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RETROSPECTIVE ANALYSIS OF PORTAL VEIN THROMBOSIS IN PATIENTS WITH LIVER DISEASE

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RETROSPECTIVE ANALYSIS OF PORTAL VEIN THROMBOSIS IN PATIENTS WITH LIVER DISEASE (Abstract): Background: Portal vein thrombosis (PVT) is a hepatic vascular disorder characterized by the presence of a thrombus with the portal vein trunk or its intrahepatic branches. The most frequent cause of PVT is chronic liver disease, in particular liver cirrhosis and hepatocellular carcinoma, both of which significantly impact morbidity and mortality. Evaluating clinical, etiological and hematological factors together with thrombus localization may improve understanding of disease progression and patient management. **Materials and methods:** A retrospective observational study was conducted on patients diagnosed with portal vein thrombosis and hospitalized at a tertiary care center in Bucharest between 2016 and 2025.Statistical analysis was performed using IBM *SPSS Statistics 25* and *Microsoft Office* tools. Quantitative variables were assessed for normality using the Shapiro-Wilk test and reported as means ± standard deviations or medians with interpercentile ranges. Categorical variables were expressed as absolute values and percentages. Group differences for qualitative variables were analyzed using Fisher's exact test, while Z-tests with Bonferroni correction were applied for post hoc analysis. Statistical significance was set at $\alpha = 0.05$. **Results:** The study cohort had a mean age of 64.95 ± 11.56 years and was predominantly male (72.2%). Most common liver diseases were alcoholic cirrhosis, hepatitis C-related cirrhosis and hepatitis B-related cirrhosis; HCC was present in 39.6% of patients. Chronic thrombosis with partial vascular obstruction was most frequent. Complications of portal hypertension-including ascites, esophageal varices and splenomegaly-were frequently observed. Statistically significant associations were found between splenomegaly and thrombocytopenia, as well as between portal vein thrombosis and hepatocellular carcinoma. **Conclusions:** Portal vein thrombosis occurs predominantly in patients with advanced liver disease. **Keywords**: PORTAL VEIN THROMBOSIS, LIVER CIRRHOSIS, HEPATOCELLULAR CARCINOMA, PORTAL HYPERTENSION, THROMBOCYTOPENIA, THROMBUS LOCALIZATION.

INTRODUCTION

Chronic liver disease represents a major global health burden, characterized by progressive hepatic dysfunction and a wide spectrum of complications that significantly impact morbidity and mortality. Among these, vascular complications play a central role, particularly portal vein thrombosis (PVT), which is increasingly recognized as a frequent and clinically relevant condition in patients with advanced liver disease (1-3). Portal vein thrombosis (PVT) is a vas-

cular disorder defined by partial or complete obstruction of the portal vein lumen by a thrombus. It occurs most commonly in patients with liver cirrhosis, with a reported prevalence of 5-26% and in those with hepatocellular carcinoma (HCC), both conditions associated with reduced hepatic reserve and poor prognosis (1,4,5). Less frequently, PVT may develop in the context of local or systemic inflammation, myeloproliferative disorders, thrombophilia or sepsis. The thrombotic process may involve the main portal vein or extend to other vessels within the portal venous system, such as the splenic or mesenteric veins. The pathogenesis of PVT is multifactorial and is commonly explained by Virchow's triad, which includes reduced portal blood flow, endothelial injury and hypercoagulability (6). In patients with liver cirrhosis, portal hypertension and decreased portal venous velocity contribute significantly to a prothrombotic state (7). In addition, systemic conditions such as inflammatory diseases, infections, inherited or acquired thrombophilia and myeloproliferative disorders further increase the risk of thrombosis (8,9). Liver cirrhosis ranks as the 11th leading cause of death worldwide, with mortality increasing over the past decade and accounting for approximately 2.4% of total global deaths (10). The one-year cumulative mortality reaches up to 40% in patients with decompensated cirrhosis (11). Moreover, cirrhosis remains one of the main predisposing factors for PVT due to vascular architectural distortion, reduced portal flow and imbalances between procoagulant and anticoagulant mechanisms (12). The occurrence of PVT in cirrhotic patients is associated with a higher incidence of hepatic decompensation and consequently, with a worse prognosis (4). Additionally, hepatocellular carcinoma (HCC) represents another major condition associated with PVT. HCC is the sixth most commonly diagnosed cancer worldwide and the second leading cause of cancer-related death (13). The presence of PVT in patients with HCC reflects an advanced stage of disease and is associated with poor prognosis, with a prevalence ranging from 25% to 50% (5, 14, 15). Moreover, the coexistence of PVT and HCC limits therapeutic options and may lead to severe complications, including portal hypertension, ascites, metastasis and life-threatening gastrointestinal bleeding (16). The coexistence of malignancy and cirrhosis further complicates the pathophysiological landscape, making it difficult to distinguish between tumor-related and non-tumoral thrombosis, as well as to identify the factors influencing thrombus localization and extension (16,17). Despite growing evidence on the clinical significance of PVT, several aspects remain insufficiently characterized. In particular, the relationship between thrombus localization and the underlying etiology of liver disease, as well as its association with hematological parameters and clinical complications, remains incompletely understood (2,18). Furthermore, potential differences related to patient characteristics, including sex-related variations and comorbidity profiles, warrant further investigation (3, 19). Therefore, the aim of this study is to evaluate the clinical, etiological and hematological characteristics of patients with PVT and to investigate their association with thrombus localization and related complications. This study integrates multiple clinical and biological parameters to provide a more comprehensive understanding of PVT in the context

of chronic liver disease, with potential implications for improved risk stratification and therapeutic management.

MATERIALS AND METHODS

Study design and population

This retrospective observational study included patients diagnosed with portal vein thrombosis (PVT) who were admitted to a tertiary care center in Bucharest, between January 2016 and December 2025. The diagnosis of PVT was established using imaging techniques, including abdominal ultrasound, computed tomography (CT) and magnetic resonance imaging (MRI). All adult patients (≥18 years) with a confirmed diagnosis of PVT were eligible for inclusion. Patients with incomplete medical records or insufficient diagnostic data were excluded from the analysis. Clinical, demographic, etiological and hematological data were collected from hospital medical records and electronic databases.

Data collection

For each patient, demographic characteristics such as age and sex were recorded. Clinical variables included the presence of liver cirrhosis of various etiologies, hepatocellular carcinoma, pancreatitis, as well as other digestive and extra-digestive malignancies. Complications related to portal hypertension-including ascites, splenomegaly and esophageal varices-were also documented. Etiological factors underlying liver disease were categorized as viral hepatitis (HBV, HBV+HDV coinfection, and HCV) and alcohol-related liver disease. Hematological assessment focused on platelet count and patients were classified into three categories: thrombocytopenia, normal platelet count and thrombocytosis, according to standard laboratory reference ranges. Characteristics of portal vein thrombosis were evaluated in terms of duration, degree of vascular obstruction and anatomical localization. Thrombosis was classified as acute or chronic based on clinical and imaging findings. The extent of vascular obstruction was categorized as either partial or complete. In addition, the anatomical localization of the thrombus within the portal venous system was recorded, including involvement of the main portal vein, the splenic vein or the presence of thrombi affecting multiple venous segments simultaneously.

Statistical Analysis

Statistical analysis was performed using IBM SPSS Statistics version 25 (IBM Corp., Armonk, NY, USA) and Microsoft Office (Excel and Word, 2024). Quantitative variables were assessed for normality using the Shapiro-Wilk test and were expressed as mean ± standard deviation for normally distributed data or median with interquartile range (IQR) for non-normally distributed data. Categorical variables were presented as absolute values and percentages. Differences between independent groups for categorical variables were analyzed using Fisher's exact test. Post hoc comparisons for contingency tables were performed using Z-tests with Bonferroni correction. A p-value < 0.05 was considered statistically significant.

Institutional Review Board Statement. The study was conducted in accordance with the Declaration of Helsinki. The study protocol was approved by the Ethics Committee of "St John" Clinical Emergency Hospital No. R4123 on 31.03.2026, due to the retrospective nature of the study.

Informed Consent Statement. Written informed consent for participation and for the publication of these details was obtained from all subjects involved in the

study, as documented in the hospital admission form.

RESULTS

Demographic and clinical characteristics of the study population

A total of 252 patients diagnosed with portal vein thrombosis, admitted to St John Emergency Hospital between 2016 and 2025 were included in this retrospective study. Due to incomplete medical records, the number of observations (N) varied across analyses. The mean age of the study population was 64.95 ± 11.56 years, with a median of 65 years (IQR 58-73). The cohort was predominantly male (72.2%). The distribution of hospital admissions showed a peak in 2024 (15.5%), followed by 2017 (13.9%) and 2023 (11.5%), while the lowest number of cases was recorded in 2021 (2%). Regarding underlying liver disease, alcoholic cirrhosis was the most frequent etiology, present in 31.3% of patients. Viral-related cirrhosis was also common, including HCV-related cirrhosis (22.5%), HBV-related cirrhosis (15.8%) and HBV-HDV coinfection (3.3%). Hepatocellular carcinoma (HCC) was identified in 39.6% of patients. Other digestive malignancies were present in 26.9% of cases, while extra-digestive neoplasms were observed in 14.9%. Pancreatitis was documented in 10% of patients (tab. I).

TABLE I.

Demographic and clinical characteristics of the study population

Parameter	Value
Year of hospitalization (No., %)	
2016	25 (9.9%)
2017	35 (13.9%)
2018	28 (11.1%)
2019	24 (9.5%)
2020	21 (8.3%)
2021	5 (2%)
2022	20 (7.9%)
2023	29 (11.5%)
2024	39 (15.5%)
2025	26 (10.3%)
Age(Mean ± SD) (Median (IQR))	64.95 ± 11.56, 65 (58-73)
Sex (Male) (No., %)	182 (72.2%)
Comorbidities	(No., %)
Liver cirrhosis - HBV(N=240)	38 (15.8%)
Liver cirrhosis - HBV+HDV (N=240)	8 (3.3%)
Liver cirrhosis - HCV (N=240)	54 (22.5%)
Alcoholic cirrhosis (N=240)	75 (31.3%)
Hepatocellular carcinoma (N=240)	95 (39.6%)
Pancreatitis (N=241)	24 (10%)
Other digestive neoplasms (N=242)	65 (26.9%)
Metastasis	42 (16.7%)
Other extra - digestive neoplasms (N=241)	36 (14.9%)
Other causes of thrombosis (N=243)	38 (15.6%)

Hepatocellular carcinoma and type of liver cirrhosis

Among patients with hepatocellular carcinoma, viral etiologies were predominant. HCV-related cirrhosis was the most frequent condition (45.7%), followed by HBV-related cirrhosis (26.1%) and HBV-HDV coinfection (5.4%). Overall, viral cirrhosis accounted for 73.9% of HCC cases. Alcohol-related cirrhosis was also frequently observed and occurred in 38% of patients with HCC. These findings emphasize the strong association between chronic liver disease and the development of hepatocellular carcinoma (tab. II).

TABLE II.

Distribution of patients with hepatocellular carcinoma by type of cirrhosis

Cirrhosis type - HCC (N=92)	No.	Percent (Total)
HBV	24	26.1%
HBV+HDV	5	5.4%
HCV	42	45.7%
Viral cirrhosis	68	73.9%
Alcohol - related cirrhosis	35	38%

Hematologic profile

Evaluation of hematological parameters showed that 50.9% of patients had normal platelet counts, while 43.9% presented thrombocytopenia and 5.2% thrombocytosis. Thrombocytopenia was frequently associated with splenomegaly and portal hypertension, common complications of advanced liver disease (tab. III).

TABLE III.

Hematologic profile of the study population

Hematologic profile (No., %) (N=212)	Value
Thrombocytopenia	93 (43.9%)
Normal platelets	108 (50.9%)
Thrombocytosis	11 (5.2%)

Characteristics of portal vein thrombosis

Most patients presented chronic thrombosis (98.4%), whereas only 1.6% had acute PVT. Partial thrombosis was more frequent (63%) compared with complete thrombosis (37%). Complications related to portal hypertension were highly prevalent: ascites was present in 77.8% of patients, portal hypertension in 77.1%, esophageal varices in 64.5% and splenomegaly in 58.9%. The overall in-hospital mortality rate was 31.7% (tab. IV).

TABLE IV.

Characteristics of portal vein thrombosis in the study population

Duration of thrombosis (No., %) (N=62)	Value
Acute	1 (1.6%)
Chronic	61 (98.4%)
Type of thrombosis (No., %) (N=54)	
Partial	34 (63%)
Total	20 (37%)
Portal hypertension HTP (No., %) (N=188)	145 (77.1%)
Ascites (No., %) (N=221)	172 (77.8%)
Varices (No., %) (N=183)	118 (64.5%)
Splenomegaly (No., %) (N=168)	99 (58.9%)
Mortality (No., %)	80 (31.7%)

Additional etiologies associated with portal vein thrombosis

Additional etiological factors were identified in 38 patients (15.6%). The most frequent conditions included digestive sepsis (15.8%), hereditary thrombophilia (13.1%), angiocholitis (5.3%), polycythemia vera (5.3%) and SARS-CoV-2 infection (5.3%). Less frequent factors included metabolic conditions such as obesity and postoperative states (e.g., sleeve gastrectomy) (tab. V).

TABLE V.

Distribution of patients with additional or miscellaneous causes of portal vein thrombosis

Other causes	No	%
Angiocholitis	2	5.3%
Bronchopneumonia	2	5.3%
Cirrhosis of unspecified etiology	6	15.8%
Primary sclerosing cholangitis	1	2.6%
Cholecystitis	1	2.6%
Antithrombin III deficiency	1	2.6%
Gastric sleeve	1	2.6%
Hemochromatosis	1	2.6%
Hepatitis C	2	2.6%
Previous hiatal hernia surgery	1	2.6%
Thrombophilia	5	13.1%
Obesity	1	2.6%
Polycythemia vera	2	5.3%
SARS COV 2	2	5.3%
Budd-Chiari syndrome	1	2.6%
Myeloproliferative disorder	1	2.6%
Bilateral breast sectorectomy	1	2.6%
Intra-abdominal sepsis	6	15.8%
Pulmonary sepsis	1	2.6%

Association between sex and liver disease etiology

A statistically significant association was observed between sex and alcohol-related cirrhosis, which was more frequent in male patients compared to females (37.1% *vs*. 15.4%, p = 0.001) (fig. 1).

Regarding viral liver disease, hepatitis B-related cirrhosis was significantly more frequent in men (19.4 % versus 6.2 %, p=0.016) (fig. 2).

HCV-related cirrhosis was significantly more prevalent among female patients (41.5% *vs*. 15.4%, p < 0.001) (fig. 3).

Platelet count and splenomegaly

A significant association was identified between splenomegaly and thrombocytopenia. Patients with thrombocytopenia presented splenomegaly more frequently compared with those without thrombocytopenia (62.8% *vs*. 27.7%, p < 0.001). Conversely, patients with normal platelet counts were less likely to present splenomegaly (63.8% *vs*. 34.9%) (fig. 4).

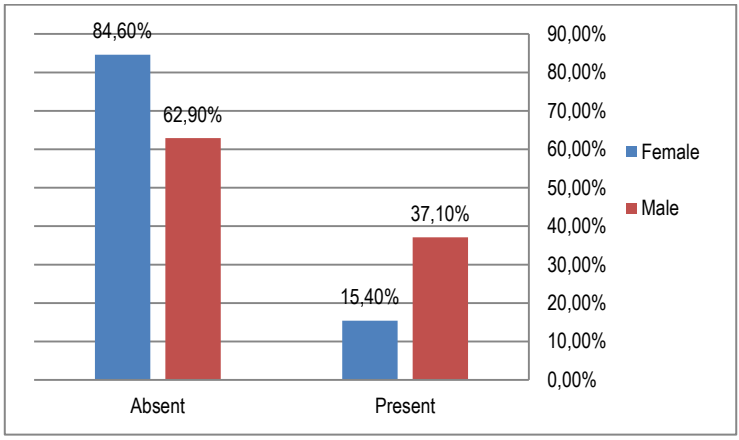

Fig. 1. Distribution of patients by sex and the alcoholic cirrhosis status

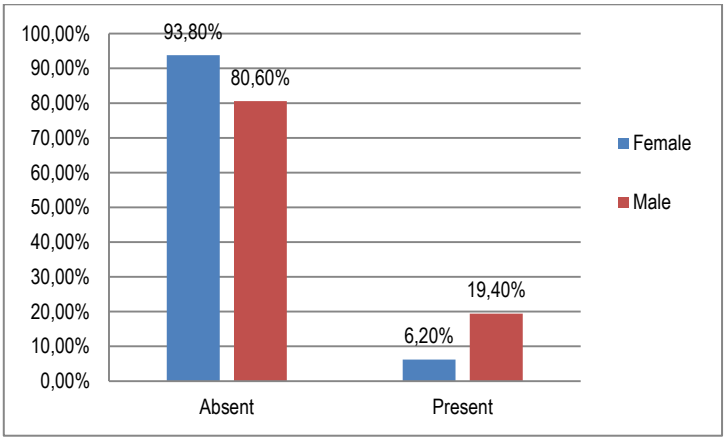

Fig. 2. Distribution of patients by sex and HBV-related cirrhosis

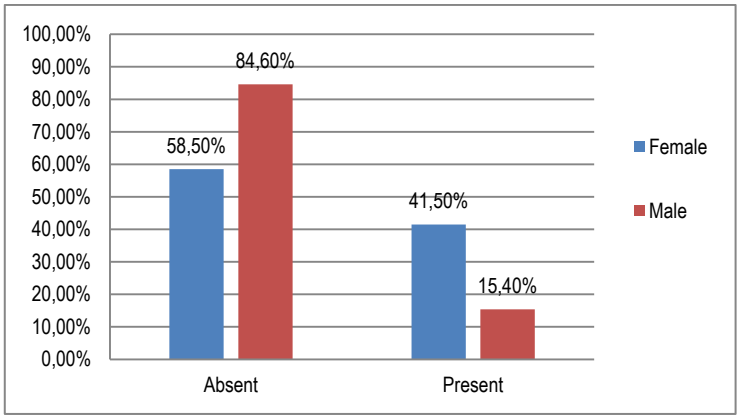

Fig. 3. Distribution of patients by sex and HCV-related cirrhosis

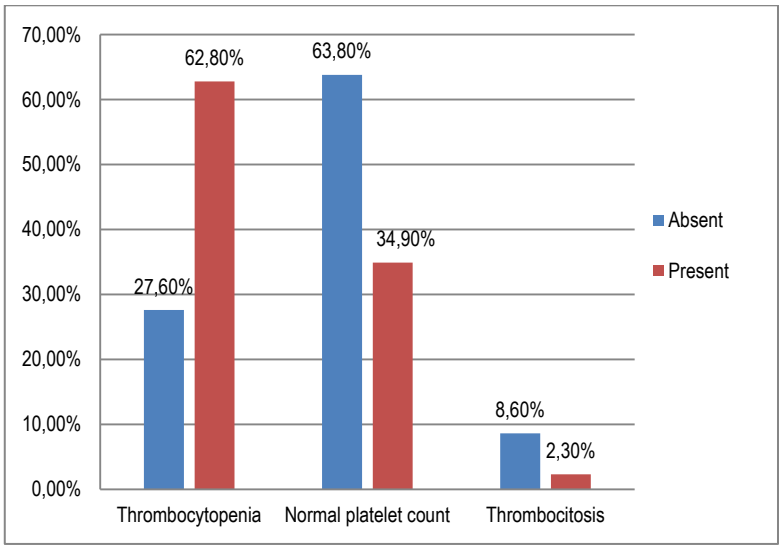

Fig. 4. Distribution of patients by splenomegaly status and platelet count

Among patients with alcoholic cirrhosis, thrombocytopenia was also significantly associated with splenomegaly (68.6% *vs.* 33.3%, p = 0.020) (fig. 5).

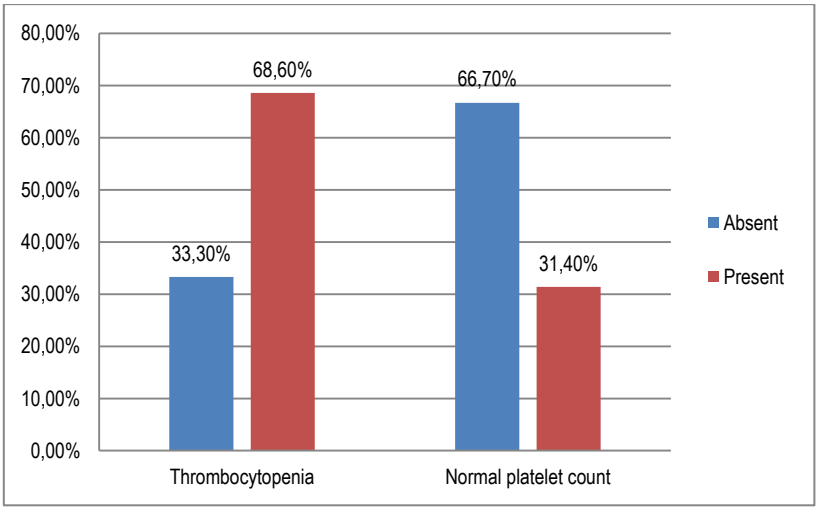

Fig. 5. Distribution of patients with alcoholic cirrhosis by splenomegaly status and hematologic profile

Thrombus localization

The portal vein was the most frequent site of thrombosis. A statistically significant association was identified between HBV-related cirrhosis and thrombus localization (p = 0.010), with all patients in this subgroup presenting portal vein thrombosis (fig. 6).

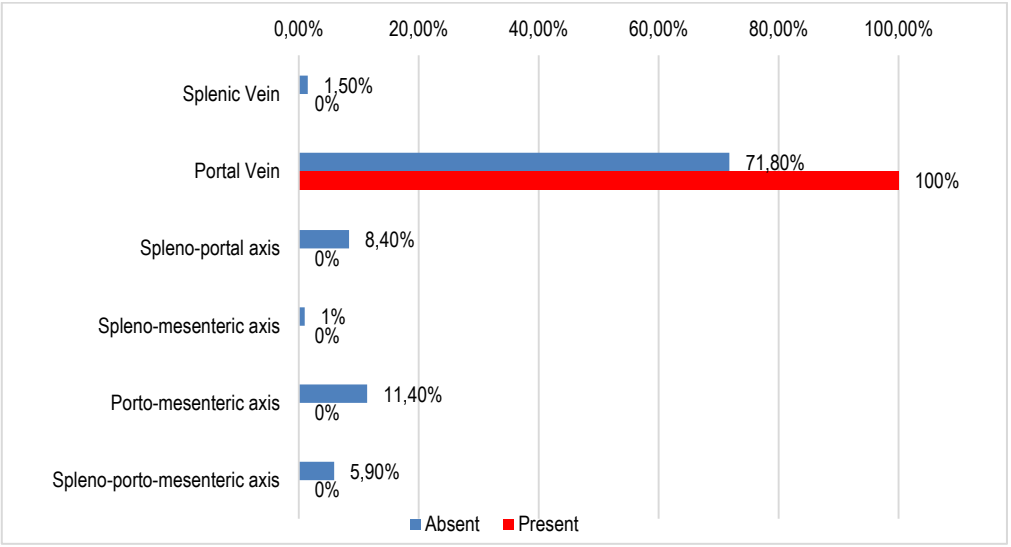

Fig. 6. Thrombus localization in patients with HBV-related cirrhosis

Hepatocellular carcinoma and thrombus localization

A significant association was observed between hepatocellular carcinoma and thrombus localization. Patients with HCC more frequently presented portal vein thrombosis compared with those without HCC (87.1% *vs*. 69.4%, p = 0.036) (fig. 7).

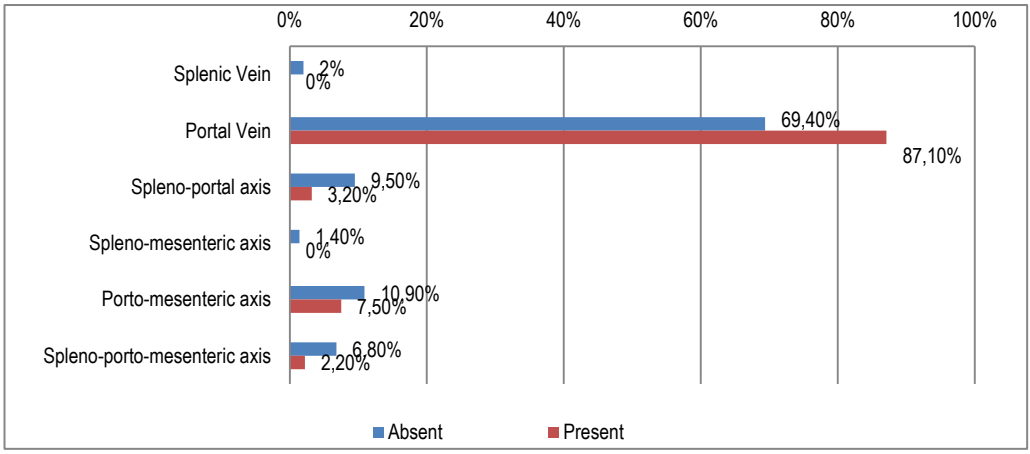

Fig. 7. Thrombus localization in patients with hepatocellular carcinoma

DISCUSSION

Principal Findings

In this retrospective study, we evaluated the clinical, etiological and hematological characteristics of patients with portal vein thrombosis in the setting of liver disease. The main findings indicate that PVT occurs predominantly in older male patients with advanced chronic liver disease, most frequently associated with alcoholic and viral

cirrhosis. Our findings align with previous studies reporting a higher prevalence of PVT in older male patients with chronic liver disease, particularly in advanced cirrhosis and portal hypertension. Several epidemiological analyses have shown a pooled PVT prevalence of approximately 13.9% in cirrhotic patients, increasing with disease severity and age and frequently associated with complications such as hepatocellular carcinoma and impaired portal flow (20). In a large retrospective cohort of patients with decompensated cirrhosis, PVT prevalence was also substantial (~9.4%), highlighting that disease progression relates to higher PVT rates (21). The study we conducted showed a high prevalence of hepatocellular carcinoma, with most cases presenting chronic and partially occlusive thrombosis (5,14). In addition, significant associations were identified between splenomegaly and thrombocytopenia, as well as between hepatocellular carcinoma and thrombus localization.

Etiology of liver disease and PVT. Our results confirm that liver cirrhosis remains the principal underlying condition associated with PVT, most commonly of alcohol-related or viral etiology. The predominance of alcoholic cirrhosis in male patients is consistent with previously reported epidemiological patterns and may reflect differences in alcohol consumption behavior and disease progression (22). Sex-specific reviews demonstrate that alcohol-associated liver disease has been historically more common in men compared to women, likely due to higher alcohol intake and longer exposure in male populations (23). Interestingly, viral cirrhosis-particularly hepatitis C-was strongly associated with hepatocellular carcinoma, accounting for the majority of HCC cases in our cohort. This finding is consistent with data from the literature emphasizing the oncogenic potential of chronic viral hepatitis, which identifies chronic HBV and HCV infection as major risk factors for hepatocellular carcinoma and outlines molecular mechanisms of viral hepatocarcinogenesis (24,25). The coexistence of cirrhosis and malignancy likely enhances the prothrombotic state, contributing to the high prevalence of PVT observed in these patients (14,26).

Hematological profile and portal hypertension. A substantial proportion of patients presented thrombocytopenia, which was significantly associated with splenomegaly. This finding reflects the well-established mechanism of hypersplenism in the context of portal hypertension, where splenic sequestration of platelets leads to decreased circulating platelet levels (7, 27). Elevated D-dimer levels have also been proposed as a potential biomarker for ongoing thrombotic activity in advanced cirrhosis, although their clinical utility remains under investigation (28). Moreover, the high prevalence of portal hypertension-related complications including ascites, esophageal varices, and splenomegaly-supports the concept that PVT is frequently a marker of advanced hepatic decompensation (9,12). These results highlight the complex interplay between hemodynamic alterations and hematological abnormalities in patients with chronic liver disease (29).

Characteristics and localization of thrombosis. The predominance of chronic and partially occlusive thrombosis observed in our study suggests that PVT often develops insidiously and may remain clinically silent until advanced stages, which may explain the high proportion of patients diagnosed during hospitalization for com-

plications of liver disease. We identified a significant association between hepatitis B-related cirrhosis and thrombus localization, with all patients in this subgroup presenting portal vein involvement.

Although the underlying mechanism remains unclear, this may reflect disease-specific vascular changes or differences in the progression of portal hypertension. Furthermore, hepatocellular carcinoma was significantly associated with portal vein thrombosis localization, consistent with prior reports on tumor-related thrombus (15,17,30). Tumor-related thrombosis is typically linked to more aggressive disease and poorer prognosis. In our cohort, PVT predominantly involved the portal vein trunk rather than extending into the mesenteric veins. While mesenteric involvement was less frequent, progression of thrombosis can result in small bowel ischemia and in severe cases, intestinal infarction. These findings are consistent with previous reports emphasizing the prognostic and clinical relevance of thrombus distribution in cirrhotic patients (29,30). Given the complex biological behavior of liver-related malignancies, immunohistochemistry may provide additional insights into tumor biology and may help differentiate tumor-related from non-tumoral thrombosis. Its value in tumor characterization has also been demonstrated in other malignancies, such as sporadic colorectal cancer (31).

Additional Risk Factors. Beyond liver-related etiologies, a subset of patients presented additional prothrombotic conditions, including sepsis, hereditary thrombophilia, myelo- proliferative disorders and recent infections such as SARS-CoV-2. These findings support the multifactorial nature of PVT and the relevance of systemic contributors to thrombus formation. Although less frequent, metabolic conditions and postoperative states were also identified, suggesting that clinicians should maintain a broad differential when evaluating patients with PVT.

Clinical Implications. The findings of this study have several important clinical implications. First, the strong association between PVT and advanced liver disease underscores the need for careful screening in high-risk populations, particularly in patients with cirrhosis and hepatocellular carcinoma. Second, the association between thrombocytopenia and splenomegaly may serve as an indirect marker of portal hypertension severity and could aid in risk stratification. It has been described in prior literature that splenomegaly, resulting from portal hypertension, leads to platelet sequestration and destruction, contributing to thrombocytopenia (9, 32). This relationship not only reflects the severity of portal hypertension but also has prognostic implications, as patients with more pronounced hypersplenism are at higher risk of bleeding complications and may require closer clinical monitoring or earlier intervention. Finally, the high in-hospital mortality rate observed highlights the severity of this condition and the importance of early diagnosis and multidisciplinary management.

Strengths and Limitations. This study has several strengths, including a relatively large sample size and the comprehensive evaluation of clinical, etiological and hematological parameters in a real-world cohort. However, several limitations should be acknowledged. First, the retrospective design may introduce selection and information bias. Second, incomplete data for some variables may have influenced the robustness of certain analyses. Third, the study was conducted in a single tertiary center, which may limit the generalizability of the findings. Additionally, the lack of

differentiation between tumor and non-tumor thrombosis represents an important limitation.

CONCLUSIONS

Portal vein thrombosis occurs predominantly in patients with advanced liver disease, particularly those with cirrhosis and hepatocellular carcinoma. It is frequently associated with portal hypertension-related complications such as splenomegaly, ascites, and varices. Recognition of clinical, etiological and hematological factors-including thrombus localization-can aid in risk stratification and inform management strategies. Early detection of thrombosis, especially when limited to the portal vein trunk, is essential to prevent progression to mesenteric involvement and potentially life-threatening intestinal ischemia.

CONFLICT OF INTEREST AND FUNDING

The authors declare that there is no conflict of interest, and they received no specific funding regarding this scientific research.

ABBREVIATIONS

The following abbreviations are used in this manuscript: PVT (Portal vein thrombosis), HCC (Hepatocellular carcinoma), HBV (Hepatitis B virus), HDV (Hepatitis D virus), HCV (Hepatitis C virus).

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