Incidence and clinical presentation of portal vein thrombosis in cirrhotic patients

Yasir Furkan Cagin, Yahya Atayan, Mehmet Ali Erdogan, Firat Dagtekin and Cemil Colak

Malatya, Turkey

BACKGROUND: Portal vein thrombosis (PVT) is due to many risk factors, but its pathogenesis is still not clearly understood. To identify the risk factors for PVT, we analyzed the clinical characteristics and complications associated with PVT in cirrhotic patients.

METHODS: We studied patients with liver cirrhosis who were admitted to our unit from April 2009 to December 2014. The patients were divided into the PVT and non-PVT groups, and were compared by variables including gender, age, the etiology of cirrhosis, stage of cirrhosis, complications, imaging, and treatment.

RESULTS: PVT was found in 45 (9.8%) of 461 cirrhotic patients admitted to our hospital. Most patients (45.9%) had hepatitis B virus (HBV)-related cirrhosis, with a similar distribution of etiologies between the groups. However, there was no positive relationship between PVT and etiologies of cirrhosis. Most patients (71.5%) were in the stage of hepatic decompensation. No statistically significant differences were found in complications including esophageal varices, ascites, and hepatic encephalopathy between the groups. However, there was a significant positive correlation between hepatocellular carcinoma (HCC) and PVT (P<0.01). In 30 patients with PVT, thrombosis occurred in the portal vein and/or portal branches, 37.8% were diagnosed on ultrasound.

CONCLUSIONS: The incidence of PVT was 9.8%, mainly in patients with HBV-related cirrhosis. The development of PVT was associated with the severity of liver disease and HCC.

(***Hepatobiliary Pancreat Dis Int 2016;15:499-503***)

KEY WORDS: portal vein thrombosis; cirrhosis; clinical presentation

Author Affiliations: Division of Gastroenterology (Cagin YF, Atayan Y and Erdogan MA); Division of Internal Medicine (Dagtekin F); and Division of Biostatistics and Medical Informatics (Colak C), Medical Faculty, Inonu University, Malatya 44280, Turkey

Corresponding Author: Yasir Furkan Cagin, MD, Division of Gastroenterology, Medical Faculty, Inonu University, Malatya 44280, Turkey (Tel: +90-422-3410660ext4112; Fax: +90-422-3410036; Email: yafur@hotmail.com)

© 2016, Hepatobiliary Pancreat Dis Int. All rights reserved.
doi: 10.1016/S1499-3872(16)60092-9
Published online May 9, 2016.

Introduction

Cirrhosis is the last stage of various types of chronic liver disease (CLD).[1] Cirrhosis is an increasing cause of morbidity and mortality worldwide.[2] Significant clinical complications, such as hepatic encephalopathy, ascites, hepatorenal syndrome (HRS), and esophageal variceal hemorrhage, may be due to portal hypertension inherent in cirrhosis.[3]

Portal vein thrombosis (PVT) is considered a rare[4] but gradually increasing complication that is more likely to occur during late-stage liver cirrhosis.[5] The incidence of PVT in patients with cirrhosis varies according to the severity of the disease.[6] The prevalence of PVT in cirrhosis ranges from 0.6% to 26% in different studies.[7, 8] Although several risk factors have been proposed for PVT, it is considered to be a multifactorial process,[9] and its pathogenesis is still not clearly understood.[10] Hepatic structural derangement reduces portal flow which may be the main responsible mechanism for thrombosis in cirrhotic patients.[11] The outcomes of PVT depend on the extension, duration, and site of the thrombus.[12] PVT may occur acutely or chronically. Although the two types of PVT have similar pathologic causes, the treatments are different.[13] PVT is considered an acute event when symptoms occur within 60 days, with no evidence of portal hypertension or collateral circulation on clinical, radiological, or endoscopic evaluation.[14] Doppler ultrasound is usually sufficient for a diagnosis of PVT, whereas computed tomography (CT) and magnetic resonance (MR) angiography are more sensitive in the assessment of the extent of the thrombus within the portal venous system.[13]

The aim of the present study was to determine the clinical characteristics and complications associated with PVT in cirrhotic patients.

Methods

From April 2009 to December 2014, a total of 461 cirrhotic patients (286 males and 175 females) were identified in the computerized hospital administrative registration

system. The various parameters, including demographic data, clinical manifestations, complications, and PVT were analyzed. Liver cirrhosis was diagnosed based on clinical findings or morphological features. Decompensation was defined *a priori* as a composite outcome involving the appearance of one or several of the following features: clinically detectable ascites, hepatic encephalopathy, variceal bleeding, jaundice, and serum bilirubin of >2.5 mg/dL. A total of 45 cirrhotic patients were found to have PVT in this study. All registered PVT diagnoses were based on either Doppler ultrasound, CT angiography, or MR imaging. Each PVT was identified as partial or complete, with the extension of the thrombus definitions. The patients were analyzed according to the location and degree of thrombosis, imaging findings, and treatment.

The cirrhotic patients were classified into two groups: PVT group (n=45) and non-PVT group (n=416). We measured the above-mentioned variables and compared the two groups statistically.

Statistical analysis

The data were given as frequencies with percentages and medians (range). The relationships between group variables and other categorical variables were examined with the Chi-square test and Yates' correction for continuity. The test of normality for continuous variables was performed with the Shapiro-Wilk test. The Mann-Whitney U test was utilized to compare the continuous variables. All 2-tailed P values <0.05 were considered statistically significant. SPSS version 22.0 was used for all analyses.

Results

PVT was found in 45 patients (26 males and 19 females) with an overall prevalence of 9.8% in cirrhotic patients in the current study. Age and gender were not different between the two groups. The etiology, cirrhotic stages, and incidences of hepatic encephalopathy, ascites, and esophageal varices were not significantly different between the two groups (P>0.05, Table 1). PVT occurred in 16 (35.6%) of the patients with hepatitis B virus (HBV)-related cirrhosis, and in 12 (26.7%), 8 (17.8%), 4 (8.9%), and 2 (4.4%) patients with cryptogenic cirrhosis, hepatitis C virus (HCV)-related cirrhosis, alcoholic hepatitis, and autoimmune hepatitis, respectively. Among the patients with PVT, 10 (22.2%) were in hepatic compensated stage and 35 (77.8%) in hepatic decompensated stage. The incidence of HCC was significantly higher in the PVT group than in the non-PVT group (28.9% vs 13.4%, P=0.009, Table 1). Four PVT patients were diagnosed simultaneously with the diagnosis of HCC.

The occurrence of acute PVT in the cirrhotic patients was 5.2%, but in chronic PVT, 4.6%. Complications, including ascites and varices, were observed in >60% of the patients with PVT.

Most PVT patients were diagnosed by CT angiography and 80% of the PVT patients received conservative treatment. Esophageal and gastric varices were treated with band ligation prior to initiating anticoagulation therapy. Anticoagulation therapy was given with low molecular weight heparin and then switched to an oral anticoagulant (warfarin) when the patient was stable and with no planned invasive procedures. Hemorrhage occurred in 3 patients (2 chronic PVT and 1 acute PVT) and they were discontinued the anticoagulation therapy.

Systemic anticoagulation was initiated in six patients who had no risk of bleeding from varices according to the endoscopic sign. Band ligation was performed in two patients due to the risk of esophageal bleeding. Anticoagulation was also used in patients with chronic mesenteric venous thrombosis. Three patients with complete mesenteric thrombosis were complicated with intestinal necrosis based upon clinical, radiographic, or laboratory parameters. Abdominal exploration was performed and the infarcted small bowel was resected. These patients died after 3, 7 and 10 days, respectively. Liver transplantation as a therapeutic option was performed in 4 patients.

Partial PVT was diagnosed in 60% of the patients and complete PVT in 40%. Solely the portal vein or its branches were thrombosed in 30 (66.7%) patients. The thrombosis extended to the splenic vein (SV) in four (8.9%) patients

Table 1. The descriptive statistics of the groups

Risk factors	PVT (n=45)	Non-PVT (n=416)	Total (n=461)	P value
Age* (yr)	56 (6-83)	53 (16-87)	53 (6-87)	0.26
Gender** (n, %)				
Male	26 (57.8)	260 (62.5)	286 (62.0)	0.39
Female	19 (42.2)	156 (37.5)	175 (38.0)	
Etiology** (n, %)				
HBV	16 (35.6)	196 (47.1)	212 (46.0)	0.28
HCV	8 (17.8)	40 (9.6)	48 (10.4)	0.33
Alcohol	4 (8.9)	15 (3.6)	19 (4.1)	0.36
Cryptogenic	12 (26.7)	114 (27.4)	126 (27.3)	0.40
Autoimmune	2 (4.4)	7 (1.7)	9 (2.0)	0.42
Mixed	3 (6.7)	44 (10.6)	47 (10.2)	0.35
Liver function** (n, %)				
Compensated	10 (22.2)	121 (29.1)	131 (28.4)	0.41
Decompensated	35 (77.8)	295 (70.9)	330 (71.6)	0.42
Complication** (n, %)				
HE	12 (26.7)	82 (19.7)	94 (20.4)	0.26
Ascites	30 (66.7)	249 (59.9)	279 (60.5)	0.66
Esophageal varices	29 (64.4)	271 (65.1)	300 (65.1)	0.51
HCC	13 (28.9)	56 (13.5)	69 (15.0)	**0.009**

*: Quantitative values in brackets are expressed as median (range); **: categorical variables are expressed as percentage. HBV: hepatitis B virus; HCV: hepatitis C virus; HE: hepatic encephalopathy; HCC: hepatocellular carcinoma; PVT: portal vein thrombosis.

Download English Version:

https://daneshyari.com/en/article/8735471

Download Persian Version:

https://daneshyari.com/article/8735471

Daneshyari.com