ORIGINAL RESEARCH

Assessment of correlation of portal vein thrombosis with the color doppler in patients of chronic liver disease

[1]Umesh Kumar Prajapati, [2]Anupam Raj Gaurab, [3]Sanjay Rawat, [4]Bharat Batham

[1]Senior Resident, Department of Endocrinology, All India Institute of Medical Sciences, Bhopal, Madhya Pradesh, India
[2]Senior Resident, Department of Endocrinology, University College of Medical Sciences, New Delhi, India
[3]Senior Resident, Department of Medicine, Government Medical College, Ratlam, Madhya Pradesh, India
[4]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

Received: 21 December, 2023 Accepted: 13 January, 2024

ABSTRACT

Background: The prevalence of portal vein thrombosis can vary depending on age, underlying hepatic disease, velocity of portal venous blood and pro-versus anticoagulant status of the patient. The present study was conducted to correlate portal vein thrombosis with the color doppler, in patients of chronic liver disease. **Materials & Methods:** 100 patients diagnosed as a chronic liver disease of alcoholic and non-alcoholic aetiologies of both genders were enrolled. A detailed history, clinical examination, and laboratory profile of the patients were recorded. All patients were subjected to fasting glucose, liver function test and the coagulation profile (Prothrombin time, INR). **Results:** Of the 100 study subjects in the present study majority were from age group 36 to 55 years of age (55%). The mean age of participants was 43.69±12.40. The majority of participants (90) have Hb value below 11 gm%. Serum bilirubin level was above normal in 47 participants. The SGPT/SGOT values were above normal in each 37 participants. Blood coagulation markers (INR in 39 and PT in 38) were above normal in participants. The 33 and 30 participants have below-normal serum albumin and platelet count values, respectively. The subjects with portal vein thrombosis have portal vein diameter 2.06±0.44 mm which was significantly higher ($p<0.05$) than those not having portal vein thrombosis. In biochemical markers the Child Pugh score was significantly associated with platelets count, INR and total bilirubin ($p<0.05$). **Conclusion:** Portal vein thrombosis could worsen the rate of hepatic decompensation and survival of cirrhosis. The prognostic value of PVT in cirrhosis remains a grayzone. Early diagnosis, treatment and monitoring of the patients can prevent the occurrence of PVT in liver cirrhosis and also improve the liver function and survival.

Keywords: chronic liver disease, coagulation profile, portal vein thrombosis

INTRODUCTION

The prevalence of portal vein thrombosis can vary depending on age, underlying hepatic disease, velocity of portal venous blood and pro-versus anticoagulant status of the patient.[1] In patients with cirrhosis of Childpugh score A and B incidence of newly diagnosed PVT after 1 and 5 years has been reported to be 4.6% and 10.7% respectively. The relative risk of developing PVT in the presence of cirrhosis is increased more than seven-fold above the risk observed in the general population which is estimated to be <1 %.[2] While patients with compensated cirrhosis are rarely affected, PVT is frequently detected in advanced stages upto 25% in liver transplantation candidates and 35% in cirrhotic patients with HCC. However, in large studies including patients evaluated for liver transplantation,6.3% were diagnosed with PVT, in particular when cirrhosis was related to NASH. Moreover, in patients with advanced cirrhosis and those undergoing liver transplantation a prevalence of between 5% and 16% has been reported.[3]

The signs and symptoms of PVT are very heterogenous and range from incidental diagnosis during diagnostic procedures for unrelated reasons to severe complications due to intestinal infarction or to the development of portal hypertension such as variceal bleeding that can occur esophageal and /or gastric fundic varices when splenic vein thrombosis is present.[4] Therefore, the prognosis and treatment of

PVT depend on the localization, the degree of extension and the rapidity of development as well as risk factors for thrombosis and the stage of chronic advanced liver disease.[5] The present study was conducted to correlate portal vein thrombosis with the color doppler, in patients of chronic liver disease.

MATERIALS & METHODS

The present study consisted of 100 patients diagnosed as a chronic liver disease of alcoholic and non-alcoholic aetiologies of both genders. All gave their written consent to participate in the study.

Data such as name, age, gender etc. was recorded. A detailed history, clinical examination, and laboratory profile of the patients was recorded. All patients were subjected to fasting glucose, liver function test and the coagulation profile (Prothrombin time, INR). Data thus obtained were subjected to statistical analysis. P value < 0.05 was considered significant.

RESULTS

Table I Age-wise distribution of study participants

Age group	N	%
15-25 Years	14	14%
26-35 Years	12	12%
36-45 Year	27	27%
46-55 Year	28	28%
55-65 Year	19	19%
Age (Mean ±SD)	43.69±12.40	

Of the 100 study subjects in the present study majority were from age group 36 to 55 years of age (55%). The mean age of participants was 43.69±12.40.

Table II Distribution of study participants according to biochemical parameters

Variable		N	%
Hb gm%	<7	19	19%
	7-9	34	34%
	9-11	37	37%
	≥11	10	10%
Platelet count	Below Normal	30	30%
	Normal (1.5-4.5 Lakhs/ml)	70	70%
PT (Sec)	Normal (11-13.5 Sec)	62	62%
	Above Normal	38	38%
INR	Normal (0.8-1.1)	61	61%
	Above Normal	39	39%
Total Bilirubin	Normal (<1.2mg/dl)	53	53%
	Above Normal	47	47%
SGOT	Normal (7-55 IU/L)	63	63%
	Above Normal	37	37%
SGPT	Normal (8-48 IU/L)	63	63%
	Above Normal	37	37%
Serum Albumin	Below Normal	33	33%
	Normal (2.4-4 gm/dl)	67	67%
Serum Creatinine	Normal (60-110 mmol/L)	72	72%
	Above Normal	28	28%
Total		100	100%

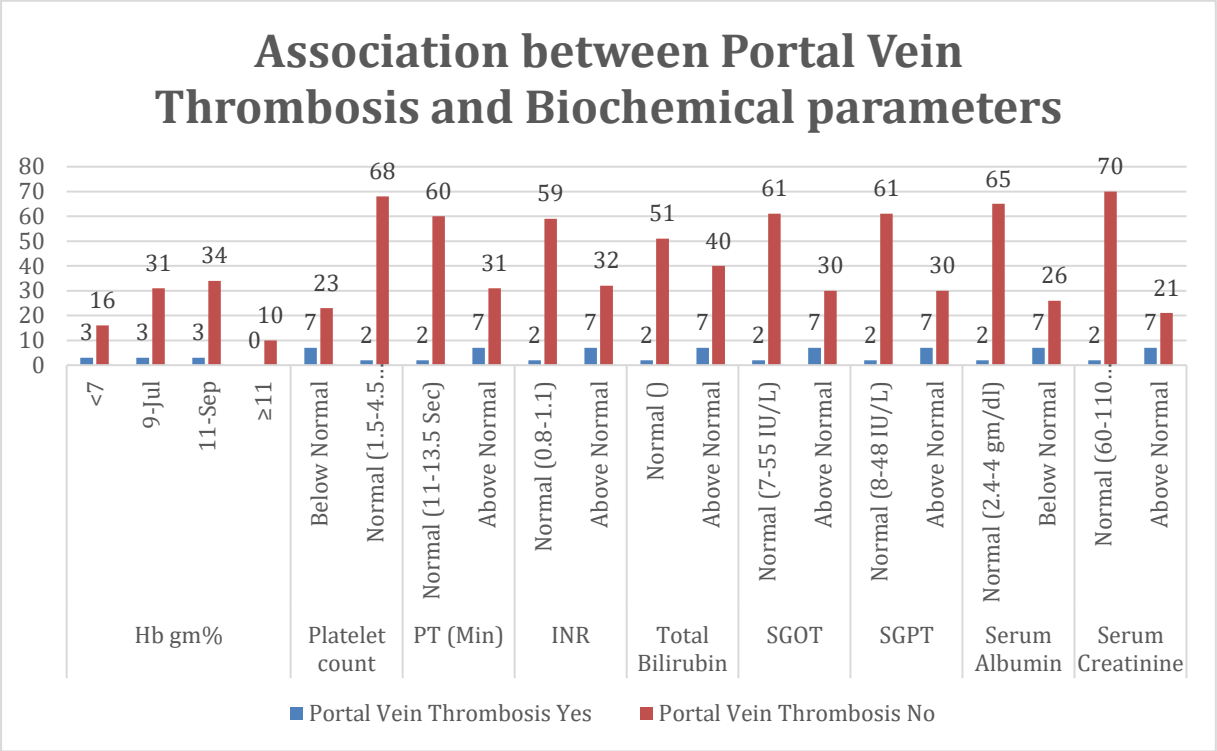
The majority of participants (90) have Hb value below 11Gm%. Serum bilirubin level was above normal in 47 participants. The SGPT/SGOT values were above normal in each 37 participants. Blood coagulation markers (INR in 39 and PT in 38) were above normal in participants. The 33 and 30 participants have below normal value of serum albumin and platelets count respectively.

Table III Association of Portal Vein Thrombosis and diameter of Portal and Splenic Vein

Variable		Portal Vein Diameter, in mm (Mean ±SD)	Splenic Vein Diameter, in mm (Mean ±SD)
Portal Vein Thrombosis	Yes	2.06±0.44	1.97±0.59
	No	1.43±0.20	2.10±1.00
P value		0.001	0.701

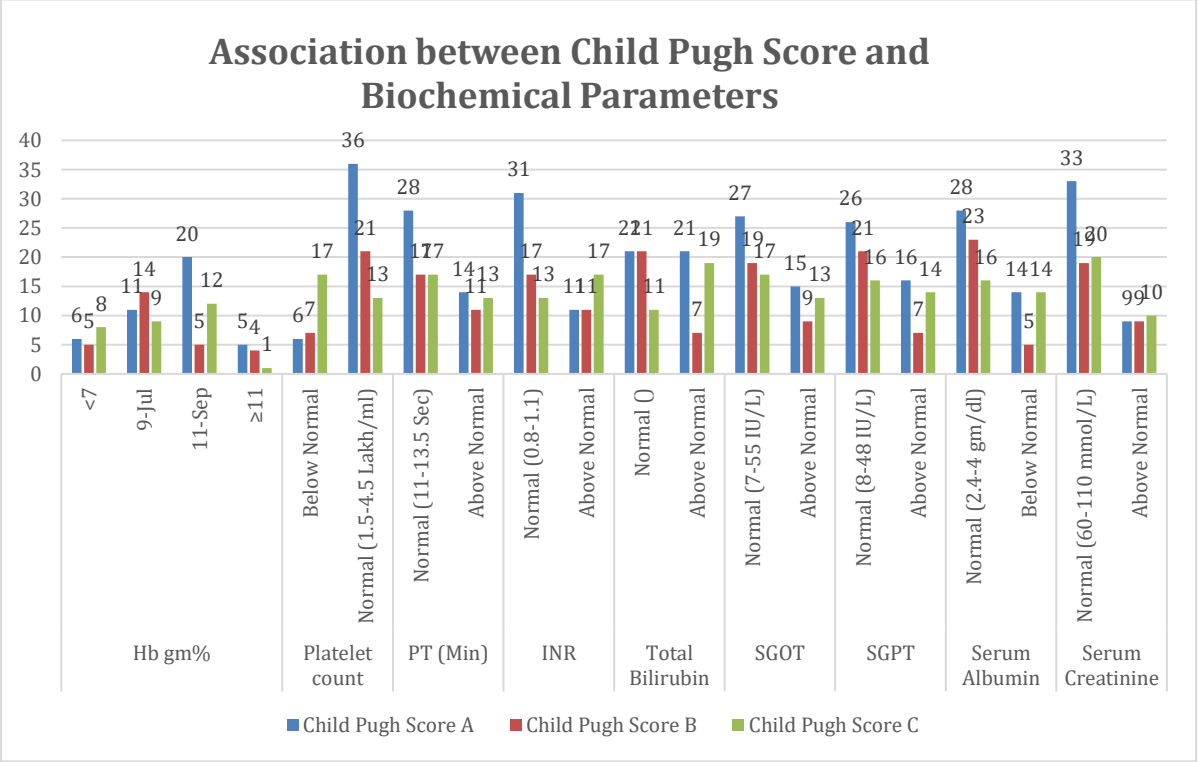
The subjects with portal vein thrombosis have portal vein diameter 2.06±0.44 mm which was significantly higher ($p<0.05$) than those not having portal vein thrombosis

Graph I Association between Portal Vein Thrombosis and Biochemical parameters



All 9 patients with portal vein thrombosis have Hb< 11 Gm%. Platelet count, PT (Min), INR, Total Bilirubin, SGOT, SGPT, Serum Albumin and Serum Creatinine all were abnormal in 7 patients each. Hemoglobin is significantly found low in PVT, when compared to non PVT, with a p value of 0.553. Platelet counts PT/INR value serum albumin, Serum creatinine is significantly found deranged is PVT, when compared to non PVT group with p value of 0.003, 0.025, 0.026, 0.005, 0.002 respectively. Whereas Total Bilirubin SGOT, SGPT values not significantly associated between PVT and non PVT group with P value of 0.079, 0.12, 0.12.

Graph II Association between Child Pugh Score and Biochemical parameters



In biochemical markers the Child Pugh score was significantly associated with platelets count, INR and total bilirubin ($p<0.05$).

DISCUSSION

The term portal vein thrombosis refers to the complete or partial obstruction of blood flow in the portal vein, due to the presence of a thrombus in the vessel lumen.[6] The first case of portal vein thrombosis was reported in 1868 by Balfour and Stewart, describing a patient presenting with splenomegaly, ascites, and variceal dilation. Portal vein thrombosis is a relatively rare condition with an overall incidence of 0.05%-0.5% in autopsy studies. The incidence varies, depending on the group of patients studied and the method used to diagnose portal vein obstruction.[7]

Although in the general population portal vein thrombosis is considered a rare event, its prevalence among cirrhotic patients ranges between 4.4%-15% and is responsible for about 5-10% of overall cases of portal hypertension.[8] Of all cases of portal hypertension in developing countries, 40% are attributed to portal vein obstruction, presumably secondary to an increased incidence of pylephlebitis associated with abdominal infections. In Japan, the frequency of portal vein obstruction is less than the Western countries.[9]

Sex and age-related demographics, no sex differences have been reported overall, except for a slight male predominance in patients whose obstruction is secondary to cirrhosis. The age distribution at the presentation of portal vein thrombosis depends upon the underlying disease. Primary portal vein thrombosis from coagulopathies occurs with equal frequency in adults and children.[10]

We found that of the 100 study subjects in the present study majority were from age group 36 to 55 years of age (55%). The mean age of participants was 43.69±12.40. Bolondi et al[11] reported that US could only identify 42% of patients with portal hypertension based on size of portal vein. However, a big portal vein, for example 15 mm in diameter is pathogonomic of presence of portal hypertension.

The majority of participants (90) have Hb value below 11 gm%. Serum bilirubin level was above normal in 47 participants. The SGPT/SGOT values were above normal in each 37 participants. Blood coagulation markers (INR in 39 and PT in 38) were above normal in participants. The 33 and 30 participants have below-normal serum albumin and platelet count values, respectively. Iwao T et al[12] showed that the liver vascular index is a highly sensitive and specific Doppler ultrasound parameter in the diagnosis of cirrhosis and portal hypertension. The best cut-off values were considered to be 13 cm/se of portal venous velocity and 1.1 of hepatic arterial pulsatility index, showing sensitivity and specificity of 83, 85, 84, and 81%, respectively. The best cut-off value of the liver vascular index was 12 cm/s with a sensitivity and specificity of 97 and 93%, respectively. The subjects with portal vein thrombosis have portal vein diameter 2.06±0.44 mm which was significantly higher than those not having portal vein thrombosis.

All 9 patients with portal vein thrombosis have Hb< 11 gm%. Platelet count, PT (Min), INR, total bilirubin, SGOT, SGPT, serum albumin, and serum creatinine all were abnormal in 7 patients each. Hemoglobin is significantly found low in PVT when compared to non-PVT. Platelet counts PT/INR value serum albumin, Serum creatinine is significantly found deranged is PVT, when compared to a non-PVT group, whereas total bilirubin SGOT, SGPT values not significantly associated between PVT and non-PVT group. We found that in biochemical markers the Child Pugh score was significantly associated with platelets count, INR and total bilirubin. Moriyasu et al[13] confirm an increase in the cross-sectional area of the portal vein in patients with portal hypertension. In this study, the CI in the patients with cirrhosis was 2.5 times higher than that in the normal subjects. Several factors that affect the CI of portal vein can be mentioned: portal venous pressure, portal vascular resistance in the liver, portal blood flow, and the development of portosystemic collateral pathways. In addition, the duration of the abnormal hemodynamics is an important factor affecting the CI, because the pathologic changes in the portal vein are progressive. The portal vein pressure-volume curve, or compliance curve, is not linear. Increased flow volume is compensated for by deformity of the blood vessel cross-section from elliptical to round, while elevation of portal pressure is minimized.[14] However, this is true only in the case of normal hemodynamics. When the pressure is elevated, a higher part of the compliance curve is used, and the change in pressure is larger than the volume change.

CONCLUSION

Authors found that 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. Early diagnosis, treatment and monitoring of the patients can prevent the occurrence of PVT in liver cirrhosis and also improve the liver function and survival.

REFERENCES

1. Niederau C et al. Sonographic measurements of the liver, spleen, pancreas, and portal vein. Radiology 1983; 149: 537-540.
2. Vine HS et al. Portal vein aneurysm, AJR 1979; 132:557-560.
3. Potze W, Arshad F, Adelmeijer J, et al. Routine coagulation assays underestimate levels of antithrombin-dependent drugs but not of direct anticoagulant drugs in plasma from patients with cirrhosis. Br J Haematol 2013;163:666-673.
4. Ageno W, Beyer-Westendorf J, Garcia DA, Lazo-Langner A, McBane RD, Paciaroni M. Guidance for the management of venous thrombosis in unusual sites. J Thromb Thrombolysis 2016;41:129-143.
5. Intagliata NM, Maitland H, Northup PG, Caldwell SH. Treating thrombosis in cirrhosis patients with new oral agents: ready or not? Hepatology 2015;61:738-739.

6. Martinez M, Tandra A, Vuppalanchi R. Treatment of acute portal vein thrombosis by nontraditional anticoagulation. Hepatology 2014;60:425-426.
7. Intagliata NM, Henry ZH, Maitland H, et al. Direct oral anticoagulants in cirrhosis patients pose similar risks of bleeding when compared to traditional anticoagulation. Dig Dis Sci 2016;61:1721-1727.
8. Ponziani FR, De Candia E, De Cristofaro R, Pompili M. Portal vein thrombosis occurrence in a cirrhotic patient during treatment with rivaroxaban. Liver Int 2017;37:1251.
9. Lenz K, Dieplinger B, Buder R, Piringer P, Rauch M, Voglmayr M. Successful treatment of partial portal vein thrombosis (PVT) with low dose rivaroxaban. Z Gastroenterol 2014;52:1175-1177.
10. Pannach S, Babatz J, Beyer-Westendorf J. Successful treatment of acute portal vein thrombosis with rivaroxaban. ThrombHaemost 2013;110:626-627.
11. Bolondi L et al. Ultrasonography in the diagnosis of portal hypertension: diminished response of portal vessel to respiration. Radiology 1982; 142:167-172.
12. Iwao T. 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):1012-7.
13. Moriyasu F et al. Congestion index of the portal vein. AJR 1986; 146: 735-739.
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.