CASE REPORT

Acute Pancreatitis Occurring in Gastric Aberrant Pancreatic Tissue


Virtually all known diseases of pancreatic tissue have been reported to occur within the gastric wall when aberrant pancreatic tissue is present at that site (1). When we reviewed the literature, however, we were able to find only one report (2) of histologically proven acute pancreatitis in gastric aberrant pancreatic tissue.

This case report is presented to document such an association. The patient presented as a diagnostic problem with severe abdominal pain and a fluctuating epigastric mass, which proved to be due to acute pancreatitis in gastric aberrant pancreatic tissue.

CASE REPORT

A 38-year-old Caucasian male was admitted to hospital with a 6-day history of severe postprandial epigastric pain associated with anorexia, occasional vomiting, and gross lethargy. In the prior one month he had suffered vague epigastric discomfort with some relief after taking antacids. At the time of admission he had lost 7 kg in weight and was unable to eat or take any fluid without immediate severe epigastric pain. There was no history of prior peptic ulceration, pancreatitis, unexplained severe abdominal pain, or gastrointestinal tract bleeding. The patient smoked three packets of cigarettes daily, consumed a modest amount of alcohol, and had regularly ingested compound analgesic powders. His father had died three years previously from carcinoma of the pancreas.

Examination revealed a dehydrated man in considerable distress due to severe pain. A firm and exquisitely tender mass was palpable in the epigastrium. The blood pressure was 130/70 mm Hg, the chest was clear to auscultation, and icterus was not apparent.

Investigations

The hemoglobin was 15 g/liter, the total white cell count was 13,100 mm$^3$ with 68% segmented neutrophils, and the ESR was 75 mm in 1 hr (Westergren). The serum amylase was 350 mlU (normal 300 mlU), the liver enzymes were mildly elevated, SAP 250 (20–95 mlU) SGOT 60 mlU (<40 mlU) SGPT 60 mlU (<40 mlU), and the amylase creatinine clearance ratio 3.2% (normal <5%) (3). Serum calcium fasting triglycerides and cholesterol were normal. The isotopic liver scan was within normal limits. Upper gastrointestinal tract panendoscopy revealed gross edema of the antrum and prepyloric region. The instrument was passed into the duodenum with difficulty, and no duodenal pathology was seen. An ultrasonic echoscan was subsequently performed and was reported to show a mass in the region of the lesser sac, measuring $12 \times 9 \times 5$ cm. The elevated liver enzymes and serum amylase returned to normal levels within one week.

The barium meal showed a circumferential narrowing of the distal antrum and pyloric canal, with a deep projection of barium from the greater curvature of the pylorus (Figure 1). It was considered that this appearance was due either to a penetrating antral ulcer or to a sinus tract associated with either ectopic pancreatic tissue or Crohn’s disease involving the stomach. The possibility of adult hypertrophic pyloric stenosis was also raised.

The abdominal pain decreased with intravenous fluid and nasogastric suction and the epigastric mass diminished in size over the course of four days. Endoscopy and barium studies were repeated at ten days but were essentially unchanged. Progress abdominal echoscanning confirmed that the mass had diminished in size. The primary diagnoses considered at this stage were localized perforation of a peptic ulcer, pancreatitis with pseudocyst or abscess formation, and Crohn’s disease involving the stomach.

During the following four weeks in the hospital the patient experienced two further episodes of severe abdominal pain; on each occasion the tender, firm epigastric mass increased in size, and this was accompanied by slight elevation of the serum amylase (500 mlU). The first relapse followed within 24 hr of gastroscopy and the second followed a progress barium meal. On each occasion
GASTRIC ECTOPIC PANCREATITIS

Fig 1. Barium meal demonstrating the narrowed and elongated pyloric canal and the deep projection of barium on its inferior aspect (arrow), which proved to be the duct of the aberrant pancreatic tissue.

Fig 2. Pyloric canal of resected stomach. Long arrow indicates central umbilation of polypoid lesion. Short arrows point to acute superficial erosions.

Fig 3. Mounted section of wall of pyloric canal. Short arrows point to pancreatic tissue in submucosa and muscle. Long arrow indicates site of subserosal fat necrosis.