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LETTERS TO THE EDITOR

Portal vein thrombosis: Etiology and clinical outcome of cirrhosis and malignancy-related non-cirrhotic, non-tumoral extrahepatic portal venous obstruction

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Abstract

The etiology and pathogenesis of portal vein thrombosis are unclear. Portal venous thrombosis presentation differs in cirrhotic and tumor-related versus non-cirrhotic and non-tumoral extrahepatic portal venous obstruction (EHPVO). Non-cirrhotic and non-tumoral EHPVO patients are young and present with well tolerated bleeding. Cirrhosis and tumor-related portal vein thrombosis patients are older and have a grim prognosis. Among the 118 patients with portal vein thrombosis, 15.3% had cirrhosis, 42.4% had liver malignancy (primary or metastatic), 6% had pancreatitis (acute or chronic), 5% had hypercoagulable state and 31.3% had idiopathy, 12% had hypercoagulable state in the EHPVO group.

Key words: Portal vein thrombosis; Cirrhosis; Malignancy; Extrahepatic portal venous obstruction

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TO THE EDITOR

We read with great interest the article "Portal hypertension due to portal venous thrombosis: Etiology, clinical outcomes" by Harmanci *et al*[1] in the May 14, 2007 issue of World Journal of Gastroenterology. We agree that portal vein thrombosis (PVT) should be considered under two different categories [acute or chronic PVT (noncirrhotic and nontumoral), (b) PVT due to cirrhosis and tumors], as the presentation, clinical course and prognosis are different in the two categories.

Table 1 Clinical and laboratory findings in 118 patients with portal vein thrombosis

Parameters	Extrahepatic portal vein obstruction (*n* = 50)	Cirrhosis and tumor-related portal vein thrombosis (*n* = 68)
Male:Female	33:17	51:17
Age (yr)[2]	25 (9-40)	52 (23-80)
Hematemesis	36 (72%)	10 (14.7%)
Hypersplenism	30 (60%)	6 (8.8%)
Pain abdomen	18 (36%)	42 (61.8%)
Abdominal distension	8 (16%)	36 (52.9%)
Awareness of splenomegaly	6 (20%)	-
Jaundice	5 (10%)	18 (26.5%)
Cholangitis	1 (2%)	5 (7.4%)
Splenomegaly[2] cm[1]	4 (2-14); (*n* = 35)	2 (2-4); (*n* = 7)
Hepatomegaly[2] cm[1]	2 (1-3); (*n* = 8)	3 (2-6); (*n* = 16)
Etiology		
Hepatocellular carcinoma	-	31
Cirrhosis liver	-	18
Metastases liver	-	6
Pancreatic carcinoma	-	7
Cholangio carcinoma	-	3
Carcinoma gallbladder	-	2
Duodenal carcinoid	-	1
Esophageal Varices	35 (70%)	32 (47%)
Gastric Varices	11 (22%)	3 (4.4%)
GoVI	1	2
GoV2	8	1
IGV1	2	
Portal hypertensive gastropathy	8 (16%)	48 (70.4%)
Mild	6	40
Severe	2	8
Hemoglobin[2] (gm/dL)	7.2 (1.7-14.7)	8.0 (6.0-11.6)
Total leucocyte count[2] (10^3/mm^3)	2.9 (1.0-6.8)	6.4 (1.36-29.71)
Platelet count[2] (10^5/mm^3)	0.49 (0.14-1.54)	1.52 (0.29-2.59)
Bilirubin[2] (mg/dL)	1.0 (0.4-18.5)	1.2 (0.5-15.1)
AST[2] (U/L)	38 (18-231)	79 (39-961)
ALT[2] (U/L)	34 (18-254)	65 (18-1146)
SAP[2] (U/L)	264 (110-2849)	348 (140-3140)
Protein/albumin[2] (mg/dL)	6.8 (5-7.5)/3.8 (3-4)	6 (5.3-8)/3.3 (2-4.6)
Prothrombin time prolongation[2] (seconds)	2 (1-3)	4 (2-14)
Portal biliopathy	4 (8%)	
Choledocholithiasis	2	
CBD stricture	2	
EVL sessions[2]	3 (2-4)	-
Glue injection (*n*)	4	-
Superior mesenteric vein thrombosis	11 (22%)	6 (8.8%)
Inferior vena cava thrombosis	4 (8%)	3 (4.4%)
Splenic vein thrombosis	4 (8%)	4 (5.6%)
Deep vein thrombosis	-	1 (1.4%)
HBsAg	0	27 (39.7%)
Anti-HCV	0	3 (4.4%)

[1]Centimeter below costal margin; [2]Median (range).

Of the 118 cases of PVT admitted to our hospital over the 2-year period (from January 1, 2005 to December 31, 2006), 50 were due to extrahepatic portal vein obstruction (EHPVO) and 68 were due to cirrhosis and tumors. The clinical and laboratory characteristics of 50 patients with EHPVO are given in Table 1.The patients were young and commonly presented with features of hematemesis, hypersplenism, abdominal pain and distension. Ten patients had acute PVT and 2 had presentation as acute Budd-Chiari syndrome, 2 patients had pregnancy and delivered the fetus at term with supportive treatment. Thirty-six patients who presented with hematemesis were managed with endoscopic variceal ligation (2-4 sessions), and four patients were treated with gastric varices glue injection. These patients were maintained on beta-blockers and follow-up endoscopic surveillance. Four patients with symptomatic portal biliopathy were managed with stent placement (n = 2) and common bile duct (CBD) stone extraction (n = 2). Antithrombin-Ⅲ deficiency was present in 2 patients, antiphospholipid antibody in 2 patients, factor V Leiden (FVL) mutation in 1 patient and paroxysmal nocturnal hemoglobinuria in 1 patient. All the 6 patients were started on heparin and warfarin with warfarin continued to maintain a 2-3 INR. Four (8%), 2 (4%) and 1 (2%) patients had chronic pancreatitis, acute pancreatitis and liver abscess, respectively. During the mean follow-up period of 9 mo (3-24 mo), none of the patients had symptomatic hypersplenism.

EHPVO affects young individuals who present with well-tolerated bleeding. The etiology and pathogenesis of PVT are unclear. It was initially proposed that umbilical sepsis or catheterization of umbilical veins in the neonatal period is responsible for PVT[2]. In recent years, the presence of congenital or acquired prothrombotic conditions has been considered an interesting hypothesis for the causation of PVT. FVL mutation is known to be less common among Asians as compared to the population of European descent. Koshy *et al*[3,4] reported that FVL mutation has been found in 3% patients with portal vein thrombosis and prothrombin G20210A gene in patients with portal vein thrombosis in a south Indian study. In our study, FVL mutation was present in 2% of cases.

The clinical and laboratory features of 68 patients in cirrhosis and tumor groups are given in Table 1. The clinical presentation was abdominal pain and distension and jaundice. During a mean follow-up period of 7 mo (range 1-24 mo), 48% of the patients died.

Of 118 cases of PVT admitted to our hospital, 15.3% had cirrhosis, 42.4% had liver malignancy (primary or metastatic), 6% had pancreatitis (acute or chronic), 5% had hypercoagulable state and 31.3% had idiopathy. The higher percentage in idiopathic group might be due to a low prevalence of hypercoagulable factors, abnormality and attribution of umbilical sepsis in childhood. The role of JAK2 mutation in early diagnosis of overt or silent myeloproliferative disease cannot be undermined but requires standardization[5].

In conclusion, the presentation, etiology and prognosis in non-cirrhotic and non-tumoral EHPVO patients are different from those in cirrhosis and tumor-related portal vein thrombosis patients.

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