Stenosis of the colon following acute pancreatitis

STEPHEN BREARLEY
M.A., M.B., B.Chir., F.R.C.S.

DUNCAN J. CAMPBELL
B.Sc., M.B., F.R.C.S., F.R.C.S. (Edin.)

Department of Surgery, Selly Oak Hospital, Birmingham 29

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 x 10^9/l and 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 hemicolectomy, splenectomy, distal pancreatectomy and resection of the first loop of jejunum. No intrinsic lesion was found in either the colon or the jejum 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 septicemia 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 x 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 x 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;
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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; Mohiudd- 
din et al., 1971; Lindhal, Veiulsted 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 pan-
creatitis. It is indeed surprising that colonic com-
plications 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 refer-
able 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 con-
centration, 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 iden-
tified 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 spontane-
ously. 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-pancreaticitic 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,
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