

Assessment of Portal Vein Thrombosis in Cirrhotic Liver Patients

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ABSTRACT

Background: PVT has a number of frequent causes, including cirrhosis of the liver, abdominal inflammation, tumour invasion, and thrombophilic disorders.

Aim: To find out how frequently liver cirrhosis patients get portal vein thrombosis.

Study Design: Cross-sectional study, Department of Medicine, Liaquat University Hospital Hyderabad, Sindh, Pakistan's from 1st April 2021 to 30th September 2021. One hundred and twenty eight patients were enrolled. The demographic information like age, sex, and body mass index were noted. Hepatocellular carcinoma patients and history of thromboembolism propensity were excluded. Both male and female patients with hepatic cirrhosis and age ranged from 20 to 50 were included. The monitoring of portal vein thrombosis, Doppler ultrasonography was performed on all patients.

Results: There were 40(34.4%) female patients and 84(65.6%) male patients with average age was 51.95 7.54 years and BMI was 31.87 2.64 kg/m². Seventy nine patients (61.7%) had hepatitis C, 50 patients (39.3%) had hepatitis B, 66 patients (51.6%) had diabetes mellitus, 81 patients (63.3%) had hyperlipidaemia, and 69 patients (57.9%) had hypertension. The prevalence of portal vein thrombosis (PVT) was 81(63.3%), with 48(59.3%) of the cases involving men and 33 (40.7%) involving females. Of them, 25 patients (50%) had hepatitis B and 44 (55.7%) had hepatitis C.

Conclusion: Patients with liver cirrhosis experienced portal vein thrombosis often, and hepatitis C patients made up the majority of those afflicted.

Keywords: Thrombosis, Portal vein, Liver cirrhosis, thrombophilic disorders

INTRODUCTION

When a blood clot forms in the PV, it prevents normal blood flow, which is known as portal vein thrombosis (PVT). From a pathophysiological perspective, thrombus development and, eventually, PVT, are both attributed to an imbalance of the haemostatic system. PVT is more common, and it becomes more common in cirrhotic individuals with hepatocellular carcinoma (HCC) when the disease is more advanced¹⁻⁴. This diagnosis is more common because of advancements in imaging tests and increased physician awareness. However, there are several disputes around the management.

The size and extent of the thrombus have been used by Yerdel et al⁵ to categorise PVT. The functional ramifications of PV blockage, which can have a negative impact on liver function, are not discussed at all, in this categorization. A more thorough categorization scheme for PVT in cirrhosis has been established by Sarin et al⁶ emphasising PVT functioning.

In their daily clinical work, hepatologists must make difficult decisions on how to treat PVT in cirrhotic patients. What is the least dangerous course of anticoagulant therapy for PVT, or what is the ideal dosage and duration of treatment, taking into account portal hypertension? The importance of each of these queries increases for transplant patients. There are still questions that need to be answered definitively about PVT's effects and how it affects the prognosis of cirrhosis. Patients with liver cirrhosis or portal hypertension are predicted to have a PVT prevalence of 0.6 to 15.8%⁷⁻⁹. With the progression of cirrhosis, PVT prevalence rises. It has been found to range from as low as 1%⁷ to 8-25% in patients with compensated cirrhosis.⁹⁻¹¹ The total frequency of PVT was 15.9% in a recent study¹² of 219 cirrhotics awaiting LT, similar to the 8-25% described in earlier studies¹³. The stated incidence varies depending on the imaging technique used to evaluate PVT. According to ultrasonography, the prevalence ranges from 10 to 25 per cent^{9,10}.

In 885 candidates for LT, cirrhosis caused by the hepatitis B virus and alcohol were identified as common causes of PVT when looking at aetiology.⁹ In contrast, no correlation between the aetiology of liver disease and the incidence of PVT was discovered in a different research of 219 candidates for LT.¹⁴ PVT is typically identified in individuals with Child-Pugh classes B and C cirrhosis. Patients with cirrhosis and HCC are more likely to experience PVT,

which can occur up to 35% of the time¹⁵⁻¹⁷. PVT development in cirrhosis involves a number of factors. In liver cirrhosis, lower portal flow velocity is a key risk factor for PVT^{18,19}.

Deficiencies in pro-coagulant factors, problems in fibrinolysis and cirrhotics have historically been thought to be more prone to bleeding²⁰. But recently, there has been mounting proof that hypercoagulability is a significant aspect of the cirrhosis haematological spectrum²¹, which is confirmed by the discovery of elevated thrombin production in portal blood samples from 28 cirrhotic patients²².

Existing data, however, came from case reports from tiny studies. In a few recent investigations, relationships between PVT expression in cirrhotic patients and thrombopoietin receptor agonists were discovered.

The objective of the study was to find out how frequently liver cirrhosis patients get portal vein thrombosis.

MATERIALS AND METHODS

This cross-sectional study was conducted at Department of Medicine, Liaquat University Hospital Hyderabad, Sindh, Pakistan's from 1st April 2021 to 30th September 2021 and after permission from IRB 128 patients were enrolled. After obtaining written consent, specific demographic information, such as age, sex, and body mass index, was logged. Hepatocellular carcinoma patients, those with a history of thromboembolism propensity, and those who were not included in the study were eliminated. Both male and female patients with hepatic cirrhosis were included. The ages of the patients ranged from 20 to 50. For the purpose of monitoring portal vein thrombosis, Doppler ultrasonography was performed on all patients. The SPSS-17.0 was used to analyse all the data.

RESULTS

There were 40(34.4%) female patients and 84(65.6%) male patients. The patient's average age was 51.95 7.54 years, and their average BMI was 31.87 2.64 kg/m². Seventy nine patients (61.7%) had hepatitis C, 50 patients (39.3%) had hepatitis B, 66 patients (51.6%) had diabetes mellitus, 81 patients (63.3%) had hyperlipidaemia, and 69 patients (57.9%) had hypertension (Table 1).

The prevalence of portal vein thrombosis (PVT) was 81(63.3%), with 48(59.3%) of the cases involving men and 33(40.7%) involving females. Of them, 25 patients (50%) had hepatitis B and 44(55.7%) had hepatitis C (Table 2).

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Table 1: Demographics of the people with cirrhosis

Variable	No.	%
Gender		
Male	84	65.6
Female	44	34.4
Age (years)	51.95±7.54	
BMI (Kg/m ²)	31.87±2.64	
Types of Diseases		
Chronic viral hepatitis B	50	39.1
Chronic viral hepatitis C	79	61.7
Diabetes mellitus	66	51.6
Hyperlipidaemia	81	63.3
Hypertension	69	53.9

Table 2: Prevalence of portal vein thrombosis with different diseases

Disease	Portal Vein Thrombosis	
	Yes	No
Hepatitis B	25 (50%)	25 (50%)
Hepatitis C	44(55.7%)	35(77.3%)
Diabetes mellitus	47(71.2%)	19(28.8%)
Hyperlipidaemia	57(70.4%)	24(29.6%)
Hypertension	49(71.0%)	20(29.0%)

The duration of liver cirrhosis in patients is shown in table 3.

Table 3: Duration of liver cirrhosis

Duration of liver cirrhosis (months)	n	%age
6-12	28	21.9
12-24	63	49.2
> 24	37	28.9

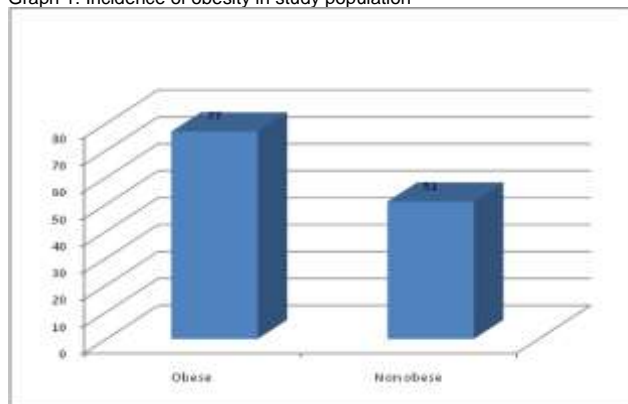
Residence of the study population is given in table 4.

Table 4: Residence of the study population

Residence	n	%age
Urban	69	53.9
Rural	59	46.1

The Incidence of obesity in study population is given in graph 1.

Graph 1: Incidence of obesity in study population



DISCUSSION

Portal vein thrombosis has a variety of clinical manifestations. If thrombus development is partially occlusive in the acute phase, it may not cause any symptoms or it may be accompanied by minor stomach discomfort, nausea, vomiting, diarrhoea, and lack of appetite. If PVT is fully developed, however, it may present as sudden or recurrent abdominal pain as well as signs of chronic liver disease decompensation, such as variceal haemorrhage or ascites. Bloody diarrhoea, peritonitis symptoms, intestinal ischemia, and portal cholangiopathy are possible additional signs and symptoms. A cirrhotic patient's abrupt clinical deterioration which indicates the start of PVT is ascites, and resistant to diuretics, thus it is important to carefully evaluate this possibility.

Life-threatening intestine infarction may occur when the superior mesenteric vein is affected. There may also be involvement of the splenic veins⁷.

In comparison to individuals with cirrhosis alone, the risk of portal hypertensive haemorrhage is three times higher in people suffering from PVT. Hepatic artery vasodilatation is often able to retain liver function in the event of an abrupt total blockage of the PV. Due to the possibility of pre-existing portal hypertension and collaterals from chronic liver disease, it might be challenging to discriminate between acute and chronic PVT in cirrhosis²³.

Portal vein thrombosis occurs incidentally in the majority of cirrhotic patients, frequently during routine ultrasonography, CT, or MRI examination. Doppler ultrasonography is the primary PVT detection method²⁴.

Portal vein thrombosis is a frequent side effect of advanced liver disease in liver transplants. The total prevalence of PVT in our sample of cirrhotic patients was 63.3%, which is comparable to the 8% to 25% frequency found in other studies of individuals who have had liver transplants. The solely employed sonography to diagnose PVT or that included less critical patients the incidence of PVT was 0.6%²⁵.

In the present study, the average age was 51.95±7.54 years and BMI was 31.87±2.64kg/m². Eighty four (65.6%) were males and 40(34.4%) patients were females. These results were matched with the study of Saleem et al²⁶. Despite the fact that Lertpipometha and Auewarakul's²⁷ investigation revealed that HBV was the primary etiological factor in the development, they noted that 10 of 15 patients who had portal venous thrombosis tested positive for either HCV (66.7%) or HBV (33.3%).

The main etiological factor in this investigation was HCV. Additionally, it was shown that 53.3%, or 8 out of 15 patients, were in the 45 to 70 age range when they developed portal vein thrombosis. In individuals with the greatest PVT rate of cirrhosis associated with the alcoholic and hepatitis B viruses, several prior research have discovered that cirrhosis aetiology can have an impact on the prevalence of PVT²⁷⁻²⁹.

CONCLUSION

Patients with liver cirrhosis experienced portal vein thrombosis often, and hepatitis C patients made up the majority of those afflicted.

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2. Drafting the manuscript or revising it critically for important intellectual content.
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