Stenosis of the colon following acute pancreatitis

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Summary

Two patients who had developed incomplete large bowel obstruction were shown to have colonic stenoses on barium enema. At laparotomy, inflammatory masses secondary to acute pancreatitis were found in both patients, though pancreatitis had not been suspected clinically. Resection of affected bowel in such cases is contra-indicated and the stenoses usually resolve spontaneously.

Introduction

Acute pancreatitis is followed by complications in between 12% (Trapnell, 1971) and 38% (Lukash, 1967) of cases. Colonic complications are rare but include spasm (Schwartz and Nadelhaft, 1957), stenosis with or without obstruction (Miln and Barclay, 1952), pancreatico-colonic fistula (Berne and Edmondson, 1966), necrosis (Katz, Dorman and Aufses, 1974) and perforation (Gatch and Brickley, 1951). Colonic stenosis following acute pancreatitis was first described by Forlini in 1927 and three cases have subsequently been reported from Great Britain.

Case reports

Case 1

A 62-year-old man presented with a 2 month history of weight loss, constipation and epigastric discomfort. He was pyrexial (38.7°C) and had a palpable mass in the left hypochondrium. His haemoglobin was 11.0 g/dl, leucocyte count $12.3 \times 10^9/l$, ESR 30 mm/hr and serum amylase concentration 100 Somogyi units. Seven plasma calcium estimations performed over a 3-week period gave results ranging from 2.85 mmol/l to 3.15 mmol/l.

The patient passed a solitary bloody stool and on barium enema was reported as having a 'ring carcinoma' at the splenic flexure (Fig. 1). At laparotomy 7 days after admission, a sclerotic inflammatory mass was found in the region of the pancreatic tail. Its resection en bloc necessitated a left hemi-colectomy, splenectomy, distal pancreatectomy and resection of the first loop of jejunum. No intrinsic lesion was found in either the colon or the

jejunum histologically but the pancreas was fibrotic and chronically inflamed, containing several abscess cavities. Fat necrosis, arteritis and fibrosis were present in the surrounding tissues. The patient died from septicaemia on the fifteenth post-operative day.

Case 2

A 38-year-old man presented with a 3-week history of hypogastric colic, constipation and abdominal distension. He had been intolerant of fatty food for 6 months. His haemoglobin was 11.1 g/dl, leucocyte count $12.0 \times 10^9/l$ and ESR 87 mm/hr. Serum amylase was not estimated and the biochemical profile was normal. Plain abdominal X-ray failed to confirm the preliminary diagnosis of intestinal obstruction.

The leucocyte count rose to $16.7 \times 10^9/l$ during the next 5 days, when an epigastric mass became palpable. Barium enema (Fig. 2) showed a stenotic segment 10 cm long in the transverse colon, felt to be due to external compression. The duodenum was widened on barium meal. Laparotomy, performed under glucagon cover, revealed an inflammatory mass fused to the under-surface of the liver, involving gall-bladder, transverse colon, greater omentum, stomach and duodenum. Surgery was restricted to omental biopsy and drainage of a large volume of blood-stained peritoneal fluid.

The omental biopsy showed extensive fat necrosis, arteritis and early fibrotic changes. Prolonged ileus and hypocalcaemia were the main features of a protracted postoperative course. Daily amylase estimations ranged from 450 to 900 Somogyi units, finally returning to normal on the tenth day. Barium enema, performed 3 months later, showed that the stenosis had completely resolved. Oral cholecystography at this time revealed a non-functioning gall bladder.

Discussion

There have been 19 previous reports of colonic stenosis following acute pancreatitis (Forlini, 1927; Baylin and Weeks, 1944; Remington, Mayo and Dockerty, 1947; Miln and Barclay, 1952; Rose, 1953; Price, 1956; Schwartz and Nadelhaft, 1957;

FIG. 1. Barium enema in case 1, reported as showing a 'ring carcinoma' at the splenic flexure.

FIG. 2. Barium enema in case 2, showing a 10 cm stenosis of the transverse colon, thought to be due to external compression.

Bolam, 1960; Aronson and Davis, 1961; Lukash and Bishop, 1967; Bruno and Ober, 1971; Mohiuddin *et al.*, 1971; Lindhal, Vejlsted and Backer, 1972; de Ford and Kolts, 1973; Agrawal *et al.*, 1974; Theodoropoulos, Archimandrites and Kalos, 1975; Hunt and Mildenhall, 1975; Mair, McMahon and Goligher, 1976; Thompson, Kelvin and Reed, 1977), describing 28 patients of whom 24 were male.

The pathogenesis is complex (Schwartz and Nadelhaft, 1957) but most authors have attributed the stenoses to the formation of inflammatory adhesions around the colon. This was the main feature of the two cases described above. The proximity of the splenic flexure of the colon to the tail of the pancreas and the anatomy of the peritoneal reflexions of the transverse mesocolon make it likely that these structures would become involved in acute pancreatitis. It is indeed surprising that colonic complications do not occur more frequently.

The aetiology of such colonic stenoses is rarely evident preoperatively. Of 26 adequately documented cases, 12 presented with symptoms attributable to pancreatitis but 13 presented with symptoms referable to the colon and one with acute cholecystitis. The patients reported here both presented with a clinical picture of colonic disease and pancreatitis was only diagnosed at operation. Though 74% of reported patients had a raised serum amylase concentration, this investigation may be omitted in patients presenting with colonic disease.

The condition may be managed in one of two ways, depending upon whether pancreatitis is identified as the cause pre-operatively. In 13 patients pancreatitis was diagnosed pre-operatively. Elective operation for colonic stenosis should be postponed in these circumstances (Mohiuddin *et al.*, 1971), with a second barium enema being performed at 6 weeks. This policy has only been followed twice but in these cases the stenoses resolved spontaneously. Should the stenosis persist, the possibility of malignant disease or of chronic stricture formation would demand a laparotomy.

An early operation was necessitated in 4 cases by large bowel obstruction and in 5 by an undiagnosed acute abdomen. In a further 7 cases the colonic stenosis simulated a carcinoma and pancreatitis was not suspected before operation. When the diagnosis of post-pancreatic colonic stenosis is made at laparotomy, resection of the stenosed segment should be avoided. Freeing of adhesions is desirable as it may prevent dense stricture formation but biopsy and peritoneal lavage have been followed by resolution in all surviving cases. Defunctioning of obstructed bowel may be required. Should the stenosis not resolve after a conservative procedure, a second, planned, operation will carry less risk than bowel resection in the presence of active pancreatitis,

which, as in the first case above, is associated with septic complications and a 50% mortality.

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