Hepatic artery aneurysm

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Summary: We describe a 58 year old woman who had an aneurysm involving the common hepatic artery, the right and left hepatic arteries and the cystic artery which had ruptured into the liver. It was successfully treated by proximal ligation. The features and management of hepatic artery aneurysm are discussed.

Introduction

Although rarely seen, aneurysm of the hepatic artery is the second most common visceral aneurysm. Diagnosis is difficult and the mortality may be as high as 80% (Ariyan *et al.*, 1975; Countryman *et al.*, 1983; Guida & Moore, 1966). However, current evidence suggests that repair of these aneurysms is unnecessary – a proximal tie being all that is required. This knowledge, together with advances in diagnosis, should considerably improve the survival of patients with hepatic artery aneurysm.

Case report

A 58 year old woman was admitted with a 5 day history of fever, dry cough, a rigor and pleuritic chest pain. She was known to have had chronic lymphatic leukaemia for 20 years and this was associated with a secondary hypogammaglobulinaemia.

On examination she was pyrexial and tachypnoeic. The right lower zone of her chest was dull to percussion. Her abdomen was not tender and no masses were palpable. A diagnosis of lobar pneumonia was made.

Chest X-ray revealed a large right pleural effusion and possibly consolidation of the lower lobe of her right lung. An electrocardiogram was normal. Treatment with erythromycin 500 mg q.d.s. was given.

Over the next few days she became jaundiced and liver function tests showed an obstructive pattern. Ultrasound examination of her abdomen showed a cystic lesion, 6 cm in diameter at the porta hepatis lying between the common bile duct and the portal vein and extending into the right lobe of the liver. The biliary tree was dilated. A computed tomographic scan gave no additional information. In view of the above

history, a diagnosis of hepatic abscess was thought most likely. An attempt was made to aspirate the contents under X-ray control; this produced 150 ml of altered blood.

At laparotomy the findings were a large aneurysm of the common hepatic artery and the right and left hepatic arteries commencing at the origin of the gastroduodenal artery. It extended along the cystic artery and into the gallbladder, which was ischaemic. The aneurysm had ruptured into the liver and was compressing and displacing the common bile duct and hepatic ducts. There was a large haematoma in the right lobe of the liver and the liver capsule had been stripped on the diaphragmatic surface. The hepatic artery was ligated proximal to the origin of the gastroduodenal artery, and this resulted in collapse of the aneurysm. Cholecystectomy was performed and the damaged liver surface was covered with an omental patch.

Post-operative recovery was slow and the patient spent over two months in hospital. The problems of note were an initial deepening of her jaundice which faded over several weeks; an *Escherichia coli* septicæmia which responded to cephalosporin antibiotics, and a biliary fistula at the drain site. However, by the time of her discharge from hospital she was feeling well, her liver function tests were normal apart from a slightly raised alkaline phosphatase and the biliary fistula was draining only a few mls a day. At review two months later the fistula was closed, she was symptom-free and there were no abdominal signs.

Discussion

The first known case of hepatic artery aneurysm was reported in 1819 by Wilson. The patient was a 50 year old man, who died in 1809 from rupture of an aneurysm of the left hepatic artery and the diagnosis was made at post-mortem examination (Rolland,

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1908). The first successful treatment of an hepatic artery aneurysm was by Kehr in 1903; he ligated and divided an aneurysm of the common hepatic artery which had ruptured into the gallbladder. There are now more than 300 cases reported but only 20% have been successfully treated (Countryman *et al.*, 1983; Ariyan *et al.*, 1975; Guida & Moore, 1966).

Hepatic artery aneurysms constitute 20% of visceral aneurysms and only those of the splenic artery are more common. The precise mechanism leading to the formation of the aneurysm is uncertain but atherosclerosis is an associated factor in 32% of patients and acquired degeneration of the media is found in a further 24%. Whereas at the turn of the century nearly all hepatic artery aneurysms were due to mycotic lesions, they now account for only 10% of cases (Rutherford, 1977). Trauma, including iatrogenic trauma occurring during biliary surgery, may be the cause of 22%. Less common causes are syphilis, tuberculosis, polyarteritis nodosa and cholecystitis (Guida & Moore, 1966).

Twenty per cent of hepatic artery aneurysms are intrahepatic and are usually false aneurysms following trauma. The remaining 80% are extrahepatic. Guida & Moore (1966), in their review of 170 patients, found the anatomical distribution of the extrahepatic aneurysms to be as follows: common hepatic artery 64%, right hepatic artery 30%, left hepatic artery 4% and both left and right hepatic arteries 2%. The age distribution was 10–83 years, mean 38 years. Males were affected twice as often as females.

The clinical features tend to be non-specific. Most patients complain of upper abdominal pain not related to food, and physical signs are rare, though one may find a mass, hepatomegaly or a bruit. Eighty per cent are not detected until haemorrhage occurs. Rupture occurs most often into the peritoneal cavity (43%) and this is usually rapidly fatal; 41% rupture into the gallbladder or bile ducts (Truman-Mays, 1977). The resulting haemobilia is characterized by Quincke's triad of symptoms, viz. biliary colic, gastro-intestinal haemorrhage and obstructive jaundice (Harlaftis & Akin, 1977). Eleven per cent rupture into the stomach or duodenum and 5% into the portal vein (Guida & Moore, 1966; McKinnon *et al.*, 1981). The latter may be followed by portal hypertension and varices (Wheeler & Warran, 1957). Hepatic artery aneurysms following biliary surgery have been known to present with haemorrhage from T-tubes (Quattella, 1954).

The diagnosis of hepatic artery aneurysm is difficult to make, usually requiring a high index of clinical suspicion and angiography (Cohen *et al.*, 1966; Countryman *et al.*, 1983; Guida & Moore, 1966; Shaw, 1982). Plain abdominal X-ray may reveal a ring of calcification in the upper abdomen and barium studies may show a deformed duodenum due to an extrinsic mass (Shaw, 1982). Ultrasound scan and CT scan will

often detect the mass but, as in our patient, may not give sufficient information to make the diagnosis.

With regard to treatment, hepatic artery aneurysms have traditionally been divided into two groups – those proximal to the gastroduodenal artery and those distal to it. It is generally accepted that a proximal aneurysm can be safely ligated with or without excision, without risk of liver necrosis. Arterial blood reaches the liver from the superior mesenteric artery via the inferior pancreaticoduodenal artery, superior pancreaticoduodenal artery, gastroduodenal artery and thence the hepatic arteries. This collateral pathway is not available if ligation is distal to the gastroduodenal artery, and over the years many methods have been devised to restore the flow of arterial blood to the liver. These are, as listed by Countryman *et al.* (1983), reconstructive endoaneurysmorrhaphy, excision with end-to-end anastomosis, excision with splenohepatic anastomosis, excision with saphenous vein graft reconstruction, excision with prosthetic graft reconstruction, suturing of aneurysm to the liver, wrapping of the aneurysm with cellophane, wiring of the aneurysm and hepatic lobectomy. However, there is evidence that ligation of the hepatic artery distal to the gastroduodenal artery in fact carries little risk of hepatic ischaemia because adequate oxygenation is provided by other collateral channels, aberrant vessels and increased extraction of oxygen from the portal vein blood (Madding & Kennedy, 1972).

Brittain *et al.* (1964) reported five patients in whom the common hepatic artery or right hepatic artery had been injured, and reviewed previous publications. They concluded that 'ligation of the hepatic artery or one of its branches in the patient with relatively normal hepatic function is not ordinarily fatal in the otherwise uncomplicated case.' This is supported by the fact that ligation of the hepatic artery is a recognized treatment for metastatic liver tumours and liver necrosis does not appear to occur (Murray-Lyon *et al.*, 1970).

Michels (1960) in his dissection of 200 livers, found that the vessels conformed to the standard textbook description in only 55% of people. In the remaining 45% there were collateral vessels and he described 26 possible collateral pathways. Within four days of arterial flow being interrupted collateral flow occurs via phrenic and intercostal arteries, the opposite lobe and the falciform ligament (Madding & Kennedy, 1972). In addition, splanchnic arteriovenous communications increase the blood flow through the portal vein and raise its oxygen concentration. Under normal conditions the portal vein supplies 50% of the liver's oxygen.

Selective percutaneous embolization using Gelfoam has been used in the treatment of hepatic artery aneurysms and the results appear to be encouraging. This procedure has the advantage that it can be

performed under local anaesthetic and at the same time as the diagnosis is made at angiography (Goldblatt *et al.*, 1977).

The treatment of choice for hepatic artery aneurysm, therefore, is simple proximal ligation or ligation with excision irrespective of whether the aneurysm is proximal or distal to the gastroduodenal

artery. Simple measures such as avoiding hypotension or hypoxia, using fresh blood for transfusion and resting the bowel for a few days post-operatively should render the small possibility of liver necrosis even more remote. Selective embolization may prove to be a useful alternative treatment especially in a patient unfit for surgery.

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