Preoperative Demonstration of Pancreatic Fistula by Endoscopic Pancreatography in a Patient with Pancreatic Ascites

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Endoscopic pancreatography was useful in demonstrating pancreatic ductal obstruction, fistula, and pseudocyst in the preoperative evaluation of a patient with pancreatic ascites. Surgical exploration confirmed the preoperative findings and Roux-en-Y cystojejunostomy was performed. Some salient features of pancreatic ascites are discussed.

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PANCREATIC FISTULA is an uncommon cause of abdominal ascites, but when encountered may be quite amenable to surgical correction. Therefore, a pancreatic etiology should be kept in mind whenever a new case of ascites is under investigation. The sine qua non of pancreatic ascites is the demonstration of a high amylase content of the ascitic fluid.[4] The recent popularity of endoscopic pancreatography offers an additional tool to further define the etiologic mechanisms of pancreatic ascites. The following case report demonstrates the usefulness of endoscopic pancreatography in the preoperative evaluation of pancreatic fistula.

Case Report

M.W. (#357863), a 31-year-old Negro woman was admitted to the University Hospital on December 29, 1975 with a history of rapid development of abdominal ascites some 3 months prior to admission along with anorexia, 50-lb weight loss, and epigastric abdominal pain. She initially denied, but later admitted, alcohol consumption on the order of a fifth of whiskey per day for several years.

She had initially been seen by her local physician in October, 1975 for the above complaints. He noted a pelvic mass for which exploratory laparotomy was performed. Operative findings included 2500 ml of ascitic fluid, multiple leiomyomata of the uterus, bilateral salpingo-oophoritis and a retrogastric mass. Hysterectomy and bilateral salpingo-oophorectomy were performed. The biopsy of the retrogastric mass was described as "chronic inflammation."

The ascites reaccumulated within one week and the patient was placed on salt restriction and diuretic therapy. The etiology remained obscure. After several admissions to the local hospital for treatment of weakness, hypokalemia, and hypoalbuminemia, the patient was referred to the University Hospital.

Examination of the patient at the time of admission showed a very frail, debilitated, chronically ill woman who appeared much older than her age of 31 years. There was marked muscle atrophy of the extremities with massive ascites but no hepatomegaly. The patient was too feeble to ambulate or care for herself. She was withdrawn and indifferent.

Admission laboratory studies included serum albumin of 2.7 gm%, total protein 5.3 gm%, and normal liver parameters. Serum amylase ranged between 333 and 733 Somogyi units (normal 80–180). Paracentesis was performed and studies of the ascitic fluid showed no growth on culture, negative AFB smear, Class I cytology, albumin of 2.0 gm%, and amylase of 52,000 Somogyi units.

Upper GI series (Fig. 1) showed an extrinsic mass effect on the gastric antrum. Liver scan, IVP, and barium enema had been normal at her local hospital. Celiac arteriograms were normal.

With a tentative diagnosis of pancreatic ascites, an endoscopic pancreatogram was performed on January 13, 1976. This demonstrated obstruction of the pancreatic duct at the junction of the proximal and middle thirds (Fig. 2). In addition, there appeared to be a small leak of contrast material into a cystic structure which was felt to possibly represent a pseudocyst (Fig. 3).

The patient underwent exploratory laparotomy the next day. She was found to have 4 liters of dark ascitic fluid, and a 6–8 cm

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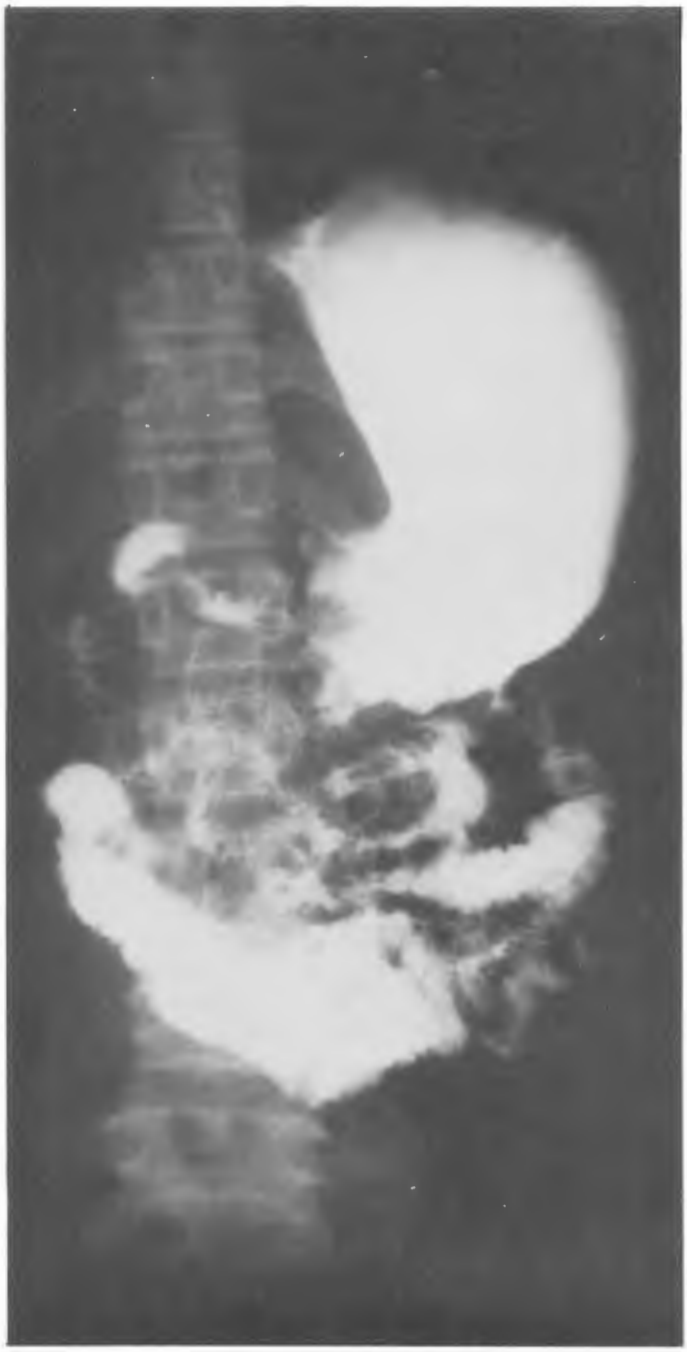

FIG. 1. Preoperative upper GI series showing extrinsic pressure defect on gastric antrum.

diameter retrogastric pseudocyst lying above and behind the lesser curvature of the stomach. There was an apparent leak in the anterior wall of this pseudocyst. The cyst wall was opened and the pancreas was found to comprise the floor of the pseudocyst. A small opening in the floor of the pseudocyst was seen to periodically emit clear fluid. A #5 infant feeding tube was placed into this opening and contrast material injected which outlined the distal pancreatic duct including a 4 cm cyst in the tail of the pancreas (Fig. 4). By repositioning the catheter, an obstruction in the proximal duct was opened and the proximal pancreatic duct was visualized with free flow into the

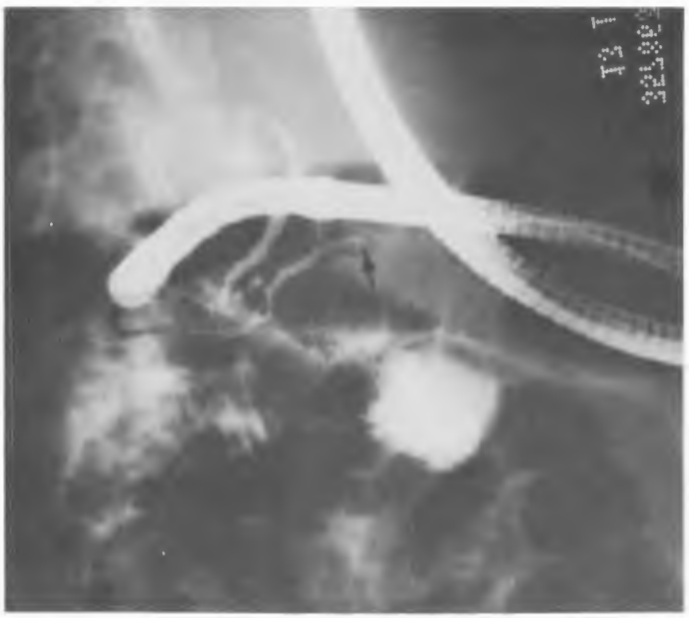

FIG. 2. Preoperative endoscopic pancreatogram showing obstruction of pancreatic duct (arrow). Cystic duct and lower gallbladder are also visible.

duodenum. The cyst was drained by Roux-en-Y cystojejunostomy. Penrose and sump drains were used freely.

Postoperative improvement was dramatic. The patient's appetite returned and her whole mental attitude brightened. Her ascites did not recur. There was minimal drainage from the drain sites and she was discharged on the 10th postoperative day.

At the time of her clinic visit 3 weeks postoperatively she was quite well with a robust appetite, 10-lb weight gain, normal liver studies, total protein of 7.1 gm%, serum albumin 3.8 gm%, and serum

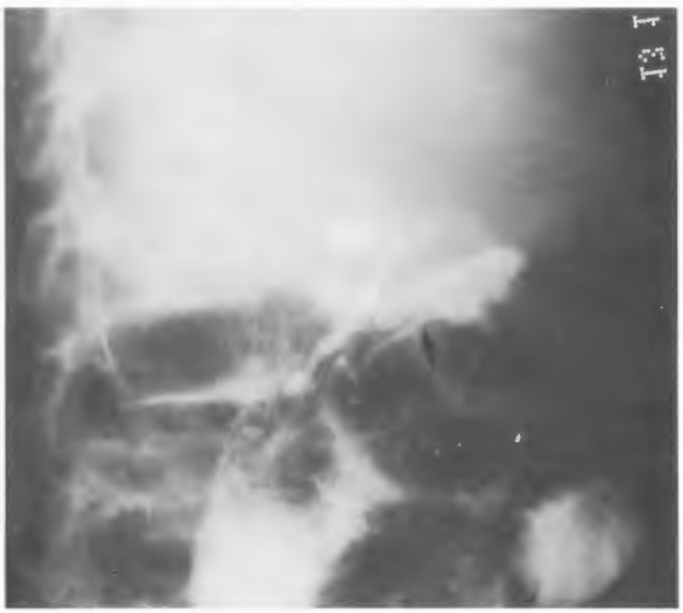

FIG. 3. Preoperative endoscopic pancreatogram (lateral) demonstrating pancreatic duct fistula (arrow) and puddling of contrast material in pseudocyst.

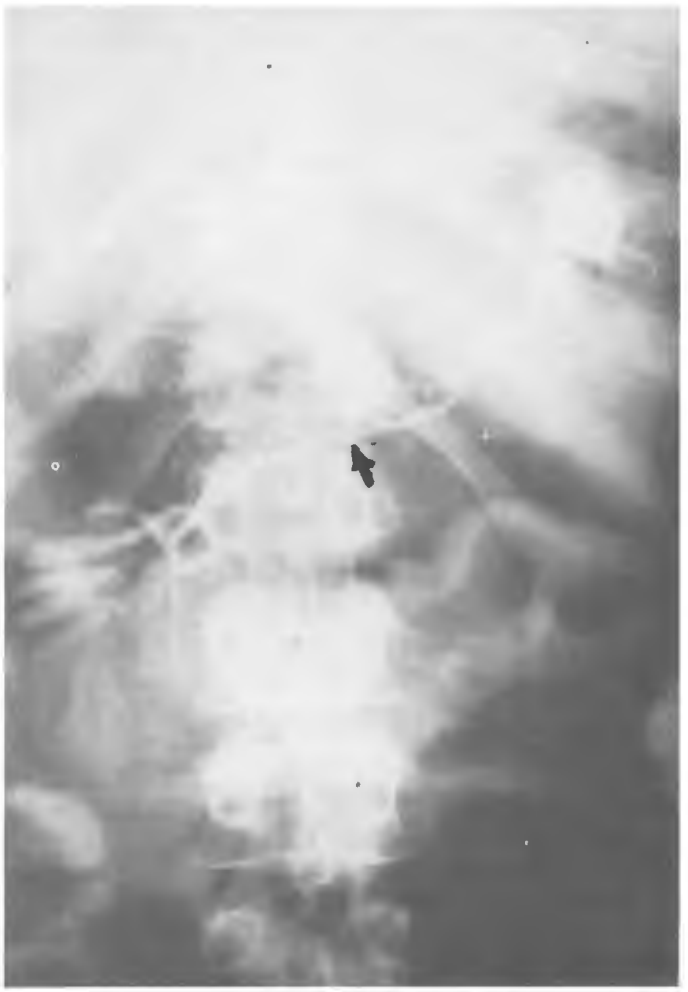

FIG. 4. Operative pancreatogram showing small catheter placed into pancreatic duct fistula (arrow), free flow into the duodenum, and small cyst in the tail of the pancreas.

amylase of 70 Somogyi units. There has been no reaccumulation of her ascites. She has not ingested alcoholic beverages and vigorously asserts she will never again do so.

Discussion

This case illustrates well several points about the diagnosis and treatment of pancreatic ascites.

Pancreatic ascites has been found to be due to disruption of one of the pancreatic ducts either with or without a leaking pseudocyst in 85% of the cases reported.[1] Although any age group can be affected, the condition is most often seen in the 20–50 year age group. Over two thirds of the patients with pancreatic ascites are alcoholics. Trauma[1,3] and acute pancreatitis[2] have been directly implicated in some cases, but the majority of cases of pancreatic ascites develop in patients with no history of recent trauma or acute symptoms. The weight loss, debilitation, and massive amount of ascites can be quite impressive.

The key to the diagnosis is to have a high index of suspicion and to check an amylase level on all ascitic fluid. While the albumin level of ascitic fluid is usually above 2.0–3.0 gm%, only pancreatic ascites will give amylase levels in the range of 20,000–50,000 units.

Upper GI series may show a mass effect if a pseudocyst or large edematous pancreas is present. Biliary tract disease should be ruled out by appropriate radiologic investigations. Selective arteriograms have not been productive in most reported cases.

Preoperative endoscopic pancreatography has been suggested,[5] but to our knowledge has not been previously employed. More experience with this method of evaluation is needed before any conclusions can be drawn about its usefulness in this condition. It is attractive to speculate that endoscopic pancreatography may be quite useful in demonstrating ductal obstruction, fistula, or pseudocyst, or, as in this case, all of these.

Certainly operative pancreatograms should be done to adequately evaluate the ductal system, to demonstrate the leak, and to rule out other leaks which may otherwise be overlooked and lead to reaccumulation of ascites (surgical failure).

Internal drainage, either by cystogastrostomy or cystoduodenostomy, depending on the particular anatomic findings, or by a defunctionalized loop of jejunum is the treatment of choice. The latter method is probably safer should an anastomatic leak develop postoperatively. External drainage is attended by a high recurrence rate.

At the present time, there appears to be little place for non-surgical treatment as this leads to further weakening and debilitation of the patient.

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