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Research Article

A retrospective study in patients with portal vein thrombosis

Portal ven trombozu olan hastalarda retrospektif bir çalışma

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Abstract

Aim: To investigate into the age, gender as well as the etiology of portal vein thrombosis in patients diagnosed with the illness.

Material and methods: The medical records of patients with portal vein thrombus (PVT), who referred to the internal medicine and gastroenterology policlinic at Dursun Odabaş Education and Research Hospital at the Faculty of Medicine in Yuzuncu Yil University between 01.01.2011 and 31.12.2013 were retrospectively analyzed.

Results: As a result of the screening within the context of various parameters, a mild dominance of males was observed (M/F = 39/31), The average diagnosis of the illness in males was found to be 47 within the context of the age; this score was 41 in females. More than one thrombophilic factors were determined in 26 patients and there was a known history of liver cirrhosis in 15 patients, as well as myeloprolifirative disease found in 16 patients. Besides these, 13 of the patients were diagnosed to have idiopathic PVT.

Conclusion: PVT should be taken into consideration in the middle and later age patients in the differential diagnosis of abdominal pain, and any underlying disease like liver cirrhosis or thrombophilia should certainly be investigated.

Keywords: Portal vein thrombosis, etiyology, age,gender

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Öz

Amaç: Bu çalışmada, portal ven trombozu tanısı almış hastalarda yaş, cinsiyet ve etiyoloji açısından değerlendirme yapılması amaçlanmıştır.

Gereç ve Yöntem: Yüzüncü Yıl Üniversitesi Tıp Fakültesi Dursun Odabaş Eğitim ve Araştırma Hastanesi İç Hastalıkları ve Gastroenteroloji polikliniğine 01.01.2011 ile 31.12.2013 tarihleri arasında başvuran portal ven trombozu (PVT) olan hastaların tıbbi kayıtları retrospektif olarak analiz edilmiştir.

Bulgular: Çeşitli parametreler çerçevesinde yapılan tarama sonucunda, erkeklerde hafif bir baskınlık olduğu gözlemlenmiştir (E/K=39/31). Hastalığın tanı aldığı yaş ortalaması erkeklerde 47, kadınlarda ise 41 olarak bulunmuştur. Yirmi altı hastada birden fazla trombofilik faktör tespit edilmiş, 15 hastada karaciğer sirozu öyküsü mevcutken, 16 hastada ise miyeloproliferatif hastalık saptanmıştır. Bunların yanı sıra, 13 hastada idiopatik PVT tanısı konulmuştur.

Sonuç: Orta ve ileri yaş hastalarda karın ağrısının ayırıcı tanısında PVT mutlaka göz önünde bulundurulmalı, altta yatan karaciğer sirozu veya trombofili gibi hastalıklar mutlaka araştırılmalıdır.

Anahtar Kelimeler: portal ven trombozu, etiyoloji, yaş, cinsiyet

Introduction

Portal vein thrombosis (PVT) is an uncommon medical condition and a significant cause of asymptomatic portal hypertension (PHT).(1) Numerous prothrombotic factors and local abdominal pathologies can contribute to the development of PVT. Therefore, understanding the age, gender, and etiological distribution of the disease is crucial for appropriate management and follow-up.

PVT results from thrombus formation in the main branch or intrahepatic branches (right or left) of the portal vein. It may also involve the splenic vein or superior mesenteric vein.(2) In some cases, it occurs in the absence of underlying liver disease, termed idiopathic PVT. Historically, PVT was often equated with extrahepatic portal vein obstruction (EHPVO).(1)

PVT is a leading cause of non-cirrhotic portal hypertension worldwide and accounts for approximately 30% of esophageal variceal bleeding and a significant portion of variceal hemorrhage in pediatric patients(3). The increased use of Doppler ultrasonography (Doppler USG) has led to a rise in diagnoses in recent years. The lifetime risk of developing PVT in the general population is estimated to be around 1% (4).

Over time, the recognized etiological spectrum of PVT has expanded. Many cases once deemed idiopathic have now been attributed to thrombophilic conditions or local predisposing factors—approximately 60% and 30%, respectively. Some patients may present with multiple prothrombotic factors. One study reported that 87% of PVT patients had at least one risk factor, including intra-abdominal inflammation (5).

Liver function is generally normal or near-normal unless cirrhosis is present. Elevated alkaline phosphatase (ALP) levels may be observed in portal hypertensive biliopathy. Liver size and weight tend to remain within normal limits, although regenerative nodules and atrophy may occur due to hepatocyte apoptosis and compensatory arterial vasodilation (6).

Material and Methods

Subjects

This retrospective cohort study was conducted at the Internal Medicine and Gastroenterology outpatient clinics of Dursun Odabaş Training and Research Hospital, Van Yüzüncü Yıl University, between January 2011 and December 2013. Ethical approval was obtained from the institutional review board.

Seventy adult patients diagnosed with PVT during the study period were included. Diagnosis was confirmed using clinical history, physical examination, complete blood count, liver function tests, evaluation of inherited and acquired thrombophilic markers, Doppler ultrasonography (USG), computed tomography (CT), and/or the presence of esophageal varices. Patients with no identifiable etiology were classified as idiopathic PVT.

Of the 70 patients, 16 (22.8%) had underlying chronic myeloproliferative disorders, 26 (37.5%) had one or more thrombophilic conditions, and 15 (21.4%) had pre-existing liver cirrhosis. Thirteen patients (18.3%) were categorized as idiopathic.

Statistical Analysis

Statistical analysis was performed using SPSS version 10.0.

Continuous variables were presented as mean and range; categorical variables were summarized as frequencies and percentages. For group comparisons, the Student's t-test was used for normally distributed variables and the Mann-Whitney U test for non-normally distributed variables. Pearson or Spearman correlation coefficients were calculated for associations. Categorical variables were compared using the Z test and Chi-square test. A p-value of <0.05 was considered statistically significant.

Results

A total of 70 patients diagnosed with PVT were evaluated based on clinical history, laboratory data, and ultrasonographic findings. Demographic, radiologic, and biochemical characteristics are summarized in the following sections.

Among the 70 patients, 31 (44.3%) were female with a mean age of 41.48 years, and 39 (55.7%) were male with a mean age of 47.33 years. The age difference was not statistically significant (p = 0.5) (Table1).

Myeloproliferative disorders—such as polycythemia vera, essential thrombocythemia, or chronic myeloid leukemia—were found in 16 patients (22.8%). Elevated ALP levels (>300 U/L) were found in 17 patients (24.2%) and elevated GGT levels (>60 U/L) in 20 patients (28.4%), with no statistically significant difference between them (p = 0.12) (Table2).

Five patients (7.1%) were previously diagnosed with diabetes mellitus (DM), which did not differ significantly from the general population rate (p = 0.07). Direct bilirubin levels were above 1 mg/dL in five patients (7.1%), but ALP and GGT elevations were more pronounced.

ALT levels were elevated in 16 patients (22.8%) and AST levels in 17 (24.2%), with comparable elevation rates among PVT patients (p = 0.01). CRP levels were elevated (>5 mg/L) in 31 patients (44.2%), attributed to acute inflammation in some and chronic PVT with secondary infection in others. (Table2)

In 26 patients (37.5%), at least one deficiency in Protein C, Protein S, or Antithrombin III was identified. Factor V Leiden mutation was evaluated in 29 patients and found to be absent in all. USG data from 60 patients showed that 31 (51.6%) had a portal vein diameter >12 mm, and 24 (40%) had a splenic vein diameter >10 mm.

Thrombus localization was as follows: 50 patients (71.4%) had isolated portal vein thrombosis; 4 (5.7%) had portal and superior mesenteric vein involvement; 1 (1.4%) had thrombus involving the portal, superior mesenteric, and splenic veins; and 4 (5.7%) had portal and splenic vein thrombosis (Table3).

Ultrasound imaging revealed splenomegaly (>12 cm) in 38 patients (63.3%) and hepatomegaly (>14 cm) in 14 patients (23.3%). Splenomegaly was significantly more frequent than hepatomegaly (p = 0.02) (Table 4).

Table 1. Demographic characteristics of the patients.

		Frequene	%		Mean	Std Deviation
Gender	Female	31	44.3	Age	41.48	13.28877
	Male	39	55.7		47.3333	17.52041
	Total	70	100			

Table 2. Laboratory values of the patients.

WBC	70	2.30	31.3	7.6043	4.59238
HB	70	7.7	20.4	13.1057	2.76074
HCT	70	23	62.5	39.2886	8.02938
AST	70	13	197	37.9429	34.61167
ALT	70	7	190	32.9571	30.47044
GLC	70	68	389	113.3286	55.10878
PLT	70	32	749	229.2714	161.48324
ALP	70	54	885	271.8857	161.3444
GGT	70	7	515	60.3143	79.34433
D.BIL.	70	0.1	25	0.75	3.06966
CRP	70	3	108	16.6857	25.43337

WBC: White blood cell count, HB: Hemoglobi, HCT:Hematocrit, AST: Aspartate aminotransferase, ALT:Alanine aminotransferase, GLC: Glucose, PLT: Platelet Count, ALP: Alkaline phosphatase, GGT:Gamma-glutamyl transferase, D.BIL: Direct bilirubin, CRP: C-reactive protein

Table 3. The location of thrombus.

	Frequency (n)	Percent(%)	Valid Percent(%)	Cumulative Percent(%)
PV	50	71.4	71.4	71.4
PV+SMV	4	5.7	5.7	77.1
PV+SMV+SV	1	1.4	1.4	78.6
PV+SV	4	5.7	5.7	84.3
SMV	1	1.4	1.4	85.7
SV	2	2.9	2.9	88.6
Non	8	11.4	11.4	100
Total	70	100	100	

PV; portal vein, SMV; superior mesenteric vein, SV; splenic vein

Table 4. Liver and spleen size.

	N	Minimum	Maximum	Mean	Std. Deviation
Spleen	38	13	26	17.9342	3.60212
Liver	25	12	21	17.12	2.22336

Discussion

This retrospective study investigated the etiological, demographic, and biochemical characteristics of 70 patients with PVT. PVT accounts for approximately 30–35% of cases of portal hypertension in adults. It may present with or without underlying liver disease and is associated with either acute or chronic thrombotic occlusion of the portal venous system. The estimated prevalence of primary PVT is between 1 and 9 per 100,000 population, and it can occur at any age (7).

Acute PVT may be asymptomatic or present with abdominal pain, fever, and symptoms of bowel ischemia(8). Without timely treatment, it may progress to intestinal necrosis and peritonitis. Chronic PVT may cause cavernous transformation and portal hypertension and is often discovered during the evaluation of hypersplenism or variceal bleeding (9). Portal cholangiopathy, a rare complication of chronic PVT, can result in cholestasis (10).

Etiologies of PVT include liver cirrhosis, myeloproliferative disorders, neonatal omphalitis or umbilical vein catheterization, localized abdominal inflammation, and inherited or acquired prothrombotic conditions. While some causes are genetic, PVT itself is not considered a hereditary disease. Idiopathic cases account for 20–40% of presentations (11,12).

Previous studies, including those by Rajani et al., have documented thrombophilic factors in 22%, myeloproliferative diseases in 11%, malignancies in 7%, infections in 8%, and idiopathic PVT in 40% of patients (11). Autopsy studies in Japan and Sweden found PVT incidences of 0.05% and 1%, respectively, indicating regional variation(.13)

In our study, 37.5% of patients had thrombophilic disorders, 21.4% had cirrhosis, 22.8% had myeloproliferative disease, and 18.3% were idiopathic. There was no significant sex or age difference. Non-invasive imaging such as Doppler USG, CT, and MRI effectively identified PVT and its complications. MRI is particularly useful in diagnosing portal cholangiopathy.

In acute PVT, differential diagnoses should include all causes of abdominal pain, while in chronic cases, portal hypertension of non-hepatic origin should be considered (14,15) Genetic counseling is advised in hereditary thrombophilia (16,17). Acute PVT treatment includes anticoagulation for 3–6 months and management of the underlying cause (18). Surgical intervention may be necessary in cases of bowel infarction (19,20).

Chronic PVT management focuses on treating complications of portal hypertension using beta-blockers, band ligation, sclerotherapy, TIPS, splenectomy, or portosystemic shunts (21,22).

Patients diagnosed early and treated appropriately have favorable outcomes, though prognosis depends on comorbidities and age (23). In our cohort, splenomegaly was more common than hepatomegaly. While liver enzymes were elevated in some patients, there was no significant difference between ALP and GGT. These elevations may reflect portal biliopathy from cavernous transformation.

Hepatic steatosis was detected in only 5 of 70 patients (8.3%), significantly lower than the estimated community prevalence (20–25%). This finding suggests that PVT patients may have a lower prevalence of hepatic steatosis.

Limitations of this study include its retrospective design, small sample size, and potential biases. Although data were collected over three years, only 70 eligible cases were analyzed.

In conclusion, portal vein thrombosis should be considered in the differential diagnosis of abdominal pain in middle-aged adults. Evaluation for underlying liver cirrhosis or hereditary thrombophilia is essential. Portal biliopathy appears to be a common cause of cholestasis in these patients. The observed lower prevalence of hepatic steatosis in PVT patients requires further prospective investigation.

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