## **LETTERS TO THE EDITOR** EDİTÖRE MEKTUP **Portal venous aneurysm and liver cirrhosis**

Portal ven anevrizması ve karaciğer sirozu

Portal vein aneurysms (PVAs) are unusual vascular abnormalities defined as localized dilatation of the portal vein. Though venous abnormalities of the portal system have been determined in detail in the past, their etiology, pathophysiology, clinical features and management were obscured because of rarity of the lesions (1). The etiology is supposed to be congenital or acquired (2). Increasing numbers of incidental cases are being reported with the recent advances in abdominal imaging modalities (3). Here, we describe a case with portal vein aneurysm which quite possibly arose following portal hypertension due to hepatic cirrhosis.

A 43-year old male patient was admitted to the hospital because of gastrointestinal tract bleeding. The liver was not palpable, and the spleen was palpable 4 cm below the left costal margin. No dilated veins were noted on the abdominal wall. No abdominal mass could be felt. Liver function tests were normal. He was HBsAg positive and anti-HCV negative. The patient's hemoglobin was 8 g/dl, and white blood cell count 3800/ml. His platelet count was persistently low, around 70,000/mm<sup>3</sup>. The laboratory analyses reflected hypersplenism. Endoscopy of the esophagus showed grade 4 varices. Contrast enhanced computerized tomography (CT) examination revealed an aneurysm with 40x45 mm dimensions on the right branch of the portal vein at the level of the fifth segment of the liver at the portal phase. There were no symptoms or signs that could be attributed to PVA in our patient. Cirrhosis was diagnosed by liver biopsy and portal hypertension by echography based on morphology (changes in form and size of the liver, splenomegaly).

In a healthy person without hepatic disease or portal hypertension, the diameter varies between

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1.07 and 1.24 cm, with an average of 1.15 cm (1). Douts and Pearce (4) stated that the maximum antero-posterior diameter of the portal vein never exceeds 1.9 cm even in cirrhotic cases. Acquired PVAs may develop as a consequence of weakening of the venous walls due to pancreatitis, injury to the portal vessel wall from trauma, or portal hypertension related to liver disease (1, 3). PVAs may compress adjacent organs, and may also cause cholestasis and jaundice. Operative treatment of PVAs depends on size, symptoms, complications, and condition of the aneurysm (3, 5). PVAs may be managed by regular observation alone; patients may have no serious complications. PVA in this patient may have been due to portal hypertention associated with liver cirrhosis. The patient's symptoms resolved by medical treatment, and no surgical intervention was performed.

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