Chronic small bowel ischaemia presenting as chronic pancreatitis

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Summary

A patient presenting with abdominal pain was initially thought to have chronic pancreatitis. Investigation revealed normal pancreatic structure but an indirect test of exocrine function showed low enzyme activity. The true diagnosis of chronic small intestinal ischaemia was demonstrated angiographically and confirmed at laparotomy. The early distinction between chronic pancreatitis and chronic small intestinal ischaemia is important because ischaemia may be the harbinger of acute and possibly fatal bowel infarction. Direct stimulation tests of pancreatic function showing normal results should turn attention to the possibility of small bowel ischaemia.

Introduction

Chronic pancreatitis and chronic small intestinal ischaemia are uncommon conditions where pain is usually the presenting symptom. The case now reported illustrates how confusion can arise between these two disorders and suggests a means of achieving an earlier diagnosis of ischaemia.

Case report

A 49-year-old man, insulin-dependent diabetic for 12 years, was referred with a 3-month history of severe epigastric pain and weight loss of 6.35 kg. The pain was unrelieved by antacids, unrelated to food, but eased by leaning forwards.

On examination he was in sinus rhythm, the blood pressure was 130/80 mmHg, there were xanthelasmas around both eyes and the posterior tibial pulses were absent. Abdominal examination was negative.

Barium meal, endoscopy and abdominal ultrasound examination were unhelpful. The fasting lipid profile was normal. The Lundh test revealed low trypsin activity at 2.8 $\mu\text{Eq H}^+/\text{ml}/\text{min}$ (normal 5-26) and fat absorption, measured by a dual isotope technique (Nelson, MacKenzie and Russell, 1980) was reduced at 38.6% (normal >95%) with a raised faecal fat of 84.2 mmol/24 hr (normal <21 mmol/24 hr). Jejunal biopsy and CT scan of

the pancreas were normal. A provisional diagnosis of chronic pancreatitis was made and the patient managed conservatively with pancreatic supplements, antacids and analgesics.

Progress was poor, his pain became increasingly more severe and he was referred for possible pancreatic surgery. Endoscopic retrograde cholangiopancreatography showed a normal duct system and a routine pre-operative free flow aortogram showed no filling of the coeliac axis (CA) or superior mesenteric artery (SMA) in the early films, although later films demonstrated paravertebral and marginal artery collateral circulations and faint filling of the SMA (Fig. 1).

FIG. 1. Free flow aortogram at 4 sec showing delayed filling of the superior mesenteric artery (SMA), and collateral circulation via an enlarged marginal artery (MA) arising proximal to an occlusion of the inferior mesenteric artery (IMA).

Laparotomy, performed one year after the initial presentation, revealed a purplish discolouration of both large and small bowel with absent mesenteric pulsation. The origin of the SMA was a hard cord with pulsation distal to the origin of the inferior pancreaticoduodenal artery and the CA was thought to be congenitally small. Severe aortic disease at the origin of the vessels precluded endarterectomy and an 8-mm knitted dacron graft was inserted between aorta and SMA. The result was an immediate improvement in intestinal circulation.

Postoperatively the pain resolved, he regained weight but had troublesome diarrhoea for 12 weeks. Fat absorption and trypsin activity, measured 4 months after revascularization, returned to normal (97.6% and 5.6 $\mu\text{Eq H}^+$ ml/min respectively). Insulin requirements were unchanged.

Discussion

The initial diagnosis of chronic pancreatitis was based on the description of the pain, fat malabsorption and reduced trypsin activity. Although demonstrable impairment of small bowel absorptive function is not a constant feature of small intestinal ischaemia (Marston, 1977), it has been documented (Tilson and Stansel, 1976) and is a recognized source of error in the Lundh test (James, 1973). The pancreas itself was not obviously affected by ischaemia as judged by its normal appearance at laparotomy and the unchanged requirement for insulin postoperatively. Abnormalities of villous

architecture have been observed in chronic intestinal ischaemia (Juergens, Spittell and Fairbairn, 1980) but this is not always the case and the jejunal biopsy in this instance was normal, angiography giving the correct diagnosis. No histological information as to the type of arterial disease was obtained although atheroma would have been the most likely.

When chronic pancreatitis is suspected, the findings of normal gland structure and abnormal indirect pancreatic function tests in a clinical setting of arterial disease raise the possibility of small intestinal ischaemia. Further investigation with a direct stimulus to the pancreas, such as the secretin-pancreozymin test and possibly angiography, might provide an earlier diagnosis of ischaemia and prevent the development of acute and often fatal infarction (Dunphy, 1936).

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