ORIGINAL RESEARCH PAPER
Volume - 11 | Issue - 05 | May - 2022 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

THE STUDY OF PORTAL VEIN THROMBOSIS PATIENTS WITH CHRONIC LIVER DISEASE

Medicine

Dr. Umesh Kumar Prajapati*	P.G. Student, Department of Medicine, Gajra Raja Medical College, Gwalior (M.P.). *Corresponding Author
Dr. Archana Kansal	Professor, Department of Medicine, Gajra Raja Medical College, Gwalior (M.P.).
Dr. Anupam Raj Gaurab	P.G. Student, Department of Medicine, Gajra Raja Medical College, Gwalior (M.P.).
Dr. Pankaj Kumar Gupta	P.G. Student, Department of Medicine, Gajra Raja Medical College, Gwalior (M.P.).
Dr. Suresh Jadi	P.G. Student, Department of Medicine, Gajra Raja Medical College, Gwalior (M.P.).
Dr. Miskila Subba	Senior Resident Department of Medicine, Gajra Raja Medical College, Gwalior (M.P.).

ABSTRACT

BACKGROUND: Portal vein thrombosis in the general population is a rare event, but it occurs relatively frequent in patients with cirrhosis and its prevalence increases with severity of the disease. 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

OBJECTIVE: To find out the incidence of portal vein thrombosis in Chronic Liver disease patients and factors and complications associated with it .

MATERIAL AND METHODS: All the patients of chronic liver disease attending department of General Medicine, Jayarogya Hospital patients were evaluated for detailed history, abdominal ultrasonography, portal vein colour doppler studies ,coagulation profile and other biochemical profile.

RESULTS: In this study 9% of patient developed PVT with commonest etiology being chronic alcohol intake. Chronic alcohol intake is a significant etiological factor for the development of PVT in patients of preexisting CLD. Hemetemesis, Encephalopathy, Splenomegaly and ascites were found significantly higher proportion in PVT group when compared with non PVT group. Portal vein thrombosis could worsen the rate of hepatic decompensation and survival of cirrhosis.

CONCLUSION: Portal vein thrombosis is considered to be a significant complication of liver cirrhosis. Portal vein thrombosis could hasten the rate of decompensation and worsen the survival of cirrhotic patient. Prognostic value of PVT is cirrhosis remain a grey zone.

KEYWORDS

Chronic liver disease, Portal vein thrombosis

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 .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%.[1]

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 .Therefore ,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 stage of chronic advanced liver disease .

Diagnosis of PVT is a significant milestone in the natural history of cirrhosis which is associated with worsening liver function ,ascites and occurrence of esophageal variceal bleeding. Portal vein thrombosis increases morbidity and mortality associated with the liver transplantion and may even contraindicate it.[2]

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 .In this study we are studying the prevalence of PVT symptoms, its clinical relevance and biochemical factors associated.

OBJECTIVES

1. To find out the incidence of portal vein thrombosis in patients of chronic liver disease .
2. To find out the factors and complications associated with portal vein thrombosis in CLD patients.

MATERIALS AND METHODS

I. Present study was a prospective study conducted in the Department of General Medicine, G. R. Medical College (M.P). 100 CLD patient was selected and evaluated for the presence of Portal vein thrombosis through USG .Patients with age less than 18 years , patients above 65 years were excluded from the study. Documented or detected cases of coronary artery diseases, pre-existing benign cardiac arrhythmia, pre-existing cardiomyopathies secondary to non-cirrhotic and nonalcoholicetiology, patients of chronic kidney disease, chronic inflammatory conditions, heart failure, hypertension, cerebrovascular diseases, COPD were also excluded from the study. CLD patients having PVT were evaluated for the presence of complications and parameters like CBC ,PT INR, LFT, RFT, PTINR and Child pugh score was calculated and compared with CLD patients without PVT .

RESULTS

Table 1 : Distributions of study participants according to USG and Colour Doppler findings

			Number	%
USG Findings	Portal Vein Thrombosis	Yes	9	9%
		No	91	91%
Colour Doppler finding	Portal Vein Diameter	Normal (7-15 mm)	71	71%
		Above Normal	29	29%
	Splenic Vein Diameter	Normal (10 mm)	11	11%
		Above Normal	89	89%
	Portal Vein Obstruction	Yes	6	6%
		No	94	94%
Child Pugh Class		A	42	42%

	B	28	28%	
	C	30	30%	
Total			100%	100%

The 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%).

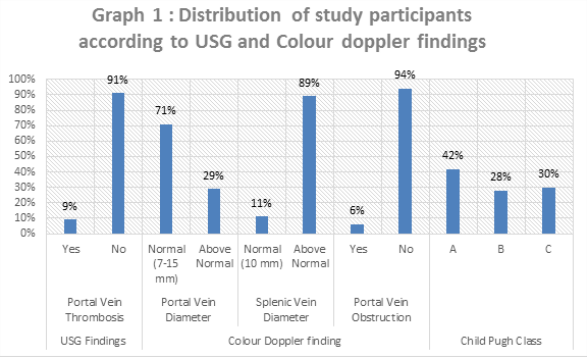


Table 2 : Association of Portal Vein Thrombosis and diameter of Portal and Splenic Vein

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±0.44mm which was significantly higher ($p<0.05$) than those not having portal vein thrombosis.

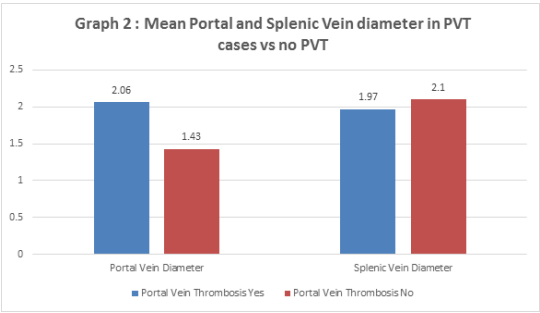


Table 3 : Association between Portal Vein Thrombosis and Etiological Factors

		Portal Vein Thrombosis				
		Yes(N =9)		No(N=91)		
		Number	Percentage (%)	N	Percentage (%)	P Value
Alcohol	Yes	4	44.44	11	12.87	0.009
	No	5	18.6	80	87.9	
HbsAg	Positive	3	33.3	17	18.6	0.294
	Negative	6	86.6	74	81	
HCV	Positive	1	11.1	3	3.3	0.253
	Negative	8	88.88	88	96.7	
Others	Yes	1	11.1	17	18.6	0.572
	No	8	88.8	74	81.31	

In etiological factors Alcohol intake showed a significant association with portal vein thrombosis. Viral infections like Hepatitis B, C and other etiologies had no statistically significant association with Portal vein thrombosis.

Chronic alcoholic intake is significantly found as on etiological factor in CLD, patients with PVT when compared to non PVT, with a p value of 0.009 whereas etiological factors title HBSAg and HCV are no significantly found associated between PVT group and not PVT group with P value of 0.294 and 0.253 respectively.

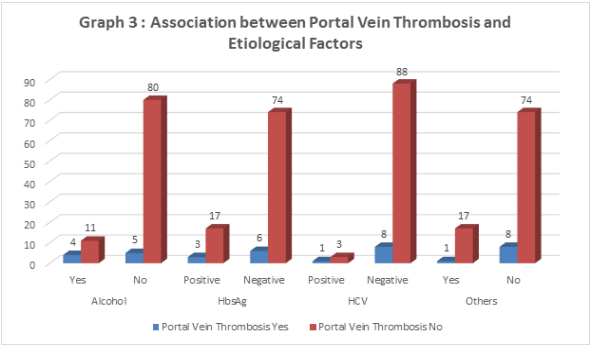


Table 4 : Association between biochemical parameters in PVT and NON PVT

Variable	PVT(9)	Non PVT (91)
Hb %	7.9±1.8	8.6±2.3
Platelet count (lacs)	1±0.5	1.9±0.9
PT (sec)	16.3±3.6	14.1±3.2
INR	1.3±0.2	1.1±0.2
Total Bilirubin (mg/dl)	4.5±4.5	3±5.2
AST(IU/l)	247±449	85±108.3
ALT(IU/L)	75.6±47	69.9±101.3
Serum albumin (gm)	2.1±0.4	2.8±0.8
Serum creatinine mg	1.3±0.4	1±0.3
CD PVD (CM)	2.1±0.4	1.4±0.2
CD SVD (cm)	2±0.6	2.1±1

In the study mean Hemoglobin in PVT group is 7.9±1.8 where as non PVT group is 8.6±2.3.Mean platelet count in PVT group is 1±0.5 lac whereas in non PVT group is 1.9±0.9.Mean PT in PVT group is 16.3 ±3.6 whereas in non PVT group is 14.1±3.2.Mean total bilirubin in PVT group is 4.5 ±4.5 whereas in non PVT group is 3±5.2.Mean SGOT in PVT group is 247 whereas in non PVT group is 85. Mean SGPT in PVT group is 75 whereas in non PVT group is 69.Mean serum albumin in PVT group is 2.1±0.4 and non PVT group is 2.8±0.8 .Mean serum creatinine in PVT group is 1.3±0.4 and non PVT Group is 1±0.3.Mean Portal vein diameter in PVT group is 2.1±0.4 whereas in non PVT group is 1.4±0.2.

Table 5 : Correlation between child pugh score and PVT

	USG portal vein thrombosis		
	Yes	No	Total
Child pugh score A	0	42	42
Child pugh score B	1	27	28
Child pugh score C	8	22	30
Total	9	91	100
P value :<0.001			

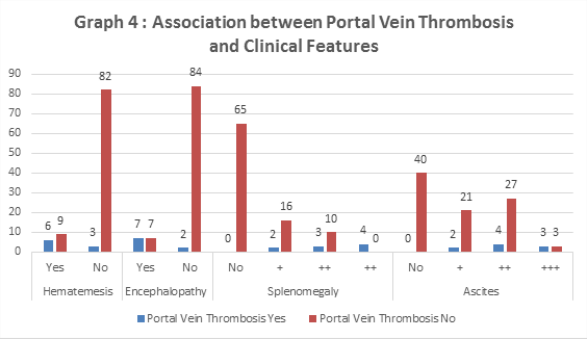
Majority of patients with portal vein thrombosis belonged to Child pugh score C ie88% and 12 % belonged to Child pugh score B There is significant relation between Child pugh scoring and Portal vein thrombosis with P value of 0.001.which shows CLD patients with PVT are having grave prognosis

Table 6 : Association between Portal Vein Thrombosis and Clinical Features

Variable		Portal Vein Thrombosis		
		Yes	No	P Value
Hematemesis	Yes	6	9	0.001
	No	3	82	
Encephalopathy	Yes	7	7	0.002
	No	2	84	
Splenomegaly	No	0	65	0.001
	+	2	16	
	++	3	10	
	++	4	0	
Ascites	No	0	40	0.002
	+	2	21	
	++	4	27	
	+++	3	3	
Total		9	91	100

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 is 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

The study is a prospective observational study carried out on 100 patients with CLD attending JAH Hospital fulfilling inclusion and exclusion criteria and patients with PVT and NON PVT among these patients has been studied.

In the study PVT (portal vein thrombosis) was found in 9% of the study population, which was comparable to study done by Francesco et al (8%).In study done by Huisong et al [3],PVT was present in 24.7%.In study by Kumaragurbaran S et al[4] PVT was found in 33 (18.1%) of which 22 were male and 11 were female. In study by Lucio Amitrano et al[5] 79(11.2%) were found to have PVT.

In etiological factors Alcohol is showing a significant association with portal vein thrombosis. Viral infections like Hepatitis B, C and other had no statistical significance with Portal vein thrombosis. Chronic alcohol intake is significantly found as on etiological factor in CLD, patients with PVT when compared to non PVT, with a p value of 0.009 whereas etiological factors like HBSAg and HCV are no significantly found between PVT group and not PVT group with P value of 0.294 and 0.253 respectively. In the study by Kumaraguruban et al[4],commonestetiology being Hepatitis B(72 .7%)In study by Huisongchen et al[3],PVT occurred in 34 cirrhotic patients following HBV, in two following HCV and 2 patients each with either autoimmune or crytogenic cirrhosis. However PVT was not observed in alcoholic patients.

Majority of patients with portal vein thrombosis belonged to Child pugh score C ie 88% and 12 % belonged to Child pugh score B and no patients belonged to child pugh A score. There is significant relation between Child pugh scoring and Portal vein thrombosis with P value of 0.001.which shows CLD patients with PVT are having grave prognosis. In study done by Huisong et al [3],4(10%) were in Child pugh class A, 20(50%) in class B and 16(40%) in class C. Child pugh score were not significantly different between patients with or without PVT .In Study by Francesco et al [6]17 % were in Child pugh score C and 42 % were in child pugh score B and 42 % were in class A.

All the 9 patients of portal vein thrombosis had Splenomegaly and Ascites while 7 patients also had Encephalopathy and 6 had Hematemesis along with portal vein thrombosis. Hematemesis encephalopathy, splenomegaly ascites is significantly found associated with PVT group when compared with non PVT with p values of 0.001, 0.002, 0.001 and 0.002 respectively. The above finding is comparable to study done by Huisongchen et al[3] where GI bleeding, abdominal pain, abdominal distension, ascites, jaundice and hepatic encephalopathy were common clinical presentations among patients in PVT group. In a study by Francesco et al[6] the common clinical presentation in PVT group were ascites and hepatic encephalopathy. In study by Lucio et al[5] the PVT group CLD patients frequently presented with portal hypertensive bleed, abdominal pain and intestinal infarction.

All 9 patients with PVT have Hemoglobin<11 gm% .with 3(33%)patients having severe anemia. No significant relation is observed in Hemoglobin values between PVT group and non PVT group with p value of 0.553.In Kumaragurahan et al[4] study haemoglobin (p value <0.001) is significantly lower in PVT group as compared to non PVT subjects. In study by Huisong et al[3] Hb level is significantly lower in PVT patients. Hb level is expected to be an independent risk factor for PVT. In fact because splenomegaly decreases the Hb level and hypersplenism is more severe in PVT patients, the lower Hb levels, may be more appropriately attributed to splenomegaly.

In the study platelet count is below normal in 7(77%) out of 9 PVT patients compared to 31 % in NON PVT patients. There is significant association is observed in platelet count between PVT and non PVT group with p value of 0.003.In the study done by Francesco et al[6] 61% of the patients with PVT had platelet count below 75000.with 25% had platelet count in the range of 76000-125000.only 5% of PVT patients have normal PVT .In study by Huisong et al[153] the mean platelet count in CLD patients with PVT is 59.1±21.4 which is very much low when compared to mean platelet count in non PVT CLD patients (94.2±59.1).In study by Kumaraguruban et al[4], the mean platelet count in PVT subjects (0.58±0.18) is lower when compared to non PVT subjects (0.86±0.21).

Lower platelet count can also be attributed to splenomegaly since splenomegaly is more evident in PVT patients than in non PVT patients.

The PT -INR value was deranged in 77.7 % of PVT group CLD patients compared to 35 % of non PVT population showing a significant higher incidence of coagulopathy in PVT patients when compared to non PVT patients. No statistically significant association is observed in PT INR values among PVT and non PVT group with p value of 0.025 and 0.026 respectively. In study done by Kumaraguruban et al[4] the mean INR of the PVT subjects (1.38±0.81) is comparable to mean in non PVT subjects (1.41±0.19) with a p value of 0.69.

In study by Huisong et al[3] the PT mean in PVT(13.7±2.0)and non PVT (14.4±3.1)subjects are not significantly related. In study by Francesco et al[154] the mean PT INR in PVT group was 1.33±0.22 whereas in non PVT subjects was 1.30±0.34 and was not significantly related with a p value of 0.818.PT INR value did not appear to affect the risk of developing PVT in these studies.

In the study mean total bilirubin in PVT patients is 4.5±0.5 and in non PVT patients is 3±0..2. Bilirubin values are not significantly related between PVT and NONPVT patients in CLD with p value of 0.079.In the study by Francesco et al[154], mean total bilirubin in PVT 2.4±3.8 and NON PVT is 2.2±3.3.In study by Huisong et al[3], mean total bilirubin in Non PVT patients are 44.6 ± 46.7 and PVT patients are 31.8±10.6.In study by Kumaraguruban et al[4] the mean bilirubin in PVT patients is 3.18± 1.41 and non PVT patients is 2.8 ±0.8.

In the study, mean albumin in PVT patients was 2.1±0.4 and non PVT patients is 2.8 ±0.8 and shows significant relation with P value of 0.005. Mean albumin in PVT group is lower when compared to non PVT group. In study by Huisong et al[3], the mean albumin in PVT patient is 29.1±4.7 and non PVT group is 26.4±6.3 In study by Francesco et al[6], the mean albumin in PVT group is 3.5±0.7 and non PVT is 3.4±0.6.

In the study mean creatinine in PVT group is 1.3±0.4 and non PVT is 1±0.3 .Mean creatinine is higher in PVT compared to non PVT which shows statistically significant relation with p value of 0.002 .In the study done by Francesco et al[6], the mean creatinine in PVT group is 0.92±0.42 and in non PVT group is 0.95 ±0.64.

CONCLUSION

Portal vein thrombosis is considered to be a significant complication of liver cirrhosis. Chronic alcoholic intake is a significant etiological factor for the development of PVT in patients of prexisting CLD . Hemetemesis, Encephalopathy, Splenomegaly and ascites were significantly found associated with PVT group when compared with non PVT group. There is significant association is observed with low platelet count, high S. creatinine and low albumin between PVT and non PVT group. Portal vein thrombosis could hasten the rate of hepatic decompensation and worsen the survival rate in cirrhotic patients.

REFERENCES

1 Tsochatzis EA, Senzolo M, Germani G .et al. Systematic review portal vein thrombosis. Aliment PharmacolTher2010 ;31:366-74.
2 Harding DJ, Perera MT, Chen F etal.Portal vein thrombosis in cirrhosis, controversies and latest development world J Gastroenterol 2015 ;21:6769-84.
3 Huisong Chen, Goolsb Trilok, Xiaolong Qi, Junjie Xiao et al Division of Gastroenterology and Hepatology, Digestive Disease Institute, Tongji Hospital, Tongji University school of Medicine, Shanghai, P R China.
4 Kumaraguruban S, Narayanaswamy K, Chezian A, Senthilkumar R, Akilandeswari AR, Santhiselvi A, Department OF Hepatology, Madras medical college, Rajiv Gandhi government General Hospital, Chennai 600003, India.
5 Lucio Amitrano et al. Risk factors and clinical presentation of portal vein thrombosis in patients with liver cirrhosis J hepatool.2004 may 40(5):736-41.
6 Francesco Viloi, Gino Robertso et al, Department of Internal Medicine and Medical specialities Sapienza University of Rome Italy.