Portal vein aneurysm ; CT, MR and MR angiography appearances

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Abstract

Portal vein aneurysm is an unusual vascular abnormality. Up to now, 35 cases were described in the literature. In this case report the diagnosis was made with ultrasound, Doppler ultrasound, CT, MRI, MR angiography, and Digital Subtraction Angiography. Clinical and imaging characteristics as well as the treatment opportunities of this vascular abnormality are discussed. (Acta gastroenterol. belg., **2002**, 65, **136-138**).

Key words : portal vein, aneurysm, CT, MRI, Doppler Ultrasonography.

0Introduction

Venous aneurysms, are rarely encountered in clinical practice and occur more often in the popliteal, jugular and saphenous veins, and less often in the femoral, forearm veins and vena cava or portal vein. Portal vein aneurysm is a rare vascular abnormality which the cause and clinical manifestations are not yet clarified. In published reports, the relation between portal vein aneurysms and portal hypertension has been emphasized (1). We report a case of portal vein aneurysm to show the characteristic MRI findings of this condition in the absence of portal hypertension.

Case report

The patient was a 70-year-old man who presented with abdominal pain for a year. No history of jaundice, hematemesis, melena, hematochezia, abdominal inflammation or trauma was reported. Physical examination was unremarkable. Laboratory studies including liver-associated enzymes were in the normal range.

Abdominal ultra-sonogram showed a cystic mass behind the head of the pancreas, freely communicating with the lumen of the porta, superior mesenteric and splenic veins that compressed the inferior vena cava. On the otherhand, the results of the ultrasound examination were normal, specifically, no splenomegaly nor portal vein thrombosis were discovered.

Color Doppler ultrasonography demonstrated a turbulent, whirl-pool-like blood flow in the aneurysm, which immediately led to the diagnosis.

Computed tomography scan (Fig. 1) showed the mass with a diameter of about 4.5 cm to be homogeneously enhanced after injection of contrast medium at the confluence of the superior mesenteric and splenic veins.

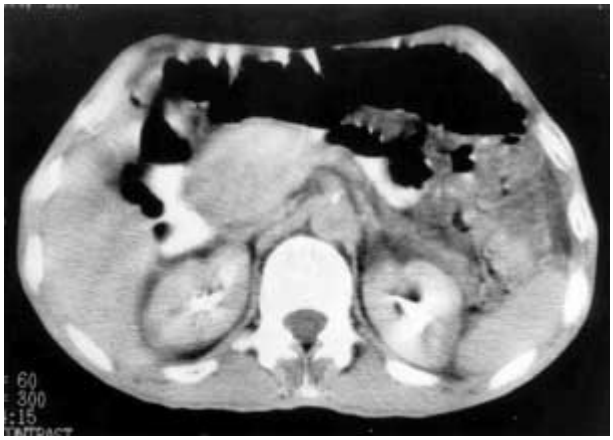

Fig. 1. — CT scan appearance of the aneurysm, the mass was homogeneously enhanced after the injection of contrast medium with a diameter 4.5 cm at the confluence of the superior mesenteric and splenic veins.

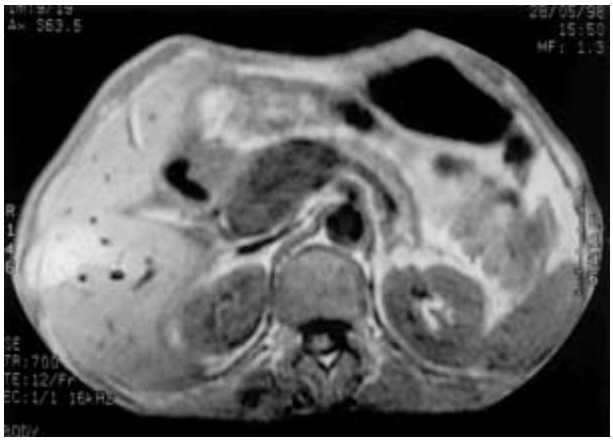

Fig. 2. — MRI appearance of the portal vein aneurysm.

MR evaluation (GE, 1.5 Tesla) showed the mass behind the head of the pancreas, communicating with the lumen of the portal vein, superior mesenteric and splenic veins that compressed the inferior vena cava (Fig. 2-3).

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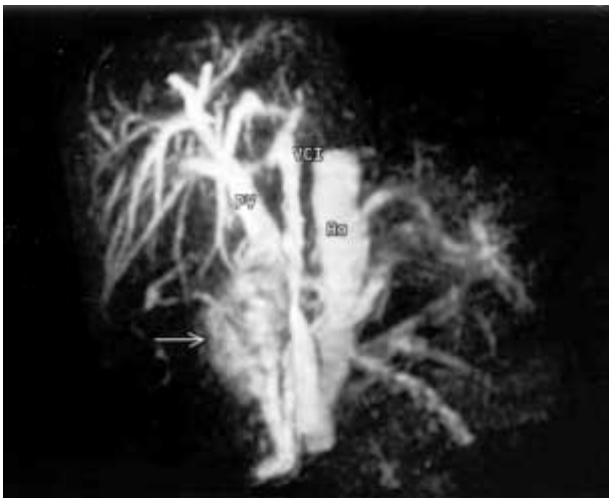

Fig. 3. — MR angiography appearance of the portal vein aneurysm.

T1 weighted MRI also demonstrated the aneurysm. The signal in the aneurysm was gray, probably due to swirling blood flow.

Discussion

The precise definition of a portal vein aneurysm (PVA) is sometimes difficult or ambiguous. PVA may be intrahepatic or extrahepatic. This vascular abnormality may be localized at the hepatic hilum, the bifurcation of the right and left portal veins or at the confluence of the superior mesenteric vein and the splenic vein (1, 2).

Although the exact cause of portal vein and superior mesenteric vein aneurysms is yet to be determined, two major etiologies, acquired and congenital, have been proposed. Acquired lesions are secondary to liver diseases, portal hypertension, trauma or degenerative changes. Others contend that portal vein aneurysms are congenital in origin, resulting from an inherent weakness of the vessel wall.

Furthermore, it is clinically difficult to determine the cause in the majority of these cases. Although the association between portal hypertension and portal aneurysm has been emphasized, this relation is now doubtful. Thirty five extrahepatic portal vein aneurysms in 34 patients were reported in the literature (Table 1) (1-4). Twelve (35%) were associated with portal hypertension and 9 of these patients had chronic liver disease. The expanded use of non-invasive medical imaging techniques has increased the number of patients with portal vein aneurysm and with no portal hypertension or chronic liver disease (1). The true frequency of congenital portal vein aneurysm remains uncertain. Congenital portal vein aneurysm can be suspected in cases in which there is no history of trauma, liver biopsy, hepatitis or portal hypertension. However, there is no way to confirm that at present.

Some portal vein aneurysms are discovered incidentally. In other cases, patients present with abdominal pain, as in our case, or upper gastrointestinal bleeding that occurs from rupture of the aneurysm in the duodenum. Some aneurysms become large enough to compress the duodenum or the adjacent common bile duct, resulting in biliary obstruction or the compression of the inferior vena cava. Large PVA may compress the portal vein or completely obstruct it when thrombosed. Acute portal hypertension and rupture have been reported (3). Although thrombosis of PVA and embolization to the liver has not been reported, it might be a potential complication with the secondary development of portal hypertension.

With the extensive use of real-time ultrasound and CT more portal aneurysms will probably be found incidentally.

With the real-time ultrasound study, demonstration of the continuity of the mass with the lumen of the portal vein or its tributaries is diagnostic. Some difficulties may arise if the mass is partly echogenic as a result of mural thrombosis. Dynamic CT scanning following bolus contrast medium injection shows enhancement of the cystic mass during the venous phase, which is diagnostic of the aneurysm and can detect mural thrombus.

The multiplanar capability of magnetic resonance imaging (MRI) along with its capacity to render angiogram-like images from vascular structures makes MRI well suited for the evaluation of such aneurysms. Additionally, T1 weighted images showed signal void within portal vein, superior mesenteric and splenic veins suggesting patent flow without the need for additional use of contrast material.

To sum up, we presented ultrasonography, CT and MRI findings of PVA. We believe that ultrasonography is the initial screening modality in-patients with abdominal pain or mass which may rarely result from PVA. CT and MRI were equally diagnostic in this case. However, the main advantages of MRI over CT are especially lack of ionising radiation and the risks of contrast reaction. Additionally it provides multiplanar images in which anatomical relationships of PVA and its thrombosis can be defined more precisely.

In the past, venous-phase mesenteric angiography and splenoportography were used to evaluate these aneurysms.

However, due to the invasiveness of angiographic procedures, ultrasonography CT and MRI are now preferred in most instances.

Operative treatment of PVA depends on size, symptoms, aneurysmal complications, anatomy and condition of the patients. Patients who have biliary obstruction or hemobilia caused by a PVA, require operation. Patients with thrombosis of a PVA that extends into the superior mesenteric and splenic veins should undergo thrombectomy and restoration of portal vein configuration. As in our case patients with no serious complaints and no signs of portal hypertension are not referred to the surgeon but are seen for follow-up ultrasound studies.

Table 1. — **Summary of the reported cases of extrahepatic portal vein aneurysms**

Author	Age-Sex	Size (cm)	Location	Portal Hypertension	Liver Disease
Ohnami	54 M	2.4 × 2.1	Hepatic hilum	–	–
Ohnami	72 M	2.1 × 1.9	Hepatic hilum	–	–
Ohnami	56 M	2.2 × 2	Hepatic hilum	–	–
Ohnami	47 F	3 × 2.5	Hepatic hilum	–	–
Ohnami	62 F	2.7 × 2	SPV-SMV C	–	–
Fulcher	35 F	2.5	PV	–	–
Fulcher	74 M	3.4 × 3.3	PV-SMV C	–	+
Fulcher	56 F	1.8	SPV-SMV C	–	–
Patricia	72 F	6	Hepatic hilum	–	–
Boyez	57 F	4.3 × 2.5	SPV SMV C	–	–
Barzilai	21 F	2	Hepatic hilum	+	+ Cirrhosis
Leonsins	52 M	8 × 4	MPV	+	+ Periportal fibrosis
Sedgwick	25 F	5	SPV-SMV C	+	+ Cirrhosis
Hermann	26 F	4 × 6	SPV-SMV C	+	+ Slight portal fibrosis
Thomas	18 M	8 × 6 × 4	SPV-SMV C	+	–
Thomas	13 F	3	Hepatic hilum	+	+ No Cirrhosis
Liebowitz	55 F	8	SPV-SMV C	–	–
Wenz	6 M	ND	MPV	+	–
Duhmke	57 F	5	Hepatic hilum	+	+ Cirrhosis
Vine	50 F	3	SPV-SMV C	–	+ Hepatic parenchymal abnormalities
Muller	52 F	9	MPV	+	+ Cirrhosis
Kane	ND	ND	SPV-SMV C	+	+ Cirrhosis
Fahrendorf	34 F	4	SPV-SMV C	–	–
Boyez	57 F	4 × 3 × 2.5	SPV-SMV C	–	–
Thompson	21 F	6	Hepatic hilum	–	–
Dognini	67 F	2.4	SPV-SMV C	–	–
Andoh	57 F	6 × 8	MPV	–	–
Lee	5 M	1.9	SPV-SMV C	–	–
Hagiwara	34 M	2.7 × 2.1	MPV		
		2.1 × 1.3	Hepatic hilum	–	–
Fukui	34 M	3	Hepatic hilum	–	–
Savastano	13 F	6 × 4 × 5	SPV-SMV C	–	–
Glazer	26 F	ND	SPV-SMV C	+	–
Malde	44 F	0.8	SPV-SMV C	+	+ Periportal fibrosis
Itoh	59 F	1.6	SPV-SMV C	–	+ Lupoid hepatitis

ND : Not described
MPV : Main portal vein
SPV-SMV C : Confluence of splenic vein and superior mesenteric vein
+ : Present
– : Absent.

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