein thrombosis.<sup>4,5</sup> Furthermore, the findings of previous studies and other investigations indicate that in the presence or absence of cirrhosis, portal vein thrombosis should be considered a multifactorial disorder resulting from the combination of inherited and acquired risk factors, including a reduction in portal blood flow among the latter.<sup>6,7</sup> The causal

relationships and clinical presentation are often complex. As PVT may cause short-term as well as long-term complications, correct management by adopting adequate diagnostic and therapeutic measures is paramount.<sup>8</sup> In this study we are studying the prevalence of PVT symptoms, its clinical relevance.

## MATERIALS & METHODS

The present study consisted of 100 patients of chronic liver disease attending the department of General Medicine, Jayarogya Hospital, Gwalior shall be considered of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. Detailed history, abdominal ultrasonography, portal vein colour doppler studies and coagulation profile other than the biochemical profile was recorded. All patients were subjected to fasting glucose, liver function test and the coagulation profile (Prothrombin time, INR). The data was analyzed using statistical software package SPSS version 21. Mean, median, and standard deviation were calculated for continuous variables. The chi-square test and multivariate regression analysis were used for the test of association.

## RESULTS

**Table I Age and gender distribution**

Age group/Gender	Male	Female	P value
15-25 Years	6	8	0.339
26-35Years	9	3	
36-45 Year	17	10	
46-55 Year	13	15	
55-65 Year	11	8	
Total	56	44	100

Table I shows that in male participants age group 36-45 years have the highest number of participants; age group 15-25 years has the lowest number of participants while in female participants age group 46-55 years and 26-35 years have the highest and lowest participants respectively. However, these groups do not differ significantly in distribution (0.339).

**Table II Distributions of study participants according to etiology**

Variable		N	%
Alcohol	Yes	15	15%
	No	85	85%
HBsAg	Positive	20	20%
	Negative	80	80%
HCV	Positive	4	4%
	Negative	96	96%
Others	Yes	18	18%
	No	81	81%
Total		100	100%

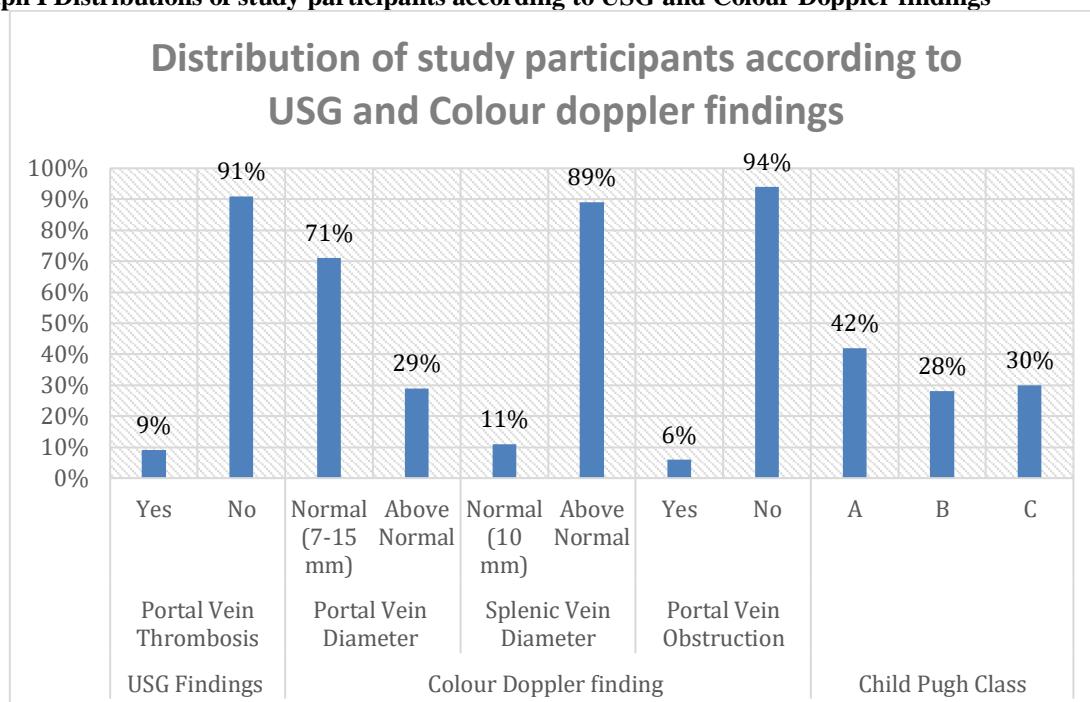
Table II shows that Hepatitis B (20%) was the most common etiology of chronic liver diseases followed by others (18%) and Alcohol (15%). Hepatitis C contributes as an etiology in 4% of chronic liver disease patients.

**Table III Distributions of study participants according to clinical features**

Variable		N	%
Hematemesis	Yes	15	15%
	No	85	85%
Encephalopathy	Yes	14	14%
	No	86	86%
Splenomegaly	No	65	65%
	+	18	18%
	++	13	13%
	+++	4	4%
Ascites	No	40	40%
	+	23	23%
	++	31	31%
	+++	6	6%
Total		100	100%

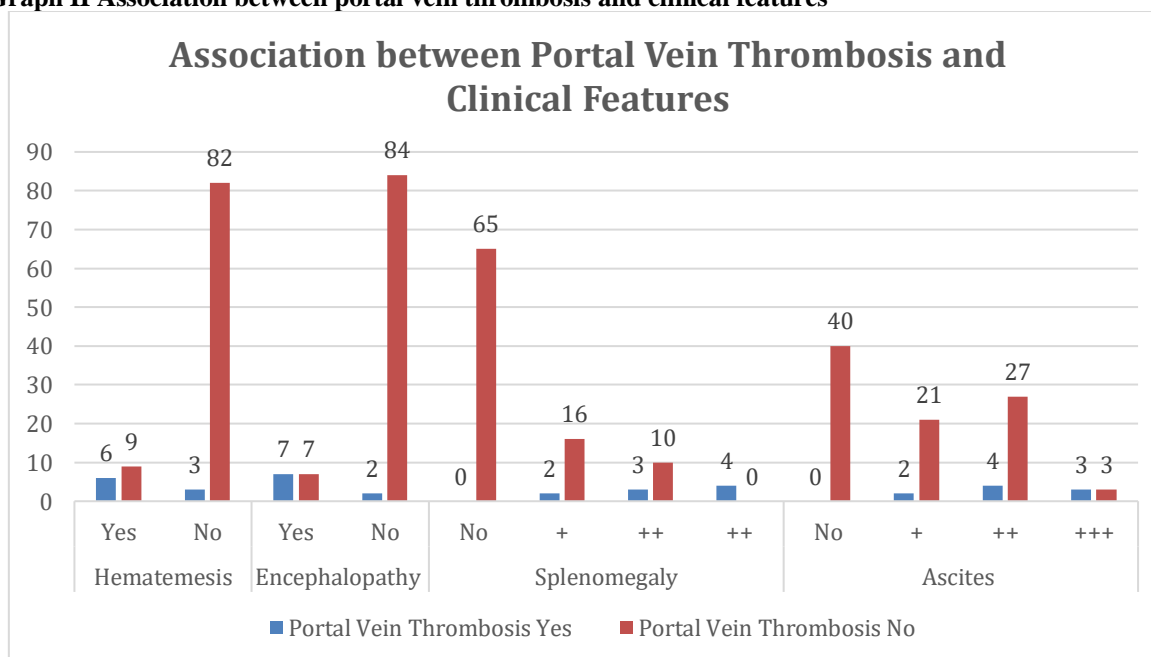
Table III shows that ascites was the most common clinical feature in the study subjects (60%) followed by Splenomegaly (35%). Encephalopathy and Hematemesis both constitute 29% in participants.

**Graph I Distributions of study participants according to USG and Colour Doppler findings**



Graph I shows that 89 participants have above normal splenic diameter while 29 participants have above-normal portal vein diameter. Portal vein thrombosis was found in 9 participants while 6 of them have portal vein obstruction. According to Child Pugh class majority of participants were in class A (42%) and lowest numbers are in class B (28%).

**Graph II Association between portal vein thrombosis and clinical features**



Graph II show that all the 9 patients of portal vein thrombosis have splenomegaly and ascites while 7 patients also have encephalopathy and 6 have hematemesis along with portal vein thrombosis. Hematemesis encephalopathy, splenomegaly, ascites are significantly found associated with PVT group when compared with non PVT p values of 0.001,0.002,0.001 and 0.002 respectively.

**DISCUSSION**

PVT formation in cirrhosis is multifactorial. Increased intrahepatic vascular resistance in combination with reduced portal flow velocity are considered important

risk factors for PVT in liver cirrhosis.<sup>9</sup> Cirrhotics have been traditionally considered prone to bleeding due to thrombocytopenia, defects of pro-coagulant factors and fibrinolysis. Recently, however, there is growing

evidence that hypercoagulability is an important part of the hematological spectrum in cirrhosis.<sup>10</sup> In this study we are studying the prevalence of PVT symptoms, its clinical relevance.

We found that in male participants age group 36-45 years have the highest number of participants; age group 15-25 years has the lowest number of participants while in female participants age group 46-55 years and 26-35 years have the highest and lowest participants respectively. However, these groups do not differ significantly in distribution (0.339). Bagheri et al<sup>11</sup> studied 219 patients (> 18 years old) with liver cirrhosis. There was no statistically significant difference in the assessed hypercoagulable states between patients with or without portal vein thrombosis. A history of previous variceal bleeding with subsequent endoscopic treatment in patients with portal vein thrombosis was significantly higher than in those without it

We found that Hepatitis B (20%) was the most common etiology of chronic liver diseases followed by others (18%) and Alcohol (15%). Hepatitis C contributes as an etiology in 4% of chronic liver disease patients. Ascites were the most common clinical feature in the study subjects (60%) followed by Splenomegaly (35%). Encephalopathy and Hematemesis both constitute 29% in participants. Nonami et al<sup>12</sup> in their study the incidence of portal vein thrombosis was examined in 885 patients who received orthotopic liver transplantations for various end-stage liver diseases between 1989 and 1990. The thrombosis was classified into four grades. Grade 1 was thrombosis of intrahepatic portal vein branches, grade 2 was thrombosis of the right or left portal branch or at the bifurcation, grade 3 was partial obstruction of the portal vein trunk, and grade 4 was complete obstruction of the portal vein trunk. Among the 849 patients without previous portosystemic shunt, 14 patients (1.6%) had grade 1, 27 patients (3.2%) had grade 2, 27 patients (3.2%) had grade 3 and 49 patients (5.8%) had grade 4 portal vein thrombosis. The incidence of portal vein thrombosis was highest (34.8%) in the patients with hepatic malignancy in the cirrhotic liver, followed by those with Budd-Chiari syndrome (22.2%) and postnecrotic cirrhosis of various causes (15.7%). The patients with encephalopathy, ascites, variceal bleeding, previous splenectomy and small liver had significantly higher incidences of portal vein thrombosis than the others. The total incidence of portal vein thrombosis among the 36 patients with previous portosystemic shunt was 38.9%, which was significantly higher than that (13.8%) of those without shunt.

We observed that 89 participants have above normal splenic diameter while 29 participants have above-normal portal vein diameter. Portal vein thrombosis was found in 9 participants while 6 of them have portal vein obstruction. According to Child Pugh class majority of participants were in class A (42%) and lowest numbers are in class B (28%). We found that

all the 9 patients of portal vein thrombosis have splenomegaly and ascites while 7 patients also have encephalopathy and 6 have hematemesis along with portal vein thrombosis. Hematemesis encephalopathy, splenomegaly, ascites are significantly found associated with PVT group when compared with non-PVT. Yerdel et al<sup>13</sup> observed that of 779 LTx, 63 had operatively confirmed PVT (8.1%): 24 had grade 1, 23 grade 2, 6 grade 3, and 10 grade 4 PVT. Being male, treatment for portal hypertension, Child-Pugh class C, and alcoholic liver disease were associated with PVT. The sensitivity of ultrasound (US) in detecting PVT increased with PVT grade and was 100% in grades 3-4. In patients with US-diagnosed PVT, an angiogram was performed and ruled out a false positive US diagnosis in 13%. In contrast with US, angiograms differentiated grade 1 from grade 2, and grade 3 from grade 4 PVT. Grade 1 and 2 PVT were managed by low dissection and/or a thrombectomy; in grade 3 the distal SMV was directly used as an inflow vessel, usually through an interposition donor iliac vein; in grade 4 a splanchnic tributary was used or a thrombectomy was attempted<sup>14</sup>.

## CONCLUSION

Authors found that portal vein thrombosis is considered to be a frequent complication of liver cirrhosis. Portal vein thrombosis could worsen the rate of hepatic decompensation and survival of cirrhosis. Prognostic value of PVT in cirrhosis remains a grayzone.

## REFERENCES

1. Charco R, Fuster J, Fondevila C, Ferrer J, Mans E, García- Valdecasas JC. Portal vein thrombosis in liver transplantation. *Transplant Proc* 2005;37:3904-3905.
2. Nery F, Chevret S, Condat B et al. Causes and consequences of portal vein thrombosis in 1234 patients with cirrhosis results of longitudinal study *hepatolbaltimMd* 2015;61: 660-7.
3. Ogren M, Bergqvist D, Bjorck M et al. Portal vein thrombosis prevalence characteristics and lifetime risk a population study based on 23796 consecutive autopsies. *World J Gastroenterol* 2006;12:2115 -19.
4. Chawla YK, Bodh V. Portal vein thrombosis. *J ClinExpHeapatol* 2015; 5:22-40.
5. Stine JG, Shah NL, Argo CK et al. Increased risk of portal vein thrombosis in patients with cirrhosis due to NASH.
6. Harding DJ, Perera MT, Chen F et al. Portal vein thrombosis in cirrhosis, controversies and latest development *world J Gastroenterol* 2015;21:6769-84.
7. Bayraktar Y, Harmanci O, Etiology and consequences of thrombosis in abdominal vessels *World J Gastroenterol* 20006;12:1165 -1174.
8. Wang JT, Zhao HY, Liu YL. Portal vein thrombosis, *Hepatobiliary Pancreat Dis Int.*2205;4:515 -518.
9. Wani ZA, Bhat RA, Bhadoria AS, Maiwall R. Extrahepatic portal vein thrombosis in special situations. Need for a new classification *Saudi J Gastroenterol.* 2015; (3):129-38.

10. Amitrano L, Guardascione MA, Brancaccio V, Margalione. Risk factors and clinical presentation of portal vein thrombosis in patients with liver cirrhosis. *J Hepatol* 2004;40:736-741
11. Bagheri Lankarani K, Homayon K, Motevalli D, Heidari ST, Alavian SM, Malek-Hosseini SA. Risk factors for portal vein thrombosis in patients with cirrhosis awaiting liver transplantation in Shiraz, Iran. *Hepat Mon* 2015;15:26407.
12. Nonami T, Yokoyama I, Iwatsuki S, Starzl TE. The incidence of portal vein thrombosis at liver transplantation. *Hepatology* 1992;16:1195-1198.
13. Yerdel MA, Gunson B, Mirza D, et al. Portal vein thrombosis in adults undergoing liver transplantation: risk factors, screening, management, and outcome. *Transplantation* 2000;69:1873-1881.
14. Varshney A, Rawat R. A cross-sectional study of echocardiographic characteristics of patients diagnosed with SARS-CoV-2 delta strain. *Glob Cardiol Sci Pract.* 2023 Aug 1;2023(3):e202319. doi: 10.21542/gcsp.2023.19.