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Portal Vein Thrombosis in Cirrhotic Patients: Risk Factors Involved

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Abstract

The aim of this study was to investigate the risk factors associated with the occurrence of portal thrombosis in cirrhotic patients at the hepatology and gastroenterology department of Cocody University Hospital. Patients and methods: This was a retrospective analytical study of the unpaired case-control type, lasting 5 years from January 2018 to December 2022, covering 117 files of cirrhotic patients, including 21 cases of portal venous thrombosis (PVT) and 96 witnesses. The risk factors studied were age, sex, Child-Pugh score, ascites, jaundice, edema of the lower limbs, digestive bleeding, hepatic encephalopathy, etiologies of liver cirrhosis, prothrombin rate, transaminases, bilirubin, albumin, endoscopic signs of portal hypertension, propranolol intake, platelets, and hemoglobin. Univariate and multivariate logistic regression analyses were used to identify risk factors associated with the occurrence of PVT. Results: The hospital prevalence of cirrhosis was 12.9%, the average age of patients was 49.2 ± 14.8 years with a male predominance (sex ratio = 1.43), and viral hepatitis B was the main cause (48.7%). Jaundice, portal hypertension, gastropathy, and anemia were the risk factors associated with the occurrence of PVT in cirrhotics in the univariate analysis. After multivariate regression, portal hypertension gastropathy (OR = 4.4; regression coefficient $\beta = 1.59$; CI = 1.6 - 12.4; p = 0.004) was significantly associated with the occurrence of PVT, while anemia was a protective factor (OR = 0.2; regression coefficient $\beta = -1.61$; CI = 0.1 - 0.9; p = 0.03). Conclusion: Patients with portal hypertension gastropathy are more likely to develop portal vein thrombosis during cirrhosis.

Keywords

Portal Venous Thrombosis, Cirrhosis, Risk Factors

1. Introduction

Portal vein thrombosis (PVT) is a total or partial obstruction of the portal vein and/or one of its branches, more or less extending to the splenomestral trunk [1]. In cirrhotic patients, the risk of developing PVT is proportional to the degree of liver function impairment [2]. Based on cross-sectional data from a meta-analysis, Child-Pugh scores B and C, elevated D-dimers, the presence of ascites, and the use of non-selective beta-blockers were associated with the occurrence of PVT in cirrhotic patients [3].

A study conducted by Fatma *et al*. at Habib Thameur Hospital in Tunis in 2022 concluded that taking BBNS (odds ratio = 2; 95% CI: 1.02 - 4.2; p = 0.04) and the presence of grade II or III oesophageal varices (OR = 4; 95% CI: 0.9 - 17.8; p = 0.03) were significantly associated with the occurrence of PVT [4].

To our knowledge, no studies have been conducted in sub-Saharan Africa to determine the risk factors associated with the occurrence of non-tumoural portal vein thrombosis in cirrhotic patients, hence the choice of this topic.

Aim: To study the risk factors associated with the occurrence of portal thrombosis in cirrhotic patients.

2. Patients and methods

We have a minnows Retrospective analytical study of unmatched case-control type, lasting 5 years from January 2018 to December 2022, involving 117 cirrhotic patient files, including 21 cases of portal thrombosis and 96 controls. Included as cases were all records of patients followed for cirrhosis with PVT. Included as controls were all records of patients followed for cirrhosis without PVT. The risk factors studied were age, sex, Child Pugh score, ascites, jaundice, lower limb edema, gastrointestinal bleeding, hepatic encephalopathy, etiologies of cirrhosis, prothrombin rate, transaminases, bilirubin, albumin, endoscopic signs of portal hypertension, propranolol intake, platelets, and hemoglobin. Univariate and multivariate logistic regression analyses were used to identify risk factors associated with the occurrence of PVT.

3. Results

The hospital prevalence of cirrhosis was 12.9%. The mean age of patients was 49.2 years, with a male predominance (sex ratio = 1.43). Viral hepatitis B was the main etiology (48.7%). There was no significant relationship between these risk factors, including age, sex, ascites, lower limb edema (**Table 1**), digestive hemorrhage, thrombocytopenia, hepatic cytolysis (**Table 2**), Child Pugh score (**Table 3**), propranolol intake (**Table 4**), and the occurrence of PVT. Jaundice, portal hypertensive gastropathy, and anemia were the risk factors associated with the occurrence of PVT in cirrhotic patients in univariate analysis. After multivariate regression, only portal hypertensive gastropathy (OR = 4.4, regression coefficient $\beta = 1.59$; CI = 1.6 - 12.4; p = 0.004) was significantly associated with the occurrence of PVT, while anemia was a protective factor (OR = 0.2; regression coefficient $\beta = -1.61$;

CI = 0.1 - 0.9; p = 0.03) (Table 5).

Table 1. Relationship between signs of decompensation of cirrhosis and the occurrence of PVT.

	PVT			
Signs of decompensation	**Yes (N = 21) *n* (%)**	**No (N = 96) *n* (%)**	**OR (95% CI)**	**p-value**
Ascites	19 (19.6)	78 (80.4)	2.1 (0.5 - 10.3)	0.52
lower limb edema	17 (20.0)	68 (80.0)	1.7 (0.5 - 5.7)	0.43
jaundice	10 (29.4)	24 (70.6)	2.7 (1.1 - 7.2)	**0.**04

OR: Odds Ratio; CI: Confidence Interval; PVT: Portal Vein Thrombosis.

Table 2. Relationship between biology and the occurrence of PVT.

	PVT		
Biology	**Yes (N = 21) Median (Min-Max)**	**No (N = 96) Median (Min-Max)**	**p-value**
Low TP	65 (21.9 - 100)	60.0 (20.0 - 100.0)	0.71
High AST	78.0 (18.0 - 394.0)	57.0 (10 - 970.0)	0.79
High ALT	43.0 (7.0 - 165.0)	37.0 (9.0 - 855.0)	0.30
Hypoalbuminemia	25.5 (15.2 - 58.8)	22.2 (2 - 46.1)	0.73
Hyperbilirubinemia	21.8 (7.0 - 119.0)	12.6 (1.9 - 132.0)	0.08
Anemia	10.7 (7.0 - 13.3)	9.8 (3.9 - 15.3)	0.04
Thrombocytopenia	78.0 (27 - 446)	87.5 (9 - 609)	0.38

ASAT: Aspartate Aminotransferase, ALT: Alamine Amino Transferase, TP: Prothrombin rate.

Table 3. Relationship between the severity of cirrhosis and the occurrence of PVT.

	PVT			
Child-Pugh	**Yes (N = 21) n (%)**	**No (N = 96) n (%)**	**OR (95% CI)**	**p-value**
A	2 (14.3)	12 (85.7)	0.7 (0.2 - 3.6)	1.00
B	14 (15.7)	75 (84.3)	0.6 (0.2 - 1.6)	0.27
C	5 (35.7)	9 (64.3)	3.0 (0.9 - 10.2)	0.07

OR: Odds Ratio; CI: Confidence Interval; PVT: Portal Vein Thrombosis.

Table 4. Relationship between propranolol intake and the occurrence of PVT.

	PVT			
propranolol intake	**Yes (N = 21) *n* (%)**	**No (N = 96) *n* (%)**	**OR (95% CI)**	**p-value**
Yes	18 (19.8)	73 (80.2)	1.9 (0.5 - 6.9)	0.40
No	3 (11.5)	23 (88.5)		

Table 5. Multivariate analysis in search of factors independently associated with PVT.

	PVT					
Factors	**Yes (N = 21) n (%)**	**No (N = 96) n (%)**	**β**	**SE**	**OR (95% CI)**	**p-value**
Jaundice	10 (29.4)	24 (70.6)	1.02	0.522	2.8 (0.9 - 7.6)	0.052
HTP gastropathy	14 (31.8)	30 (68.2)	1.59	0.523	4.4(1.6 - 12.4)	0.004
Anemia	4 (8.7)	42 (91.3)	-1.61	0.49	0.2 (0.1 - 0.9)	0.03

OR: Odds ratio; CI: Confidence Interval; HTP: Portal Hypertension; PVT: Portal Vein thrombosis; SE: Standard Error; β: Regression Coefficient.

4. Discussion

In our study, among the signs of decompensation of cirrhosis, only jaundice was significantly related to the occurrence of PVT (OR = 2.7; CI = 1.1 - 7.2; p = 0.04). After multivariate logistic regression, there was no significant association (OR = 2.8; regression coefficient β = 1.01; CI = 0.9 - 7.6; p = 0.052). On the other hand, Cagin *et al.* [5] in 2019 had found in their series a significant link between jaundice and the occurrence of PVT (β = 1.6 and p = 0.001). Anemia was a protective factor in our series against the occurrence of PVT (OR = 0.2; regression coefficient β = −1.61; CI = 0.1 - 0.9; p = 0.03). On the contrary, Lopez-Gomez *et al.* [6] in Spain in 2021 found a significant relationship between anemia and the occurrence of portal thrombosis with a p value = 0.01. We did not find a significant link between thrombocytopenia and PVT (p = 0.38). This result was similar to that of Fatma *et al.* [4] in 2022 in Tunisia (p = 0.08). On the other hand, Reyes *et al.* [7] (OR = 3.6; p = 0.04) and Giannitrapani *et al.* [2] (p < 0.03) found a significant link between thrombocytopenia and the occurrence of PVT. In the literature, a significant association is generally found between thrombocytopenia and the occurrence of PVT in cirrhotic patients, as they have portal hypertension in common [8] [9]. In our study, we did not find a relationship between the severity of cirrhosis assessed by the Child Pugh score (p = 1.00) and the occurrence of PVT, which was consistent with the study of Fatma *et al.* [4] 2022 in Tunisia (p = 0.9). However, in the literature, it is noted that in cirrhotic patients, the risk of developing PVT is proportional to the degree of impairment of liver function [2]. There was no relationship between hepatic cytolysis and the occurrence of PVT (ALT: p = 0.79, AST: p = 0.30). This observation was also found by Cagin *et al.* [5] (ALT: p > 0.05 and AST: p > 0.05). Propranolol intake was not a factor associated with the occurrence of PVT in our study (OR = 1.9 and p = 0.40). This was consistent with the results of the studies by Odriozola *et al.* [8] in 2022 in Spain, Xu *et al.* [10] in 2020 and Xie *et al.* [11] in 2020 in Shanghai, China, who found a p value > 0.05. On the other hand, Fatma *et al.* in 2022 found a significant relationship between taking non-cardioselective beta blockers and the occurrence of PVT (OR = 2; CI: 1.02 - 4.2; p = 0.04) [4]. Indeed, it is described in the literature that BBNS reduce portal hypertension by reducing portal blood flow, thus leading to the development of PVT [9]-[12]. In our series we found a significant link between PH gastropathy

and the occurrence of PVT (OR = 4.4; regression coefficient $\beta = 1.59$; CI = 1.6 - 12.4; p = 0.004) related to decreased portal blood flow portal hypertension. This was consistent with the data from the study by Nadinskai *et al*. [13] in 2019 in Russia, which found a value of p = 0.005.

5. Conclusions

This study shows that patients with portal hypertension gastropathy are more likely to develop portal vein thrombosis during cirrhosis.

Contrary to previous studies, anaemia is a protective factor. A prospective, multicentre study with a larger sample size is needed to confirm or refute these preliminary results.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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