CHRONIC PANCREATITIS WITH SPLENIC VEIN OCCLUSION AND RECURRENT VARICEAL HAEMORRHAGE

Y T Wang
W Y Cheong
A Heng
R K Kwok
S C Poh

Department of Medicine III
Tan Tock Seng Hospital
Moulmein Road
Singapore 1130

Y T Wang, MBBS, M Med (Int Med), MRCP (UK), AM,
Senior Registrar

S C Poh, AM, MBBS, FRCPE,
Senior Physician and Head

Department of Radiology
Singapore General Hospital
Singapore 0316

W Y Cheong, MBBS,
Medical Officer

Department of Radiology
Tan Tock Seng Hospital

R K Kwok, MBBS, DMRD, FRCR,
Registrar

Department of Surgery
Tan Tock Seng Hospital

A Heng, MBBS, AM, FRCSE, FRCS,
Senior Surgeon and Head

SYNOPSIS

We report a 49 year old man with chronic pancreatitis, splenic vein occlusion and recurrent variceal haemorrhage.

INTRODUCTION

Splenic vein occlusion with gastro-oesophageal varices is uncommon and therefore not usually considered in the differential diagnosis of gastro-intestinal bleeding. Further, extrahepatic portal hypertension resulting from chronic pancreatitis is even more uncommon. Of 164 patients with extrahepatic portal hypertension in the Mayo Clinic over a period of 20 years, only 5 had pancreatitis as a sole predisposing disease (1). We report a 49 year old man with chronic pancreatitis complicated by splenic vein occlusion resulting in recurrent variceal haemorrhage.

CASE REPORT

In July, 1972, a 39 year old Chinese man was admitted to this hospital for haematemesis and melaena. He had been treated for a left sided pleural effusion with anti-tuberculous drugs a year before. The liver was palpable 1 fingerbreadth beneath the right costal margin. The spleen was not palpable. Investigations showed: haemoglobin 4.5g/dl, total white count 4.8×10^9/1, serum glutamate-pyruvate transaminase 220 U/L, serum alkaline phosphatase 7.2 A units, serum albumin 33 g/1, serum globulin 32 g/1 and serum bilirubin 0.6 mg/dl. He was transfused to a haemoglobin of 9.6 g/dl. Barium meal studies showed no abnormalities in the stomach and duodenum. A few gallstones were present.

Apart from a brief admission for epigastric pain in July 1976, he was not seen until May 1982 when he returned complaining of anorexia, weight loss and epigastric discomfort for 1 year. He was now 49 years old and had developed diabetes mellitus 3 years earlier. He was a heavy smoker and drank alcohol regularly. He appeared very thin and pale. There was finger clubbing. The liver was palpable at 2 cm. from the right costal margin. The spleen was not palpable. His stools were melaenic. Investigations revealed: haemoglobin 2.8 g/dl, hypochromic microcytic erythrocytes on smear, serum iron 25 ug/dl, total iron binding capacity 380 ug/dl, serum folate 4.7 ug/1, serum B_{12} 320 ng/1, 2 hour post prandial blood sugar 295 mg/dl. With blood transfusion, he felt better and refused further investigations. He was discharged with tolbutamide and metformin.

In August 1982, he required transfusion again for severe anaemia (haemoglobin 3.6 g/dl). Total white count was 11×10^9/1 and platelet count 210×10^9/1. Upper gastro-intestinal endoscopy only revealed stale blood in the duodenum. Cimetidine was empirically started. Barely two weeks later, he had melaena again and a repeat endoscopy showed nothing remarkable in the oesophagus, stomach or duodenum. Barium enema also failed to disclose any abnormalities. He refused consent for visceral angiography.

In the next few months, he required transfusions so frequently (as often as once in 2-3 weeks) that he consented to angiography. Further investigations: serum glutamate-pyruvate transaminase 29 U/L . serum alkaline phosphatase 164 IU/L, serum albumin 42 g/l, total protein 58 g/l, blood sugar 474 mg/dl; D-xylose absorption test — 1.9 g (first 2 hours), 0.08 g (next 3 hours), 2.7 g (total 5 hours). Levels for blood urea, serum electrolytes, serum creatinine, serum carotene, serum calcium, serum phosphate, prothrombin time and partial thromboplastin time were normal. Coeliac and superior mesenteric arteriograms showed gastric varices, splenic vein occlusion, a patent portal vein and pancreatic calcification. (Fig. 1) There was a questionable tumour blush in the body of the pancreas. Oesophageal varices were absent. Laparotomy on 8.2.83 revealed a normal liver, a slightly enlarged spleen, gastric varices, a diffusely hard and nodular pancreas with a grossly dilated pancreatic duct and multiple gallstones. No gastric or duodenal ulcers were present. Some gastric erosions were seen. Splenectomy and distal pancreatectomy together with a cholecyctectomy, truncal vagotomy and pyloroplasty were performed. As the pancreatic duct was obstructed near its junction with the duodenum, the pancreas was drained with a pancreatico-jejunostomy Roux-en-Y. Biopsies of the liver and jejunum were taken. Microscopic examination showed the following: mild inflammatory infiltrate in the spleen; chronic cholecystitis; normal liver architecture with some congestion; normal jejunal histology; features of chronic pancreatitis with marked destruction and fibrosis of pancreatic parenchyma and ductular proliferation and dilatation. Postoperatively he recovered satisfactorily and was sitting at the bedside eating a semisolid diet by the 12th day. On the 13th day after surgery, he sustained a massive myocardial infarction with hypotension. He died a few days later on 25.2.83.

DISCUSSION

Splenic vein occlusion may be due to a variety of pancreatic pathologies including carcinoma of the pancreas, acute pancreatitis, chronic pancreatitis and pancreatic pseudocyst. (2, 3, 4, 5, 6, 7) As a result of the obstruction, blood from the spleen would have to be diverted hepatopetally or hepatofugally. To circumvent the block hepatopetal flow occurs retrogradely through the short gastric veins into the fundal plexus and thence into the coronary and portal veins, or through the left and right gastroepiploic veins into the portal vein. Hepatofugal collaterals lead to drainage into the systemic veins through the short gastric-gastrooesophageal and inferior mesenteric-haemorrhoidal pathways. (Fig. 2) When these channels fail to decompress the choked segment, portal hypertension ensues with consequent varices and splenomegaly. The varices tend to be gastric than oesophageal (6). Ruptured submucosal varices cause intraluminal gastro-intestinal bleeding whereas subserosal ones give rise to intraperitoneal haemorrhage. (7, 8) Although gastric varices may occasionally mimick gastric carcinoma on barium studies (9), they are often not evident either on radiology or endoscopy (3, 5, 10, 11). They are best demonstrated by splenoportography or transarterial portography (5). When variceal haemorrhage occurs in isolated splenic vein thrombosis splenectomy alone is adequate treatment (1, 3, 10). Indeed portosystemic shunt surgery is contraindicated as it may fail to decompress the hypertensive segment while exposing the patient unnecessarily to the risk of encephalopathy. The prognosis after splenectomy for splenic vein thrombosis and gastro-intestinal haemorrhage is very good underscoring the importance of differentiating this form of portal hypertension from the rest.

Like many of the reported cases, our patient had recurrent gastro-intestinal haemorrhage, virtually

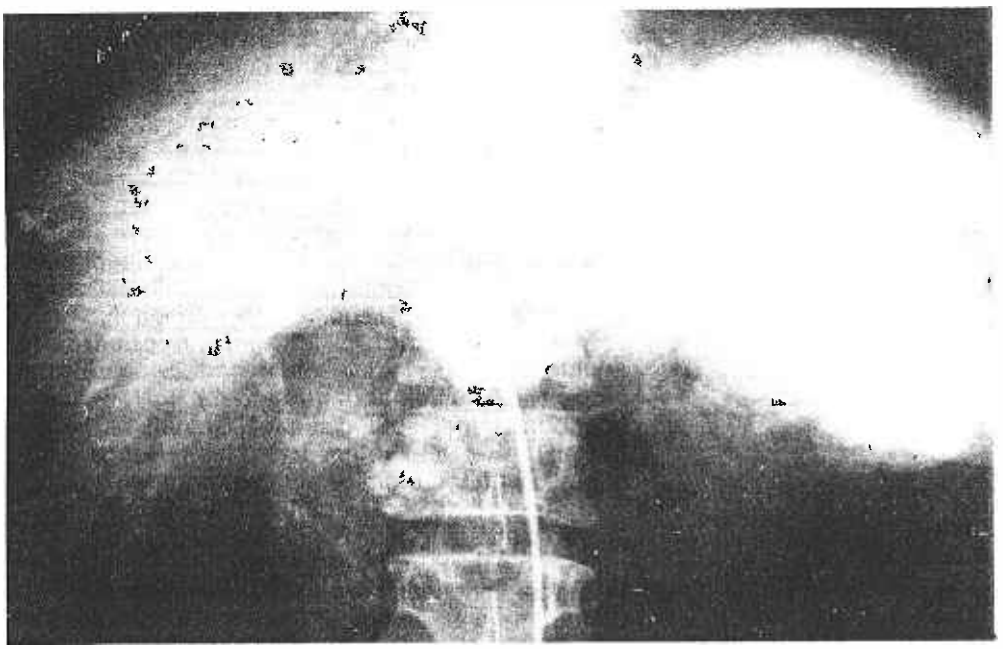

Fig. 1 Transarterial portovenogtam showing patent portal vein with segmental splenic vein occlusion. Note pancreatic calcifications and radioopaque gallstones.

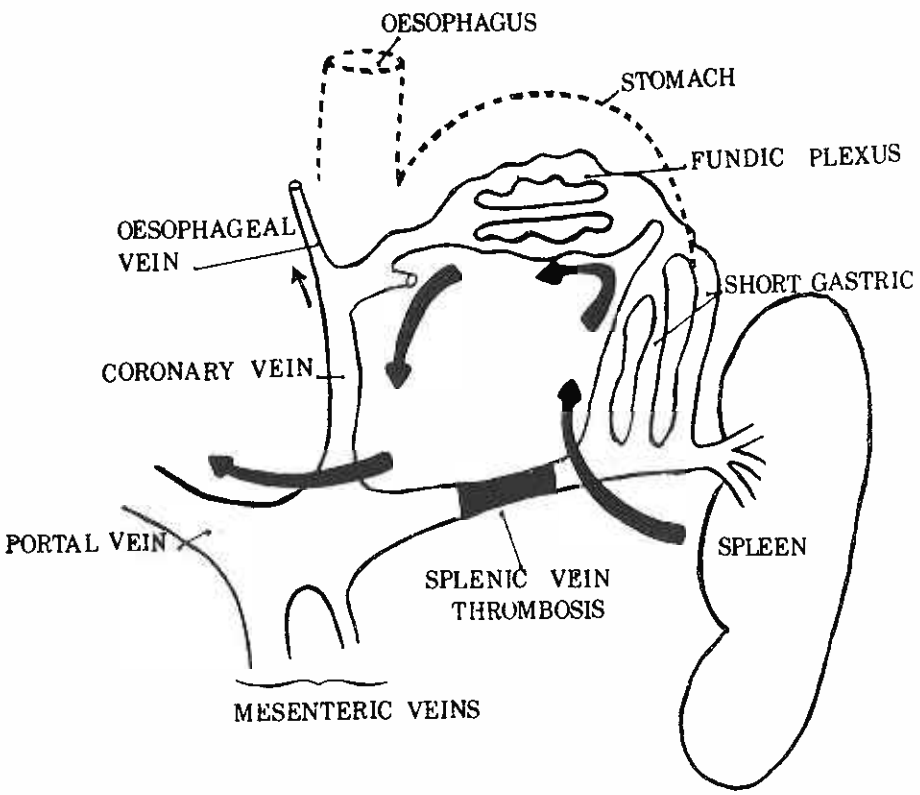

Fig 2 Venous flow in splenic vein thrombosis

normal liver function and histology, normal barium studies, non specific findings on repeated upper gastro-intestinal endoscopies, and chronic pancreatitis. Transarterial portography demonstrated isolated splenic vein occlusion with gastric varices. It was unfortunate that he died of a myocardial infarction.

Ninety-five patients with chronic pancreatitis were studied by Rosh and Herfort (12). They found various degrees of distortion and occlusion of the splenic vein in 89. Using splenoportography, Rignault et al (11) studied 20 patients with chronic pancreatitis of which 14 showed splenic vein obstruction of varying severity. We would therefore expect chronic pancreatitis to be a prominent cause of extrahepatic portal hypertenstion and haemorrhage. However, of 164 Mayo Clinic patients with extrahepatic portal hypertension, Longstreth, Newcomer and Green found only 5 with chronic pancreatitis as the sole predisposing disease. Therefore although chronic pancreatitis frequently causes distortion or even obstruction of the splenic vein, overt extrahepatic portal hypertension with splenomegaly and variceal bleeding is very uncommon.

In conclusion, we have described a case with chronic pancreatitis, splenic vein occlusion and variceal haemorrhage. We emphasize the importance of diagnosing this form of segmental portal hypertension because of its good prognosis with splenectomy. It should be considered in any patient with unexplained recurrent gastro-intestinal haemorrhage especially when an underlying pancreatic pathology is present.

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