Recurrent acute pancreatitis due to haemobilia from a hepatic artery aneurysm

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Summary

A case of recurrent acute pancreatitis due to haemobilia secondary to a bleeding hepatic artery aneurysm is presented. Embolization of the hepatic artery resulted in cessation of bleeding and resolution of pancreatitis.

KEY WORDS: recurrent pancreatitis, haemobilia, hepatic artery aneurysm.

Introduction

Haemobilia remains an uncommon cause of gastrointestinal haemorrhage. Trauma accounts for the majority of cases although other causes are inflammatory (pyogenic and helminthic), cholelithiasis, vascular malformations, pancreatitis and neoplasms. Patients usually present with the classical triad of melaena, biliary colic and jaundice, although fever, a palpable right upper quadrant mass and shock may also occur (Sandblom, 1972).

The presence of acute pancreatitis associated with haemobilia was first noted by Dean and Falconer (1912) in their report of the post-mortem findings in a young man with a hepatic artery aneurysm. Subsequently, the occurrence of acute pancreatitis following an episode of bleeding into the biliary system has been rarely noted (Caroli, 1959; Kaplan *et al.*, 1980) although the converse, bleeding into the biliary system as a result of pancreatitis and rupture of a pseudocyst, is well recognised (Brintnall, Laidlaw and Papp, 1974; Ro, Yoon and Puppula, 1976). We report here a patient with recurrent acute pancreatitis and haemobilia secondary to a hepatic artery aneurysm which was successfully embolized at angiography.

Case report

A 74-year-old woman was admitted with a 2-week

history of malaise, lethargy and anorexia and a 4-day history of vomiting. Following one episode of vomiting, she had brought up fresh blood clots. Two days before admission, she complained of a continuous aching pain in the right hypochondrium which persisted for 24 hr.

On examination, the patient was well-nourished, pyrexial 38.2°C and jaundiced. The abdomen was soft with no palpable masses. Rectal examination showed no melaena.

Investigations showed haemoglobin 12.3 g/dl, white cell count 22.4×10^9 /litre, erythrocyte sedimentation rate 43 mm/hr, bilirubin 110 μ mol/litre, alanine aminotransferase 627 iu/litre (normal <35), aspartate aminotransferase 825 iu/litre (normal <35), alkaline phosphatase 1116 iu/litre (normal range 100-280), amylase 4420 iu/litre (normal range 70-300).

A presumptive diagnosis of acute pancreatitis secondary to choledocholithiasis was made and the patient treated with bowel rest and intravenous fluids.

Over the next few days, her pain and vomiting settled and the jaundice cleared. The serum amylase returned to normal although her liver function tests remained cholestatic. Ultrasound showed a dilated common bile duct and intrahepatic ducts, swollen head of pancreas but no gall stones. One week after admission, she again developed fever and right hypochondrial pain and her amylase went up to 3910 iu/litre. This episode rapidly settled and the amylase returned to normal.

Ten days after admission, her haemoglobin had dropped to 9.5 g/dl with normal indices which was attributed to her pyrexial illness. Fourteen days after admission, she again vomited with blood clots. There was no melaena but her haemoglobin dropped to 7.1 g/dl and the amylase rose to 9380 iu/litre. A repeat

ultrasound showed no pseudocyst and endoscopic retrograde cholangiopancreatography (ERCP) demonstrated a normal papilla with no evidence of the recent passage of a stone. There was a common channel and no filling of the duct of Santorini from the pancreatic duct. The common bile duct and intrahepatic ducts were grossly dilated with no filling defects and the gall bladder could not be filled, suggesting the possibility of a stone impacted in Hartmann's pouch. Following ERCP, she passed a melaena stool and next day underwent laparotomy. The pancreas was found to be oedematous and inflamed. The gall bladder contained 2 small stones and was removed. The common bile duct was tense and distended and contained blood clots. Operative choledochoscopy revealed fresh bleeding arising from the right intrahepatic duct. A wedge liver biopsy showed changes consistent with resolving large duct obstruction.

Postoperatively, she continued to bleed and underwent hepatic arteriography which demonstrated an aneurysm arising from a branch of the right hepatic artery (Fig. 1). This was embolized using isobutyl-2-cyanoacrylate tissue glue with no ill effects (Fig. 2). Four months later, she remains well and her liver function tests have returned to normal.

FIG. 1. Selective hepatic arteriogram (arterial phase) showing 1 cm aneurysm arising from the right hepatic artery (arrowed).

FIG. 2. Post-embolization study showing complete occlusion of the right hepatic artery. Flow to the left hepatic artery and gastroduodenal artery are seen to be unimpaired.

Discussion

Although the classical triad of melaena, biliary colic and jaundice as a manifestation of haemobilia is well-recognised, the diagnosis is often overlooked (Sandblom, 1972). The subject of this case report presented with jaundice and acute pancreatitis. Although she had 2 episodes of haematemesis following episodes of vomiting, in the absence of melaena or biliary colic, haemobilia was not suspected. Laparotomy revealed gall stones which are themselves known to cause haemobilia. This is usually microscopic, although erosion of the cystic artery can lead rarely to massive bleeding (Zederfeldt, 1967). Similarly pancreatitis, particularly when associated with a pseudocyst may result in haemobilia (Ro *et al.*, 1976). In this case, endoscopic cholangiography using dilute contrast demonstrated a normal papilla with a dilated bile duct which contained no filling defects. There was no pancreatic pseudocyst. It seems, therefore, that despite the absence of classical symptoms, which led to a delay in diagnosis, the patient had intermittent bleeding from a hepatic artery aneurysm which resulted in recurrent episodes of acute pancreatitis over the course of her admission.

Kaplan *et al.* (1980) reported 2 cases of haemobilia caused by bleeding hepatic artery aneurysms which resulted in acute pancreatitis and proposed that the presence of a common channel for both pancreatic

and biliary ducts with no accessory duct drainage predisposed to the development of acute pancreatitis due to blockage of the pancreatic duct by blood clot. It is therefore of interest that our patient was shown at ERCP to have a common channel with no accessory duct visible.

The aetiology of the hepatic artery aneurysm is unknown although they are associated with gall stones in a significant number of cases. Trauma, systemic infection, polyarteritis nodosa and arteriosclerosis account for the majority of the remainder (Guida and Moore, 1966). The use of angiography and therapeutic embolisation is an effective method of managing some patients with haemobilia (Fagan *et al.*, 1980) and in this patient prevented further haemorrhage and pancreatitis.

Physicians and surgeons should therefore remain aware of haemobilia as a cause for jaundice and pancreatitis, particularly when investigations fail to reveal stones present within the common bile duct or there is evidence of gastrointestinal blood loss.

Acknowledgments

We are indebted to Dr D. J. Allison for Figs. 1 and 2 and to

Dr M. W. McNicol for permission to report details of the patient who was under his care.

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(Accepted 4 January 1983)