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Prevalence of portal vein thrombosis in pediatric patients with cirrhosis and intrahepatic non-cirrhotic portal hypertension

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ABSTRACT

Introduction: Portal vein thrombosis (PVT) has been increasingly diagnosed in pediatric patients owing to the widespread use of non-invasive radiological techniques. Although the prevalence of PVT in adults with cirrhosis and intrahepatic non-cirrhotic portal hypertension ranges from 0.6 to 26 % and 13 to 46 %, respectively, no available data exist in the pediatric population. The prevalence of PVT in children with cirrhotic and intrahepatic non-cirrhotic portal hypertension was evaluated in this study.

Methods: This retrospective study included children with cirrhosis and intrahepatic non-cirrhotic portal hypertension, which consisted of congenital hepatic fibrosis (CHF) and idiopathic noncirrhotic portal hypertension (INCPH). Patients with extrahepatic portal venous obstruction were excluded from the study. The presence of PVT was evaluated using abdominal Doppler ultrasonography and/or CT. Etiological, clinical, and laboratory findings were compared between the groups.

Results: One hundred and forty-two patients with cirrhosis (mean admission age: 64.6 months ± 66.4, mean follow-up duration: 46.8 months ± 45.6) and 41 patients with non-cirrhotic patients (CHF=16, INCPH = 25, mean admission age: 126 months ± 64.2) were enrolled in this study. The prevalence of PVT was not significantly different between cirrhotic (8.5 %) and non-cirrhotic (9.7 %) patients. The incidence of PVT was significantly higher in patients with biliary atresia than in those with other etiologies in the cirrhotic group ($p = 0.022$). The frequency of PVT was higher in patients who had Child-Pugh score ≥7 in the cirrhotic group, but the difference was not statistically significant ($p = 0066$). The PVT group required more liver transplantations than the non-PVT group ($p = 0.038$).

Conclusion: The prevalence of PVT was similar in pediatric patients with cirrhosis and intrahepatic non-cirrhotic portal hypertension in our cohort, which is compatible with adult studies. Biliary atresia is found to be an important risk factor for PVT in our pediatric population. It might be associated with rapid progression of the disease, ascending cholangitis, and embryological abnormalities. These patients should be routinely evaluated to identify portal vein complications and early warning signs during follow-up.

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1. Introduction

Portal vein thrombosis (PVT) refers to the interruption of normal blood flow in the portal vein, which may involve the main trunk and/or branches of the portal vein [1]. It is a significant complication of the natural course of cirrhosis and appears to be associated with decreased venous flow caused by portal hypertension secondary to increased resistance in hepatic venules, endothelial dysfunction, prothrombotic effects, and other factors [2].

Congenital hepatic fibrosis (CHF) and idiopathic non-cirrhotic portal hypertension (INCPH) are non-cirrhotic intrahepatic presinusoidal causes of portal hypertension. The pathogenesis of CHF involves ductal plate malformation and is defined by varying degrees of periportal fibrosis and irregular proliferation of the bile ducts. INCPH includes a histological diagnosis of obliterative portal venopathy and is characterized by portal hypertension in the absence of cirrhosis and other causes of non-cirrhotic portal hypertension. Both conditions are characterized by a normal hepatic venous pressure gradient, moderate to massive splenomegaly, and preserved synthetic liver functions [3–6]. Prothrombotic disorders and local factors around the portal vein are the incriminating factors [7]. The relationship between non-cirrhotic intrahepatic presinusoidal causes of portal hypertension and PVT remains controversial.

Owing to the widespread availability of noninvasive diagnostic imaging tests, PVT has been increasingly recognized in adult

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patients with cirrhosis. Previous studies have reported an association between PVT and survival in adult patients with cirrhosis [8]. The prevalence of PVT in the cirrhotic population ranges from 1 % in patients with compensated cirrhosis to as high as 8–25 % in patients with decompensated cirrhosis [9]. Data from recent series reveal the prevalence of PVT ranging from 13 to 46 % in adult patients with hepatoportal sclerosis [6,10,11]. While the association between PVT and adult cirrhotic patients has been well documented, no data exists regarding its prevalence and clinical significance in pediatric populations or in non-cirrhotic portal hypertension due to CHF or INCPH. The scarcity of studies addressing the development of PVT in these groups represents a significant gap in the literature.

To our knowledge, the prevalence of PVT in pediatric patients with cirrhosis and CHF and INCPH has not yet been estimated in the literature. We retrospectively explored the prevalence of PVT development in pediatric cirrhotic patients with any etiology or severity and compared it with intrahepatic non-cirrhotic causes of portal hypertension. In this study, we also aimed to determine the clinical consequences of PVT, specifically its role, if any, in the progression of cirrhosis and its influence on outcomes in patients with cirrhosis.

2. Materials and methods

This study was conducted in children with cirrhosis or intrahepatic non-cirrhotic portal hypertension (CHF and INCPH) at a single center between 1997 and 2023. Liver cirrhosis was diagnosed with histopathological and/or clinical, imaging, and laboratory findings. . The diagnosis of CHF was made when histopathology was compatible with CHF and/or when a mutation of the PKHD1 (polycystic kidney and hepatic disease 1) gene was detected. The diagnosis of INCPH was made in the absence of cirrhosis on liver biopsy and signs of portal hypertension or histological signs of INCPH [12]. Clinical data, including demographic information, clinical manifestations, laboratory indices, imaging findings, treatment, and follow-up, were collected. The Child-Pugh scores of the patients with cirrhosis were calculated. PELD or MELD scores were not used because they would cause heterogeneity in the data when patients were divided into two groups: under and over 12 years of age.

The presence of PVT in the main trunk and/or branches of the portal vein was evaluated by abdominal Doppler ultrasonography. If PVT was suspected, abdominal computed tomographic angiography was performed to confirm the diagnosis. PVT was defined as the absence of blood flow on Doppler ultrasonography or computed tomography angiography.

Due to the retrospective design of the study and the use of archived data, permission was obtained from individuals who contributed to the archive without the need for patient-informed consent. Ethical approval for the study (Research no.: 2024-387) was granted by the university board.

The data were analyzed using IBM SPSS Statistics version 25.0. Statistical analysis was performed using the appropriate parametric or non-parametric test (t-test or the Mann–Whitney test for comparisons of continuous variables between groups, corrected Chi-squared test or Fisher's exact test for categorical data). For multivariate analysis, the possible factors identified by univariate analysis were further entered into the logistic regression analysis to determine the independent predictors of PVT. Statistical significance was set at $P < 0.05$.

3. Results

A total of 183 patients with portal hypertension were included in this study. Among them, 142 had cirrhosis and 41 had intrahepatic non-cirrhotic portal hypertension. In the cirrhotic group, the mean age at admission was 64.6 ± 6.4 months. The mean follow-up duration was 46.8 ± 45.6 months. Patient characteristics are shown in Table 1.

Table 1
Clinical characteristics of cirrhotic patients.

	Patients with PVT (n = 12)	Patients without PVT (n = 130)	P-values
Age (months, mean±SD)	64.7 ± 74.5	64.6 ± 66	0.995
Male gender, n (%)	4 (33.3)	79 (60.8)	0.065
Etiology of cirrhosis			0.189
PFIC	2 (16.6)	41 (31.5)	
Cryptogenic	1 (8.3)	26 (20)	
Biliary atresia	5 (41.6)	20 (15.3)	
Wilson disease	1 (8.3)	22 (16.9)	
Autoimmune hepatitis	1 (8.3)	11 (8.4)	
Others	2 (16.6)	10 (7.7)	
Child-Pugh score			0.090
Class A, n (%)	2 (16.6)	57 (43.8)	
Class B, n (%)	9 (75)	55 (42.3)	
Class C, n (%)	1 (8.3)	18 (13.8)	
Child-Pugh score, mean±SD	7.9 ± 1.5	7 ± 2.0	0.100
Child-Pugh score > 7, n (%)	10 (83.3)	83 (63.8)	0.066
Albumin (g/dL)	3.2 ± 0.6	3.5 ± 0.8	0.141
Total bilirubin (mg/dL)	12.1 ± 10	6.7 ± 9	0.054
Direct bilirubin (mg/dL)	8.1 ± 6.8	4.2 ± 5.7	**0.030**
AST	192±141	195 ± 199	0958
ALT	96 ± 66	129 ± 156	0.478
INR	1.33 ± 0.31	1.41 ± 0.58	0.645
Platelet (1000s/mm^3)	183 ± 132	193 ± 137	0.812
Splenomegaly, n (%)	12 (100)	115 (89.1)	0.609
Esophageal varices, n (%*)	5 (83.3)	51 (67.1)	0.411
Encephalopathy, n (%)	0 (0)	8 (6.2)	0.374
Liver transplantation, n (%)	8 (66.7)	47 (36.2)	**0.038**

* Calculated from patients who underwent upper gastrointestinal endoscopy.

Forty-one patients with CHF and INCPH were enrolled in this study (CHF = 16, INCPH = 25). The mean age of the patients at presentation was 126 ± 64.2 months. The mean follow-up duration was 23.1 ± 25.6 months. The patient demographics and laboratory findings are presented in Table 2.

PVT was detected in 12 patients (8.5 %) with cirrhosis and in 4 (9.7 %) patients with intrahepatic non-cirrhotic portal hypertension (CHF = 1, INCPH = 3). The prevalence of PVT was not significantly different between patients with cirrhosis and those with intrahepatic noncirrhotic portal hypertension ($p = 0.927$). The most

Table 2
Clinical characteristics of intrahepatic non-cirrhotic portal hypertension patients.

	Patients with PVT (n = 4)	Patients without PVT (n = 37)	P-values
Age (months, mean ± SD)	119.2 ± 84	126.7 ± 63.1	0.828
Male gender, n (%)	3 (75)	22 (59.5)	0.545
Etiology of intrahepatic non-cirrhotic PHT	3 (75)	13 (35.1)	0.120
Congenital hepatic fibrosis	1 (25)	24 (64.9)	
Idiopatic non-cirrhotic portal hypertension			
Hb, g/dL	11.5 ± 3.4	10.8 ± 2	0.573
WBC, (1000 s/mm^3)	7.1 ± 2.7	4.9 ± 2.6	0.113
Platelet, (1000 s/mm^3)	110 ± 54	127 ± 98	0.742
Total bilirubin (mg/dL)	0.8 ± 0.2	0.8 ± 1.4	0.925
Direct bilirubin (mg/dL)	0.2 ± 0.1	0.3 ± 0.7	0.832
AST, (U/L)	56 ± 56	45 ± 26	0.736
ALT, (U/L)	50 ± 45	33 ± 24	0.255
INR	1.2 ± 0.1	1.11 ± 0.1	0.220
Albumin (g/dL)	3.7 ± 0.6	4.2 ± 0.5	0.062
Splenomegaly, n (%)	3 (75)	36 (97.3)	0.188
Esophageal varices, n (%)	3 (75)	34 (91.9)	0.279

common etiologies of cirrhosis were progressive familial intrahepatic cholestasis (30.3 %), cryptogenic (19 %), biliary atresia (17.6 %), Wilson's disease (16.2 %), and autoimmune hepatitis (8.5 %). Biliary atresia was the etiology of cirrhosis in five of the 12 patients with PVT. The development of PVT was significantly higher in patients with biliary atresia than in those with other cirrhosis etiologies ($p = 0.022$). The mean age at diagnosis of cirrhosis by biopsy was 7.5 ± 6.4 months in patients with biliary atresia, 79.1 ± 70.3 months in patients with other than biliary atresia and the difference was significant between these groups ($p < 0.001$). The patients were categorized according to the Child-Pugh classification: 59 patients (41.5 %) were in class A, 64 patients (45 %) in class B, and 19 patients (13.3 %) in class C. Child-Pugh Score was >7 in 80 % of patients with biliary atresia and 53.5 % of patients with other etiologies, and the difference was significant ($p = 0.024$). The serum direct bilirubin levels were higher in the PVT group than in the non-PVT group ($p = 0.015$). There were no differences in total bilirubin, albumin, INR, AST, ALT, and platelet values between the groups. A multivariate logistic backward regression model was built utilizing previously described variables at baseline associated with the presence of PVT. In the final model, no variables were independently associated with PVT ($P = 0.494$). Thirty-eight percent (55/142) of patients underwent liver transplantation. The PVT group required more liver transplantations than the non-PVT group ($p = 0.038$). None of our cirrhotic patients with PVT underwent liver transplantation solely due to portal hypertension and its complications such as variceal bleeding.

4. Discussion

This retrospective study has helped identify the prevalence of PVT in pediatric patients with cirrhosis and intrahepatic noncirrhotic portal hypertension, as well as the complex relationship between PVT development. The study population differed from that of previous studies that investigated adult patients. The present study demonstrated that the prevalence of PVT is 9.7 % in pediatric patients with intrahepatic non-cirrhotic portal hypertension and 8.5 % in pediatric patients with cirrhosis. Notably, patients with biliary atresia are at increased risk of developing PVT.

Several predisposing factors may favor thrombus formation in the portal system, as explained by the Virchow triad: hypercoagulability, reduced flow velocity, and endothelial damage [2]. The progression of liver disease causes a decrease in blood flow and an increase in bacterial endotoxins in the portal and systemic circulation, leading to thrombosis [13,14]. Cirrhosis is strongly associated with coagulopathy. Prolongation of prothrombin time, which is associated with diminished hepatic synthesis of clotting factors, is a good marker of hepatic failure. In contrast, bleeding predisposition is balanced by a deficiency of procoagulants. Chen et al. showed that PVT is associated with greater Child-Pugh score and MELD score [15]. However, in our study there was no significant relationship between the Child-Pugh classification and the prevalence of PVT. On the other hand, even though the difference was not significant, the frequency of PVT in patients with a Child Pugh score >7 was higher than that in those with a score <7. Additionally, we found that serum bilirubin level, a marker of hepatic dysfunction, was higher in patients presenting with PVT than in those without PVT.

It is known that portal flow velocity is decreased in cirrhotic patients, especially in Child-Pugh class C patients, compared to those in classes A and B. Portal blood flow lower than 10 cm/s is an independent risk factor for survival in cirrhotic patients [16]. In a recent study, reduced portal flow velocity was identified as the only factor in multivariate analysis that predicted PVT development. They demonstrated that decreased velocity and stagnation of portal blood flow may be the most important prognostic factors for PVT development [17]. In our study, there was no significant difference in the prevalence of PVT between patients with cirrhosis and those with intrahepatic noncirrhotic portal hypertension. This suggests that even if liver synthetic function is normal, there is a predisposition to PVT due to decreased portal blood flow related to portal hypertension. An increasing number of studies have reported that patients with intrahepatic non-cirrhotic portal hypertension tend to have a relatively high incidence of PVT. The development of PVT in patients with intrahepatic non-cirrhotic portal hypertension remains to be elucidated. As the portal vein becomes dilated and flow velocity decreases under portal hypertension, the slow and disrupted flow may contribute to luminal narrowing or obliteration of small portal venous branches, accompanied by dense deposits of elastic fibers. Thrombophilia, a systemic hypercoagulable state caused by abnormal coagulation and fibrinolysis, plays a dominant role in thrombotic disorders [18,19]. Immunological conditions, exposure to toxins, and genetic disorders are also potential mechanisms in the pathogenesis of the disease. PVT is common in adult patients with INCPH, with a reported prevalence ranging from 13–46 % [6,10,11]. However, there have been no reported studies on childhood. Although the mechanism of the disease suggests a possibly different pathophysiological background in portal hypertension between non-cirrhotic portal hypertension and liver cirrhosis, the results of this study indicate that non-cirrhotic portal hypertension and cirrhosis have similar risks for the development of PVT. The differing pathogeneses between the two groups can probably be attributed to a decrease in liver blood flow and the progression of portal hypertension caused by blocked portal blood flow due to thrombosis in the PVT groups.

Biliary atresia is the most common cause of liver transplantation in children. Although Kasai portoenterostomy is performed, patients may progress to cirrhosis and portal hypertension. The rate of PVT is higher in patients with biliary atresia than in those with other etiologies. Different aspects of the coagulation system have been implicated as being responsible for this phenomenon; for instance, platelet hyperreactivity has been found in many patients with cholestatic liver disease [20,21]. In a previous study, thromboelastography in patients with early stage chronic cholestatic liver disease showed plasmatic hypercoagulability, probably due to higher fibrinogen levels as compared to those found in non-cholestatic liver patients or controls [20]. Ascending cholangitis may allow bacteria to spread into the portal vasculature and damage the endothelium, thereby predisposing to intrahepatic portal vein thrombosis and disseminated intravascular coagulation [22]. There might be a predisposition for PVT after intra-abdominal surgery. However, in a Brazilian cohort, there was no difference in the frequency of PVT between groups with or without Kasai hepatoportoenterostomy prior to liver transplantation [23]. Recent reports on patients with biliary atresia have found that portal vein size, recipient age, and recipient weight at the time of transplantation are associated with an increased risk for the development of PVT [24–26]. Patel et al. confirmed that intimal thickening of the extrahepatic portal vein (called 'obliterative portal venopathy') was observed in explants of patients with biliary atresia and was significantly higher than in the non-biliary atresia group [27]. A recent study showed that only the occurrence of pre-transplant PVT was associated with the development of PVT after liver transplantation [28]. Furthermore, it has been reported that PVT increases morbidity and mortality associated with liver transplantation [29,30]. Thus, during follow-up of these patients, early identification and assessment of PVT evolution potentially contribute to tailoring treatment strategies, improving prognosis, and avoiding the potential risks related to invasive therapy.

It is clear that PVT is associated with liver disease progression [31]. However, currently available data have not been able to identify whether this relationship is causal, or whether the development of PVT is a consequence of advanced hepatic cirrhosis [31,32]. Violi et al. found that age and hepatic failure seem to play prominent roles in the development of PVT [33]. Luca et al. reported thrombotic progression did not correlate with an increase in mortality, episodes of hepatic decompensation, or the need for liver transplantation [34]. We found no relationship between cirrhosis severity and the occurrence of PVT. However, in PVT group, the rate of liver transplantation was significantly higher compared to non-PVT group. Although none of our patients had uncontrolled variceal bleeding in our PVT group, more patients needed liver transplantation because of advanced cirrhosis than in the non-PVT group. These data suggest that PVT may be a major marker of liver disease decompensation rather than a consequence of advanced liver disease.

This study has important limitations, including its retrospective design and incomplete follow-up. Additionally, the infrequency of PVT makes it difficult to analyze risk factors and differences between groups, which increases the risk of statistical errors. Procoagulant and anticoagulant factors were not obtained due to the retrospective design of our study, which was a major limitation that hindered the ability to evaluate the relationship between cirrhosis and PVT. In many studies published in the literature, biliary atresia is identified as the most common cause of liver cirrhosis in the pediatric age group. However, in our study population, while the prevalence of biliary atresia was found to be 17.3 %, the most common cause of cirrhosis was PFIC. This finding can be explained by the still high rate of consanguineous marriages in our country.

In conclusion, the prevalence of PVT was similar in pediatric patients with cirrhosis and intrahepatic non-cirrhotic portal hypertension. Patients with biliary atresia and pre-transplant patients have an increased risk of developing PVT. Therefore, these patients should be routinely evaluated to identify portal vein complications and to detect early warning signs during follow-up.

Conflict of interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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