Massive gastric distension in acute pancreatitis—a report of two cases

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Summary

Massive gastric distension was diagnosed in 2 patients with acute pancreatitis. Both showed signs of severe peripheral circulatory failure, and responded well to intravenous fluid replacement and nasogastric aspiration. A review of the literature revealed only 2 previously reported cases. The possible mechanisms linking the pathogenesis of these 2 conditions are discussed.

KEY WORDS: gastric distension, acute pancreatitis, superior mesenteric artery syndrome.

Introduction

Acute pancreatitis remains a condition with significant mortality and morbidity. Factors which can delay or prevent the correct diagnosis therefore assume great importance. One such factor is co-existing massive gastric distension, a condition in which the gross abdominal and radiological signs may mask the presence of pancreatic inflammation. Two such cases have previously been reported, where a perforated intra-abdominal viscus was suspected and the patient underwent laparotomy. Two further cases of acute pancreatitis and massive gastric distension are reported below.

It seems reasonable to conclude that in all cases of massive gastric distension the possibility of co-existing acute pancreatitis should be entertained, and the serum amylase concentration measured at the earliest opportunity.

Case reports

Case 1

A 15-year-old mentally retarded girl was admitted following 3 days of vomiting associated with increasing abdominal distension. Her permanently severe degree of motor disability excluded the possibility of ingested foreign material as a source of her abdomi-

nal problems. On examination, she was dehydrated and tachypnoeic with a pulse rate of 180/min and a systolic blood pressure of 60 mmHg. Her abdomen was grossly distended and tympanitic, with marked tenderness in the epigastrium. Bowel sounds were infrequent. Supine abdominal and chest radiographs showed gross distension of the stomach and severe spinal scoliosis (Fig. 1). The amylase level on admission was 2400 iu/litre. An intravenous (i.v.) infusion was commenced and nasogastric aspiration yielded 1,200 ml of brownish fluid. She made a steady recovery over the following 10 days.

FIG. 1. Supine plain abdominal X-ray showing massive gastric distension and severe spinal scoliosis (case 1).

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Case 2

A 47-year-old woman was admitted with a 3-day history of increasing epigastric pain radiating through to the back. She had vomited repeatedly during this period. On examination she was dehydrated and in severe pain, with a pulse rate of 110/min and a blood pressure of 90/60 mmHg. Her abdomen was markedly distended and tympanitic, with generalized tenderness, maximal in the epigastrium. Bowel sounds were infrequent. Supine abdominal and erect chest radiographs showed massive distension of the stomach with a dilated duodenum (Fig. 2). The amylase level on admission was 820 iu/litre. An i.v. infusion was commenced and nasogastric aspiration yielded 2 litres of brownish fluid. She made a steady recovery over the following 8 days. Although the initial amylase level is not diagnostic of acute pancreatitis, the clinical presentation, the rapid decrease in amylase levels, and subsequent abdominal radiographs showing some residual stasis in the proximal small bowel, all support the diagnosis. It is likely that the peak amylase levels occurred before admission, due to the relatively late presentation.

FIG. 2. Supine plain abdominal X-ray showing massive gastric distension, and dilated duodenum.

Discussion

The complications and sequelae of acute pancreatitis are well documented. Paradoxically, considering the anatomical proximity of the organ, those involving the gastro-intestinal tract itself are relatively rare. Certainly, the association between acute pancreatitis and compression of the third part of the duodenum by the superior mesenteric artery is recognized. Wilkie (1921) reported a case of chronic duodenal ileus (now known as superior mesenteric artery syndrome) and pancreatitis. Simon and Lerner (1962) described the case of a 52-year-old man with acute pancreatitis and duodenal compression by the superior mesenteric artery. They postulated that thickening of the mesentery in acute pancreatitis can be responsible for arterio-mesenteric occlusion of the duodenum.

The link between gastric distension and superior mesenteric artery syndrome is also recognized. Robinson (1900) noted dilatation of the stomach from pressure of the superior mesenteric artery on the duodenum. Conner (1906) reported the presence of dilatation of the duodenum proximal to its contact with the superior mesenteric vessels in two-thirds of a large series of cases of acute dilatation of the stomach. Bloodgood (1907) reported two cases of acute dilatation of the stomach and duodenum proximal to the site of duodenal compression by mesentery. There exists, therefore, a possible sequence whereby acute pancreatitis might cause compression of the duodenum and gastric distension. There are two previously reported cases in the literature where massive gastric distension co-existed with acute pancreatitis. In one there was documentary evidence of compression of the duodenum by the superior mesenteric artery. Keane, Fennell and Tomkin (1978) reported the case of a 16-year-old girl who suffered from anorexia nervosa. Upon refeeding she developed severe upper abdominal pain and vomiting. She had generalized abdominal tenderness and guarding. Bowel sounds were absent. Plain abdominal X-ray showed gastric distension with dilatation of the proximal duodenum. As a perforated viscus was suspected laparotomy was performed. At operation she was found to have acute pancreatitis and massive distension of the stomach. Two months after the operation she was still unable to take oral feeds properly. Hypotonic duodenography showed a narrowed duodenum. A second laparotomy revealed a dilated duodenum as far as the crossing of the superior mesenteric artery. A duodenojejunal anastomosis was performed. Following this she made a slow recovery. Mikhailichenko (1980) reported the case of a 13-year-old girl who presented with severe abdominal pain and distension, and signs of peripheral circulatory failure. Bowel sounds were absent. A diagnosis of a perforated viscus was made.

At laparotomy, the stomach was grossly distended and the pancreas showed signs of acute pancreatitis. She made a slow postoperative recovery. The two further cases reported in this paper reinforce this association between acute pancreatitis and massive gastric distension. In the first case the presence of severe spinal scoliosis would certainly have predisposed the patient to superior mesenteric artery syndrome.

An alternative possible pathogenetic sequence linking acute pancreatitis and gastric distension exists. If duodenal compression by the superior mesenteric artery was the initial event, then gastric distension might follow. The raised intra-duodenal pressure might cause passage of duodenal contents retrogradely along the pancreatic duct causing pancreatitis. McCutcheon and Race (1962) showed, experimentally in dogs, that duodenal contents under pressure can be forced past normal pancreatic duct papillae causing pancreatitis. Certainly, whatever the underlying aetiological mechanism, the association between acute pancreatitis and massive gastric distension is an important one. The combination of inflammatory losses due to the pancreatitis, and the rapid flux of body fluids in massive gastric distension is almost bound to lead to severe peripheral circulatory failure. Difficulties in diagnosis exist due to the florid physical signs which, understandably, prompted laparotomy in the 2 previously reported cases.

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