Successful Resection of a Massive Hepatic Artery Aneurysm

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SUMMARY: Hepatic artery (HA) aneurysms are seen infrequently, but many eventually rupture, often with fatal outcome. With modern diagnostic technology, aneurysms can be identified at an earlier stage, providing the opportunity for intervention. This review outlines the case of a patient with a massive, leaking hepatic artery aneurysm and discusses the aetiology and management of these lesions.

Introduction

Hepatic artery (HA) aneurysms are uncommon and most are asymptomatic. The risk of aneurysmal rupture is up to 44%(1), and the mortality rate from a ruptured HA aneurysm is high, exceeding 35%(2,3). Rupture may occur into the peritoneal cavity, gastrointestinal tract or the portal vein.

Fig 1. Lateral abdominal radiograph taken seven years prior to this presentation showing calcification of the aorta and a large hepatic artery aneurysm (arrowed). The position is more typical of a splenic artery aneurysm. The differential diagnosis includes a calcified pancreatic pseudocyst.

Case Report

In 1992, a 78 year old lady presented with a twelve hour history of constant epigastric pain, radiating to the back. Her medical history included a partial gastrectomy

in 1954 for a chronic pyloric ulcer. No arterial anomalies were mentioned in the operation note. During investigations for anaemia in 1985 an epigastric mass was detected. Radiographic (Fig 1) and ultrasound examination revealed a 6 x 4 centimetre lesion which was thought to be a splenic artery aneurysm or a pancreatic pseudocyst. The patient had been unwilling to undergo surgery and had remained well until the onset of pain.

On examination, her pulse was 80 per minute and blood pressure 180/110. A large, tender pulsatile mass distended the left hypochondrium, over which there was a bruit. Her serum amylase was slightly elevated at 189 U/l and alanine transferase was moderately raised but other liver function tests were normal.

Ultrasound scanning demonstrated a pulsatile mass measuring 13 x 10 centimetres, to the left of and separate from the aorta. Contrast-enhanced computerized tomography (Figs 2a & 2b) confirmed a large aneurysm near the origin of the splenic artery. The patient refused to undergo arteriography, but agreed this time to surgery.

At laparotomy, the mass was found to be a massive, saccular aneurysm filling the lesser sac, and arising from the common hepatic artery distal to the gastroduodenal artery. A small leak was found on the left lateral aspect. The liver appeared healthy. The aneurysm was resected and the hepatic artery ligated. Postoperatively, the liver function tests showed moderate elevation of all enzymes and a drop in serum albumin to 28 g/l; these returned to normal within ten days and the patient made an uneventful recovery.

Discussion

Aetiology

Aneurysms of visceral arteries are uncommon. Hepatic artery aneurysms are the second most frequent

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Fig 2a. Axial, contrast-enhanced CT scan at the level of the left renal artery showing a massive aneurysm separate from the mid-line aorta. The lumen of the aneurysm is very superficial, with atheroma and thrombus posteriorly. The left kidney is atrophic.

Fig 2b. L = liver, S = spleen, Ao = aorta, RK = right kidney, LK = atrophic left kidney, An = common hepatic artery aneurysm, Lu = true lumen of aneurysm.

encountered (15-20% after those of the splenic artery. The male to female ratio is 2:1 (3) and most patients present in their sixth decade (4). Stanley and Thompson (5) found that twenty per cent of HA aneurysms affect the intrahepatic vessels and that the common hepatic artery (CHA) is involved in 63% of extrahepatic aneurysms. Most HA aneurysms are single and may be saccular, fusiform or dissecting in type.

Atherosclerosis is the most frequent cause of splanchnic artery aneurysms. Inflammatory causes include polyarteritis nodosa and necrotising vasculitis. Less commonly, a HA aneurysm arises from a congenital weakness of the tunica media. Cystic medial necrosis may lead to a dissecting aneurysm. False aneurysms are associated with blunt or penetrating trauma, or may develop after upper abdominal surgery (3,6).

Clinical Features

Most HA aneurysms are asymptomatic. Expanding or leaking aneurysms commonly present with severe pain, typically in the epigastrium or right upper quadrant of the abdomen, and radiating to the back. Obstructive jaundice may result from extrinsic compression of the bile duct by the aneurysm or intrinsic obstruction by blood clot. Quincke's triad consists of abdominal pain, haemobilia and obstructive jaundice. The diagnosis may be mistaken for biliary colic or pancreatitis. Whitehouse *et al* (1) estimated the risk of aneurysmal rupture at 44%, although other investigators (3) suggest that the risk is likely to be less than 20%. The aneurysms may leak into the peritoneal cavity, biliary tree or gastrointestinal tract, and the mortality from a ruptured HA aneurysm is high, exceeding 35% (4,7).

Radiological Investigation

Plain abdominal radiography often shows ring calcification, usually in the right hypochondrium. Barium

studies may reveal an extrinsic filling defect in the duodenal cap. Isotope studies are also of limited use (7). Real-time ultrasound is more valuable and may show a pulsatile cystic structure that is echo-poor or has mixed echoes from atheroma or thrombus. Doppler studies, enhanced by colour-flow (8), will demonstrate arterial waveforms and possibly turbulent flow within the aneurysm. Computerised tomography (CT) shows a mass with possible rim calcification. CT has the advantage of multiplanar imaging but this is not yet widely available (10).

Coeliac and superior mesenteric angiography remain the cornerstone of radiological diagnosis. Angiography displays the site, size and number of aneurysms and the vascular anatomy and any collateral circulation are clearly defined (11).

Treatment

In 1903, Kehr performed the first successful surgical procedure for HA aneurysm, by ligation and division of the artery (12). The first HA reconstruction for aneurysm was carried out by Paul in 1951(4). In 1960 Michaels (13) reported that aneurysms of the CHA proximal to the gastroduodenal artery could usually be ligated without causing hepatic necrosis as a collateral supply was provided from the superior mesenteric artery via the gastroduodenal and right gastric arteries (Fig 3). Bengmark and Rosengren (14) found that a collateral supply may also develop from the inferior phrenic and intercostal arteries. Even with total loss of hepatic arterial supply, liver necrosis is unlikely provided portal blood flow and liver function are normal. Under normal conditions, the portal vein provides 50% of the hepatic oxygen supply (15). In the presence of parenchymal liver disease Stanley and Zelenock (3) advise reconstruction of the distal HA by primary repair or by aorto-hepatic interposition grafting.

Fig 3. Aneurysms of the common hepatic artery proximal to the gastroduodenal artery can be ligated as the gastroduodenal and right gastric arteries provide a collateral supply to the liver.

Intrahepatic aneurysms may warrant partial hepatectomy (16), but in the event of massive haemorrhage CHA ligation may be necessary (7). In 1989, Song (11) reported encouraging results by selective arterial embolisation using a variety of embolic materials such as Gelfoam plugs, stainless steel coils, and autologous blood clot. Embolisation of the feeder vessel may ablate the aneurysm or occlude a leak (17,18). Embolisation therapy is more effective in intrahepatic lesions, while extrahepatic aneurysms should be managed surgically (11).

Comment

This elderly and frail lady had a satisfactory outcome from her operation. Histological examination of the aneurysm wall showed some non-specific inflammatory changes. It is possible that the aneurysm developed as a late complication of her gastric surgery. But for the patient's reluctance an angiogram might have been a more useful investigation than CT, giving more precise detail of the artery involved, which did not become clear until late in the dissection.

HA aneurysms have a significant risk of rupture with exsanguinating haemorrhage and should be operated on promptly when this threatens.

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