CASE REPORT

Portal vein aneurysm causing obstructive jaundice

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Introduction

Portal vein aneurysms are not commonly encountered in clinical practice. They are usually extrahepatic and occasionally intrahepatic. Most are asymptomatic and detected incidentally; they may be associated with portal hypertension. Complications include thrombosis, rupture and formation of portosystemic shunts. A review of the English-language literature revealed 47 cases1–3 of portal vein aneurysm, but there has been only one English-language report in more than the last three decades of a aneurysm leading to cholestasis.4 We present such a case, together with a comprehensive review of the literature on portal vein aneurysms.

Case report

A 42-year-old man presented with complaints of abdominal pain, yellowish discoloration of the urine and conjunctiva and generalized pruritus. The total serum bilirubin was elevated to 17.5 mg% and direct bilirubin measured 13.0 mg%. Urinalysis yielded bile salts and pigments.

Ultrasound (US) of the abdomen revealed dilatation of the intrahepatic biliary radicles (Fig. 1), and a cystic lesion measuring 3×3 cm was seen at the porta (Fig. 2). On Doppler examination, the entire lesion filled with colour in a swirling fashion (Fig. 3), reflecting an aneurysm; this vascular malformation was continuous with the portal vein. Spectral analysis of the lesion showed a monophasic venous waveform. The biliary tract was dilated proximal to this lesion. A diagnosis of saccular aneurysm of the portal vein, compressing the bile duct, and secondary cholestasis was made. Endoscopic retrograde cholangiopancreatography confirmed extrinsic compression of the common bile duct at the level of the aneurysm (Fig. 4). Additionally, a few tiny calculi were removed from the common duct. The patient made an uneventful recovery.

There ensued a dilemma as to whether the aneurysm should be surgically removed. Eventually, as recommended in literature,5 it was decided that further management should be based on the patient’s clinical course, with follow-up US every 6 months. At the first follow-up the patient was asymptomatic, although US showed mild, persistent dilatation of the biliary tract. There was no choledocholithiasis.

Discussion

The first case of portal vein aneurysm was reported in 1956. Subsequently, 47 cases1–3 have appeared in the English-language literature. We identified four observations of a portal vein aneurysm leading to cholestasis, of which the first two cases were reported in 1967 and 1968 and the third in the Spanish literature. There was only one English-language report4 in more than the last three decades of a portal vein aneurysm leading to cholestasis.

Two schools of thought exist with regard to the aetiology of portal vein aneurysms. Theories proposing an acquired origin are based on the premise that in most reported cases there have been causative disorders such as portal hypertension, necrotizing pancreatitis or previous abdominal trauma or surgery.2 In the few reported cases occurring in utero and the neonatal period,8 the absence of underlying liver disease or other predisposing factors favours a congenital7 aetiology.

These aneurysms are often identified on colour Doppler US. Computed tomography (CT), magnetic
resonance imaging (MRI) and portography or contrast angiography are complementary procedures for confirming the diagnosis and aiding surgical planning. In our case, colour Doppler US was the only technique used to confirm the diagnosis.

The majority of these aneurysms are located at the portal confluence or bifurcation. Turbulence of flow at the confluence may contribute to this predilection. Extrahepatic aneurysms are usually larger than their intrahepatic counterparts, probably because of growth restrictions on the latter by adjacent hepatic parenchyma. Portal vein aneurysms are frequently associated with liver disease; they can undergo thrombosis leading to portal hypertension. If large, these aneurysms can compress other organs, such as the duodenum, inferior vena cava or biliary tract. In one previous report of a portal vein aneurysm leading to obstructive jaundice, the patient’s bilirubin levels suddenly fell following spontaneous rupture of the aneurysm, thus emphasizing the cause-and-effect relation between portal aneurysms and cholestasis.

The treatment of portal venous aneurysms remains controversial. In asymptomatic patients,
without evidence of portal hypertension or cirrhosis, an aneurysm is not expected to grow and conservative management with close follow-up is recommended.\textsuperscript{2,5} Surgical treatment depends upon the size, symptoms, complications and clinical condition of the patient. Surgical options include aneurysmorrhaphy\textsuperscript{4} and creation of portocaval or mesocaval shunts, with a view to reducing portal venous pressure and thus preventing progressive dilatation of the aneurysm.\textsuperscript{2}

In our case it was decided that surgical removal of the aneurysm would only be undertaken if the patient presented again with another episode of cholestasis or if the aneurysm grew in size on follow-up US studies.

References