Pancreatic Ascites:

Diagnosis and Management with Particular Reference to Surgical Technics

ROBERT B. SMITH, III, M.D., W. DEAN WARREN, M.D.
ADRIEN A. RIVARD, JR., M.D., J. RICHARD AMERSON, M.D.

From the Joseph B. Whitehead Department of Surgery, Emory University School of Medicine, Grady Memorial Hospital, Atlanta Veterans Administration Hospital and Emory University Hospital, Atlanta, Georgia

Ascites due to non-malignant pancreatic disease is being recognized with increasing frequency. Since its first descriptions approximately 20 years ago, 62 cases have been reported in the English literature, over one-half of these in the last 5 years.[8,10,33] The importance of a clear differential diagnosis from other causes of ascites is noteworthy, as operative therapy for pancreatic ascites is specific and often curative. The presumptive diagnosis is formulated from the history in the absence of cirrhosis, cancer or peritoneal tuberculosis; confirmation is easily obtained by the demonstration of an increased protein content and high amylase level in the ascitic fluid.

Seven cases of pancreatic ascites have been diagnosed in the affiliated hospitals of the Emory University School of Medicine during a recent 18-month period of time. An analysis of these cases, together with a review of the literature, affords a perspective on the proper surgical management of this condition.

Case Reports

Case 1 (R. M.). A 50-year-old man was admitted to the Atlanta Veterans Administration Hospital on November 16, 1970, complaining of weight loss and diarrhea. He had been in his usual state of good health until approximately 6 weeks prior to admission, at which time he noted the onset of progressive, painless abdominal swelling, associated with four to six watery, mucoid stools per day. A 20-pound weight loss occurred during that interval, together with the onset of pedal edema. The patient admitted to many years of alcohol consumption, but he had no previous history suggestive of pancreatitis. On admission, he was chronically ill-appearing with wasted extremities and pitting edema of the lower legs. His abdomen was markedly distended with a definite fluid wave; no abdominal tenderness was noted, and there were no palpable masses. Laboratory survey included total bilirubin 0.3 mg./100 ml., alkaline phosphatase 9.2 King-Armstrong units, total protein 6.3 Gm./100 ml. (albumin 2.7 Gm./100 ml.), prothrombin time normal and serum amylase 746 units. Examinations of the stool were negative for parasites, and stool cultures failed to show pathogenic organisms. Repeat serum amylase values ranged from 1044 units to 1854 units. A paracentesis produced tan, clear peritoneal fluid with an amylase value of 951 units and a protein of 3.6 Gm./100 ml. Cytology on this fluid was negative. Upper GI X-rays demonstrated a 0.5 cm lesser curvature gastric ulcer which was subsequently biopsied via the gastroscope and reported to be benign. No pancreatic calcification was evident on abdominal X-rays. A barium enema X-ray showed only diverticulosis of the sigmoid colon; sigmoidoscopy was negative. Follow-up upper GI X-rays confirmed complete healing of the ulcer after 3 weeks in the hospital. Because of persistent ascites an exploratory laparotomy was performed, at which time four liters of ascitic fluid was removed. On exploration of the abdomen, the entire pancreas was noted to be thickened and indurated, but without evidence of pseudocyst formation. An operative pancreatogram was obtained via the ampulla of Vater and demonstrated a dilated pancreatic ductal system with free extravasation of the contrast from the region of the tail of the pancreas into the lesser sac (Fig. 1). Further dissection revealed a 2 mm. punched-out hole in the anterior surface of the pancreas which communicated freely with the main ductal system. In order to effect internal drainage, a Roux-Y loop of jejunum was anastomosed end-to-side to the pancreas at the site of leakage. The patient's postoperative course was uncomplicated, and the serum amylase fell promptly to 140

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Address reprint requests to Robert B. Smith, III, M.D., Veterans Administration Hospital (Atlanta), 1670 Clairmont Road, Decatur, Georgia 30033.

units. When last seen in follow-up, 18 months after operation, the patient had regained his weight and showed no evidence of ascites.

Comment: Although the diagnosis of pancreatic ascites had been made preoperatively in this man, free leakage of pancreatic fluid was not discovered at operation until the disrupted duct had been demonstrated by transduodenal pancreatography. Once the site of leakage had been identified, internal drainage was easily achieved by means of a Roux-Y pancreatojejunostomy.

Case 2 (E. E.) A 38-year-old chronic alcoholic man was admitted to Grady Memorial Hospital on December 12, 1970, with complaints of abdominal pain and diarrhea. He had a past history of numerous bouts of pancreatitis. On the current admission, his abdomen was diffusely tender with massive ascites. The serum amylase was 515 units and rose to a peak of 979 units. Serum bilirubin was 0.2 mg./100 ml., alkaline phosphatase 6.3 King-Armstrong units, prothrombin time slightly elevated and serum protein 5.9 Gm./100 ml. (albumin 1.9 Gm./100 ml.). A paracentesis produced straw-colored fluid with an amylase of 328 units and a protein of 3.5 Gm./100 ml. He responded promptly to treatment with nasogastric suction, analgesics and intravenous fluids and was discharged after several days with moderate ascites still present. On his next examination 3 months later, he was observed to have tense ascites and was readmitted to the hospital with signs of an acute abdomen. On admission, free air was noted under the diaphragm on the chest X-ray. At abdominal exploration, a perforated duodenal ulcer was found. The perforation was simply plicated and the turbid ascitic fluid was evacuated. After an initial febrile course, the patient slowly recovered, and his wound healed primarily despite recurrent, massive ascites. A repeat paracentesis 2 months after operation showed an amylase of 243 units and a total protein of 2.6 Gm./100 ml. Serum amylase on that occasion was 162 units. Because of persistent ascites, a second laparotomy was performed during the 10th week of hospitalization. Approximately 2 liters of straw-colored ascitic fluid was aspirated, but exploration was severely limited by dense adhesions throughout the upper abdomen. Although there was no gross evidence of pancreatic pseudocyst or pancreatic ductal leakage, dissection in the area of the pancreas was extremely difficult, and the procedure was terminated without a definite cause for the ascites being established. Cirrhosis seemed to be excluded by the normal texture and appearance of the liver. The patient's postoperative course was complicated by upper gastrointestinal bleeding, which responded to conservative therapy. His ascites reaccumulated, but was never of the severity present prior to operation. Intravenous hyperalimentation was administered for 4 weeks, by which time the patient had begun to gain weight and had a good appetite. Since discharge from the hospital, he has been essentially asymptomatic with minimal ascites and normal serum amylase values.

Comment: Management of this patient's basic pancreatic disease and complicating ascites was difficult once the duodenal ulcer perforation occurred. In retrospect, it would have been preferable to have performed definitive surgical treatment for the pancreatic ascites on the first hospital admission, since, as it developed, surgical correction was not possible later. Fortunately, gradual, spontaneous resolution of the ascites occurred during the extended period of support on intravenous hyperalimentation.

Case 3 (C. J.) A 34-year-old known chronic alcoholic woman was admitted to Grady Memorial Hospital on May 24, 1971, with complaints of increasing abdominal swelling and weakness. She had been admitted to that hospital 1 year before with a diagnosis of Laennec's cirrhosis and ascites. A paracentesis had been performed on the earlier admission with reports of an amylase of 700 units and total protein of 3.6 Gm./100 ml. In the interval between the two admissions, she had continued to drink heavily and noted intermittent abdominal swelling. On physical examination at the current hospitalization, she was found to be a wasted, chronically ill woman with a markedly distended abdomen. No abdominal tenderness was elicited and no masses could be palpated. Admission laboratory values included total bilirubin 0.2 mg./100 ml., alkaline phosphatase 21 King-Armstrong units, prothrombin time normal and total protein 5.8 Gm./100 ml. (albumin 2.1 Gm./100 ml.). Serum amylase was 1800 units. A paracentesis produced clear, amber fluid with an amylase of 1000 units and a total protein of 1.3 Gm./100 ml. Cytology on the fluid was negative. Abdominal exploration was performed 3 weeks after admission. Three liters of ascitic fluid was aspirated from the peritoneal cavity. The entire pancreas was thickened and fibrotic. An operative pancreatogram, performed by cannulation of the duct at the ampulla of Vater, revealed a minimally dilated pancreatic ductal system with free extravasation of contrast fluid into the lesser sac (Fig. 2). Upon exploration of the tail of the pancreas, a 3 mm. hole was demonstrated in the anterior surface of the gland from which clear fluid was noted to drain. Injection of methylene blue solution via the catheter situated in the main pancreatic duct confirmed free communication between the duct and the lesser sac through the anterior defect. In an effort to drain this area, a posterior gastrotomy was performed and a pancreatogastrostomy established in two layers using interrupted cotton sutures. A decompressing gastrostomy was then placed in the anterior wall of the stomach. The patient's postoperative course was stormy. She developed pneumonia, congestive heart failure, and evidence of intra-abdominal sepsis. By the third postoperative week, the ascites had reaccumulated and she showed increasing abdominal tenderness with a spiking fever. The abdomen was re-explored on August 17, 1971, at which time a large lesser sac abscess cavity was drained. Following operation, she experienced a progressive downhill course marked by excessive drainage from the abdomen and by pulmonary insufficiency related to *Pseudomonas* pneumonia. She died on September 3, 1971; an autopsy was not permitted.

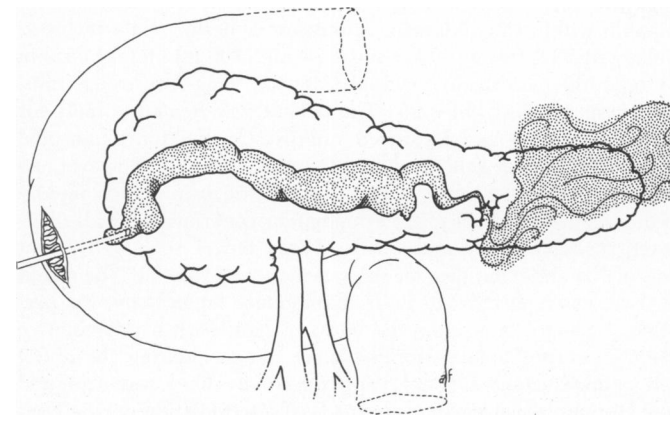

FIG. 1. Case 1. Artist's drawing of a transduodenal operative pancreatogram demonstrating a grossly dilated ductal system and free extravasation of contrast medium from a disruption of the main duct on the anterior surface of the tail of the pancreas.

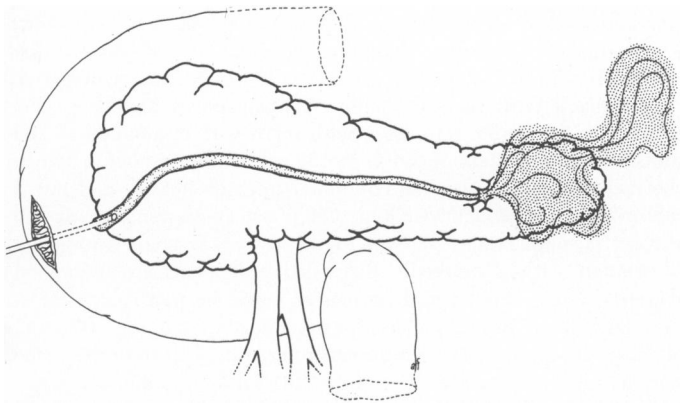

FIG. 2. Case 3. Transduodenal pancreatogram illustrating leakage from a disrupted duct in the tail of the gland.

Comment: In this case, also, operative pancreatography was most helpful in localizing the site of pancreatic leakage. Further identification of the precise area of duct disruption was then obtained by injecting a small quantity of methylene blue into the pancreatogram catheter. Internal drainage of the fistula was attempted by performing a direct anastomosis between the posterior wall of the stomach and the capsule of the pancreas around the site of leakage. The patient's subsequent course was consistent with breakdown of the pancreatogastrostomy suture-line and spillage into the lesser sac, leading to abscess formation. Subsequent drainage of the abscess resulted in a large volume gastric fistula which, in turn, contributed to death.

Case 4 (B. E.) A 49-year-old chronic alcoholic man, with a history of three previous admissions to the hospital for recurrent acute pancreatitis, was admitted to the Atlanta VA Hospital on July 10, 1971 for complaints of vomiting and epigastric pain of 6 hours' duration. On previous admissions, extensive pancreatic calcification had been demonstrated on X-ray and intermittent steatorrhea proven, but there had been no indication of diabetes mellitus. Physical examination on the current admission revealed a thin, dehydrated, chronically ill man with a scaphoid abdomen. Mild epigastric tenderness was noted, but no organs or masses were palpable within the abdomen. Admission laboratory data included hematocrit 34%, fasting blood sugar 91 mg./100 ml., total bilirubin 0.3 mg./100 ml., alkaline phosphatase 9.2 King-Armstrong units, and serum amylase 114 units. The patient was treated initially for alcoholic gastritis and improved rapidly. On the fourth hospital day, he was given oral feedings. On the 11th day, however, he developed a sudden onset of severe epigastric pain, which rapidly became generalized. Physical examination confirmed a rigid, moderately distended abdomen with absent bowel sounds. Upright films of the abdomen did not show evidence of free air. The serum amylase was reported 640 units. Exploratory laparotomy was performed several hours after the onset of pain with a preoperative diagnosis of probable perforated ulcer. Upon entering the abdomen, approximately 600 ml. of gray, cloudy fluid was aspirated from the peritoneal cavity (an amylase determination on the fluid was subsequently reported as 1046 units). The entire pancreas was markedly indurated and nodular. On close inspection, cloudy fluid was observed to be draining into the free peritoneal cavity from a perforation in the posterior parietal peritoneum, adjacent to the second portion of the duodenum. A small catheter was inserted into the tract, radiopaque contrast was injected and an X-ray demonstrated free communication of the fistulous tract with the main pancreatic duct. The pancreatic duct appeared markedly dilated, although there was some evidence of reflux into the duodenum. A Kocher maneuver allowed for precise localization of the orifice of the fistula in the posterior aspect of the head of the pancreas. Internal drainage was then established by anastomosing a Roux-Y jejunal loop to the pancreatic capsule surrounding the site of perforation. The patient's postoperative course was entirely uneventful and he was discharged on the 14th day after operation. In the first 10 months following discharge, he was readmitted to the hospital on three occasions for recurrent upper abdominal pain. Extensive tests on those admissions were negative except for some evidence of hemorrhagic gastritis on gastroscopy. Amylase values remained normal, and there was no recurrence of ascites. Sixteen months after the former operation, he returned to the hospital with a huge pancreatic pseudocyst which had eroded into the stomach and into the splenic artery causing exsanguination.

Comment: This patient illustrates an acute variety of pancreatic ascites presenting as an abdominal catastrophe. The relatively small volume of peritoneal fluid found at initial operation can be attributed to the brief period of lapsed time between rupture of the pancreatic duct and the abdominal exploration. With more time delay, there almost certainly would have been a greater accumulation of ascitic fluid. The importance of X-ray contrast studies in the proper diagnosis of these patients is again demonstrated in this rather unusual clinical situation. Identification of the disrupted pancreatic duct on the posterior aspect of the head of the pancreas by X-ray permitted prompt operative diagnosis and appropriate internal drainage.

Case 5 (C. H.) A 50-year-old chronic alcoholic man entered Grady Memorial Hospital on August 30, 1971 with a chief complaint of abdominal pain for 4 weeks. His pain was mainly epigastric in location, severe in intensity and radiated through to the back. There was no associated nausea or vomiting and no history of trauma. On examination, his abdomen was diffusely tender, but without evidence of ascites. A serum amylase was elevated to 800 units. The diagnosis of acute pancreatitis was made and the patient was treated with intravenous fluids, nasogastric suction and atropine. He showed a good clinical response. By the ninth hospital day his serum amylase had decreased to 189 units and he was tolerating oral feedings well. On the 16th hospital day, however, he developed a recurrence of abdominal pain, associated with nausea and vomiting. Over the next few days his abdomen became markedly distended and the serum amylase rose to 880 units. He was transferred to the Atlanta V.A. Hospital on September 21, 1971, for further management. Upon arrival at that facility, his abdomen was massively distended with an obvious fluid wave, but he had no abdominal tenderness and no masses were palpable. The serum amylase was 704 units; liver function tests were entirely normal. Upper GI X-rays and a barium enema X-ray were normal. Paracentesis produced cloudy ascitic fluid with an amylase of 1259 units and a total protein of 4.2 Gm./100 ml. Cytology on the fluid was negative. The patient was placed on a diuretic program, as well as salt and water restriction. He experienced a slow, but definite decrease in his ascites over the next several weeks. At the end of 4 weeks of observation in the hospital, he was determined to have minimal

ascites and was felt to be clinically stable. He was discharged to the care of his personal physician, free of pain and taking a regular diet, but with a persistent serum amylase elevation to 300 units.

Comment: The onset of pancreatic ascites in this patient within 2 to 3 weeks after an attack of acute pancreatitis raises the question of leakage from a possible pseudocyst or from a ductal disruption secondary to acute pancreatic inflammation. The negative gastrointestinal X-rays and absence of abdominal mass on physical examination do not exclude pseudocyst, since cysts too small to be detectable by these examinations can be responsible for ascites. Although this patient eventually responded to conservative treatment, he still had some ascites and mild elevation of serum amylase at the time of discharge, and, in retrospect, may have made a more rapid and complete recovery with surgical intervention.

Case 6 (J. B.) A 49-year-old man with a 20-year history of chronic alcoholism was admitted to the Atlanta V.A. Hospital on November 16, 1971, for progressive abdominal swelling and a 25-pound weight loss over the preceding 4 months. In 1968, the clinical diagnosis of cirrhosis had been made in another hospital, and since that time, he had continued to have intermittent episodes of recurrent ascites. In addition, he had experienced deep epigastric and left upper quadrant pain for 4 months prior to the current admission. On physical examination, he was a chronically ill-appearing wasted man with a markedly distended abdomen. Mild generalized abdominal tenderness was evident, together with a prominent fluid wave; no organs or masses were palpable within the abdomen. Pertinent laboratory data included total bilirubin 0.4 mg./100 ml., alkaline phosphatase 13 King-Armstrong units, SGOT 30 units, total protein 6.8 Gm./100 ml. (albumin 2.7 Gm./100 ml.), prothrombin time normal and serum amylase 857 units. A paracentesis produced straw-colored fluid which had an amylase of 3444 units and a protein of 3.9 Gm./100 ml. Chest X-ray, barium enema X-ray, and oral cholecystogram were interpreted as normal. The patient was managed initially with supportive therapy and a variety of diuretics, but had no significant regression of ascites. His epigastric pain persisted and upper GI X-rays demonstrated an active ulcer in the duodenal bulb. After 3 weeks of intensive medical therapy repeat upper GI X-rays confirmed healing of the ulcer. There was no evidence of pancreatic enlargement or calcification on any of the X-ray studies. Because of the persistent ascites and chronic serum amylase elevation, the patient underwent exploratory laparotomy on January 28, 1972. At that time, approximately 800 ml. of amber-colored peritoneal fluid was evacuated (amylase on this fluid was subsequently reported as 3040 units and the total protein as 6.1 Gm./100 ml.). On exploration, the liver appeared normal but the entire pancreas was firm and nodular and a 5 cm. pseudocyst was noted to extend from the tail of the pancreas into the transverse mesocolon. No extravasation of pancreatic fluid from the cyst could be demonstrated. A duodenotomy was performed and a retrograde pancreatogram attempted, but the study was unsuccessful due to occlusion of the pancreatic duct in the region of the ampulla of Vater. A caudal pancreatectomy with splenectomy was then accomplished including resection of the small pseudocyst. Thereafter, a pancreatogram was obtained by cannulating the distal duct at the amputated stump of the pancreas; moderate dilatation of the entire ductal system was evident with complete obstruction of the main duct in the head of the gland (Fig. 3). Caudal drainage of the pancreas was then established by means of a Roux-Y pancreatojejunostomy. The postoperative course was uneventful. The serum amylase fell promptly to 116 units following operation and there was no recurrence of ascites by the time of last examination 6 months later.

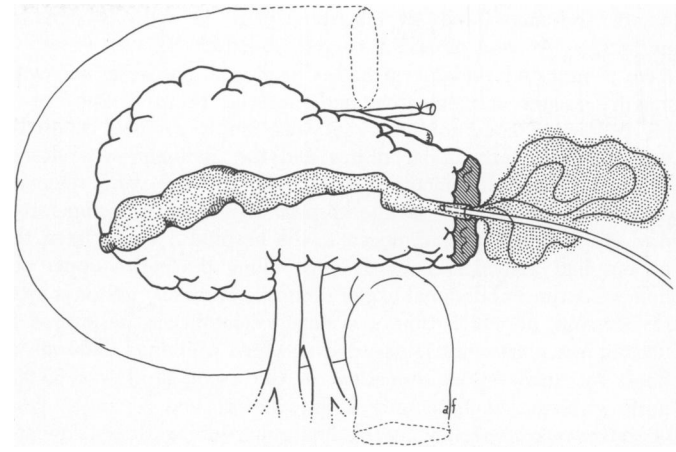

FIG. 3. Case 6. Operative pancreatogram via a catheter introduced into the distal pancreatic duct after caudal pancreatectomy, demonstrating a dilated duct with total obstruction at the duodenal end of the system.

Comment: The diagnosis of "cirrhosis with cirrhotic ascites" is a common error among patients later proven to have pancreatic ascites. This individual apparently had had ascites related to chronic pancreatic disease periodically for over 2 years before the correct diagnosis was made and appropriate therapy instituted. Although the pseudocyst did not appear to be leaking actively at the time of exploration, it was almost certainly the source for the chronic ascites. In this particular case, the pseudocyst was small and located in an area where it could be removed easily by partial pancreatectomy. Distal drainage of the pancreas was also necessary, however, since the pancreatic duct was found to be totally obstructed at its proximal end on operative pancreatography.

Case 7 (H. W.) A 52-year-old man was admitted to Emory University Hospital on January 30, 1972, for symptoms of severe abdominal pain and back pain for 8 weeks. He had sustained a 30-pound weight loss over a period of 6 months. Physical examination was unremarkable except for systemic hypertension and moderate hepatomegaly. Diabetes mellitus was confirmed by glucose tolerance test and mild steatorrhea demonstrated on a 72-hour fecal fat examination. Multiple radiologic examinations including upper GI X-rays, hypotonic duodenogram, intravenous cholangiogram and celiac-superior mesenteric arteriograms were not diagnostic. A barium enema X-ray showed diverticulosis. At exploratory laparotomy, the patient was found to have carcinoma of the head and body of the pancreas with invasion of the peri-aortic retroperitoneal tissues. A confirmatory biopsy was obtained and sump drainage of the area of dissection was established. The early postoperative course was complicated by pneumonia and an episode of severe hypertension. Three weeks following operation, he developed a tender left upper quadrant mass which was a probable pancreatic pseudocyst resulting from the recent operative trauma. Because of the known unresectable malignancy, it was decided to observe the pseudocyst and, accordingly, the patient was discharged from the hospital. Two weeks later he was readmitted with

severe abdominal and left shoulder pain, as well as signs of peritonitis. At re-exploration of the abdomen he was found to have a ruptured pseudocyst in the region of the lesser sac with massive ascites and extensive peritoneal fat necrosis. Because of his precarious condition, the cyst was simply drained externally with multiple soft rubber drains and the abdomen was closed. The drains ceased functioning after 6 days and were removed prior to his discharge from the hospital on the tenth postoperative day. By the time of readmission to the hospital 3 weeks later, the patient had a recurrent, cystic mass filling the entire upper abdomen. A third abdominal exploration was undertaken 2 days after readmission, at which time a cystogastrostomy was performed to decompress a retrogastric pseudocyst which contained 5400 ml. of fluid. An amylase determination on the cystic fluid was 65,000 units; a serum amylase only 2 days before was 51 units. Postoperative recovery following this final procedure was very slow and complicated by episodes of gastric bleeding. Intravenous hyperalimentation was administered for a 2-week period to provide support while his oral intake was inadequate. Moderate ascites reaccumulation was noted by the fourth week following operation and persisted until death on July 22, 1972. An autopsy confirmed adenocarcinoma of the pancreas with obstruction of the common bile duct and metastases to the retroperitoneum and to the liver. The cystogastrostomy appeared patent; only a 5 cm. residual space remained of the previous large pseudocyst.

Comment: Pancreatic ascites was rather abrupt in onset in this man, the result of an acutely ruptured pseudocyst. Simple external drainage of the cyst was decided upon at the second operation because: a) the cyst wall was fragile and did not appear suitable for a suture anastomosis, and b) the patient was not in condition to withstand a more complex surgical procedure. External drainage was successful in resolving the acute problem, but the pseudocyst recurred within a few weeks and required later, definitive surgical treatment. At third operation, cystogastrostomy was selected as the method of internal drainage because of the thick, common wall between the large pseudocyst and the posterior aspect of the stomach. The subsequent gastric bleeding episodes that he experienced are a recognized complication of pseudocyst-to-stomach drainage procedures. Fortunately, the bleeding responded to conservative management. The recurrence of ascites during the terminal phase of his illness was felt, in retrospect, to have been due to intra-abdominal carcinomatosis rather than to a pancreatic source, since the previous pseudocyst seemed to be adequately decompressed at the time of autopsy.

Case 8 (R. D.)* A 43-year-old man was admitted to the Tuskegee V.A. Hospital on May 12, 1971, with enormous abdominal swelling and marked malnutrition. He was a chronic alcoholic and had been losing weight steadily for about 1 year. The increase in abdominal girth had occurred over a 4-day period prior to admission. He was severely debilitated and dyspneic with massive ascites and scleral icterus. A paracentesis was not done, but tuberculous peritonitis was suspected clinically. Supportive therapy was instituted; however, his condition continued to deteriorate and he died on the sixth hospital day. An autopsy confirmed severe, generalized peritonitis and pancreatic ascites due to a large, ruptured pseudocyst. An amylase determination on the peritoneal fluid was 800 units.

Comment: This case history illustrates again the importance of a high index of suspicion and the need for positive steps to establish the diagnosis early. Prompt external drainage of the leaking pseudocyst may have been lifesaving in this rapidly worsening clinical situation.

Discussion

Diagnostic Considerations

The proper management of pancreatic ascites depends upon accuracy in diagnosis. Although it is common to have some free peritoneal fluid accumulation in severe pancreatitis and in advanced pancreatic carcinoma, it is relatively uncommon to have ascites as a complication of chronic, non-malignant pancreatic disease.[19,21,22] Patients with massive ascites and cachexia are more likely to be considered to have cirrhosis or abdominal carcinomatosis.[4,28] The former is particularly understandable since chronic alcoholism is an etiologic factor common to both cirrhosis and chronic pancreatitis.[5] The literature, as well as our own case reports, contain a number of examples of patients treated for cirrhotic ascites, sometimes for months with no response, only to find later that the liver is normal and the pancreas has been responsible for the ascitic fluid. Differentiation between the two conditions is further confused by the fact that as many as 20 per cent of cirrhotics have an elevated serum amylase and serum lipase, without any evidence of pancreatic disease.[9] A third condition that has caused major confusion in the differential diagnosis of this condition is tuberculous peritonitis.[1,28] Nephrosis, constrictive pericarditis, polyserositis, and the ascites of marked starvation must also be considered in the differential but are more easily excluded on clinical grounds. Once pancreatic ascites is suspected, definitive diagnosis can be established easily by demonstrating a high protein content and high amylase level in the ascitic fluid.[5] The total protein content of ascitic fluid in cirrhosis is usually in the 0.5–1.5 Gm./100 ml. range, whereas in pancreatic ascites the protein is generally elevated to 2.9 Gm./100 ml. or higher.[4] The amylase content of the ascitic fluid is always elevated in pancreatic ascites, sometimes into the thousands. By contrast, ascitic fluid from patients with uncomplicated cirrhosis, intra-abdominal carcinoma or tuberculous peritonitis can be expected to show a normal amylase value.[1,20] Peritoneal fluid amylase may well be elevated in several acute abdominal conditions—perforated ulcer, intestinal infarction, and acute pancreatitis—but these are not likely to be confused clinically with chronic pancreatic ascites.[20] Cytologic studies and bac-

* The authors express appreciation to the staff of the Veterans Administration Hospital, Tuskegee, Alabama for permission to include this case report in the present series.

terial cultures of the peritoneal fluid should be negative in benign pancreatic ascites; red and white blood cell counts may be elevated in this exudative type response.[31] Ascitic fluid lipase has been reported elevated in one patient to date.[38] The appearance of the fluid removed at paracentesis is of no real aid in differential diagnosis. Most patients have clear, straw-colored fluid, but turbid, sanguinous and even chylous fluids have been reported in patients with well documented pancreatic ascites.

Serum amylase levels are elevated at some time during the hospital course of the majority of individuals with pancreatic ascites, but this test is not a reliable indicator since it occasionally remains entirely normal, as in our Cases 2 and 7.[4] When the serum amylase is elevated, the value is generally less than that of the peritoneal fluid amylase. The two concentrations, in fact, often appear to have little correlation with one another.[5,20]

The clinical setting in which pancreatic ascites develops is variable, but certain features of the history and physical examination are common enough to alert the physician to the possibility. The age of the patient is of little importance, as pancreatic ascites spans the age range from infancy to late adulthood. Among the 70 patients reported to date, 16 are children and 54 are adults (range: 4 months to 57 years). The average age of the adults is 39 years. Males are affected twice as often as females. The majority of the adult patients with pancreatic ascites are chronic alcoholics, 78 per cent (Table 1). Some of these adults have a past history suggestive of acute pancreatitis, but many do not. Most have some abdominal pain associated with development of the ascites; some are totally pain free.[5,20] Duration of the abdominal distention varies from a few hours in some to many months in others, but the majority have had abdominal distention for several weeks when they are admitted to the hospital.[2] Weight loss, weakness, anorexia and diarrhea are commonly associated complaints among these patients. Many of them show profound malnutrition and cachexia as a result of the severe clinical decline that often accompanies the accumulation of massive ascites. In addition to the clinical history, the patient's hospital course itself may provide a clue to the correct diagnosis. These patients typically have resistant ascites that responds poorly, if at all, to salt restriction-diuretic regimens and recurs promptly after paracentesis.[14]

On physical examination, the most constant findings are massive ascites and evidence of severe weight loss. Fever is an infrequent positive finding. The occasional patient presents with a palpable upper abdominal mass and/or abdominal tenderness, but many have no detectable mass on physical examination, even after much of the fluid has been removed by paracentesis.[5]

Among patients in the pediatric age group, the best clue to a possible pancreatic etiology for ascites may be a history of blunt trauma to the abdomen. Seven of the 16 children reported had a fairly convincing history of abdominal trauma in the recent or remote past (Table 1).[36] The authors have had no personal experience with pediatric patients with this condition, but have been unable to ascertain from case reports in the literature any findings on physical examination that would be more specific for pancreatic ascites than those seen in adults.

TABLE 1. *Etiology of Pancreatic Disease among 70 Reported Cases of Pancreatic Ascites*

Children		16 Total Patients
	Trauma	7
	Recurrent pancreatitis	1
	Unknown	8
Adults		54 Total Patients
	Alcoholism	42
	Trauma	5
	Biliary pancreatitis	2
	Unknown	5

Preoperative roentgen studies, if positive, support the diagnosis; if negative, they do not exclude it. Evidence of a pancreatic pseudocyst may be seen by distortion of gas patterns on abdominal plain films or by extrinsic compression of the gastrointestinal tract on GI X-rays.[23] Calcification of the pancreas on X-ray suggests that organ as a possible source for ascites, but this finding has not been reported often among these patients.[36] Pleural effusions have been noted in association with pancreatic ascites in a number of patients but this also constitutes a rather nonspecific diagnostic aid, as many other forms of ascites have accompanying pleural effusion, as well.[3,23,38]

In summary, two steps are necessary for the diagnosis of pancreatic ascites: a) a high index of suspicion of the condition in any patient, young or old, with unexplained ascitic fluid; and b) a diagnostic paracentesis for peritoneal fluid analysis. Barua *et al.* have recommended further refinement of the diagnosis by means of peritoneoscopy.[1] They performed peritoneoscopy in four patients with pancreatic ascites and saw evidence of a pseudocyst in all four. Since most patients with pancreatic ascites require a formal laparotomy for definitive therapy however, peritoneoscopy would seem to be an unnecessary procedure in the majority of these individuals.

Therapeutic Considerations

Successful treatment for pancreatic ascites depends upon an understanding of the pathophysiologic mechanisms involved. A number of different theories have been offered over the years to explain the development and perpetuation of massive ascites in a patient with chronic pancreatitis and/or pseudocyst. Some have attributed the fluid to lymphatic obstruction in the retroperitoneum or mesentery by pressure from a pancreatic pseudocyst.[8] Others have theorized that portal venous

TABLE 2. *Pathological Findings Presumably Responsible for Development of Pancreatic Ascites among 70 Reported Cases*

Pancreatic pseudocyst	41
Pancreatic duct disruption	11
Pancreatitis	9
Unknown or not stated	9

obstruction by extrinsic pressure from the pancreas may result in portal hypertension and secondary ascites.[26,37] Chronic peritoneal inflammation by the transudation of pancreatic enzymes from an inflamed gland and the osmotic effect of severe hypoproteinemia have also been mentioned as contributing factors.[23,25] Some or all of these mechanisms may be at work in individual cases, but the explanation that seems most plausible for the greatest number of patients was offered by Cameron *et al.* in 1967.[4] They found that pancreatic duct disruption, with or without pseudocyst formation, was the most common etiologic factor among their own patients and those reported in the literature with pancreatic ascites. Pancreatic secretions which leak directly from an open duct or via the wall of an incomplete pseudocyst, even if not activated, irritate the peritoneum and evoke an exudative response.[5] Ductal rupture can be related to pancreatitis from alcoholism or biliary disease in most of the adults and to blunt trauma in many of the children involved.[12,30] Table 2 summarizes the pathologic findings at operation, peritoneoscopy or autopsy in the patients reported to date and indicates that nearly 60 per cent have had pancreatic pseudocysts in association with the ascites. Another 16 per cent have had pancreatic duct disruption with direct leakage into the peritoneal cavity.

At the time of abdominal exploration, identification of the site of duct rupture may not be easy under conditions of chronic inflammation, retroperitoneal edema and peripancreatic adhesions. Operative pancreatography has proven to be a very useful diagnostic procedure, especially in those patients who do not have an obvious pseudocyst draining into the abdominal cavity. The first pancreatogram for identification of the source for pancreatic ascites was reported by Sulamaa and Viitanen in 1964.[35] Since then, 12 additional cases have been described, including four from the present series, in which contrast studies were instrumental in the proper selection of therapy.[2,4,5,31,38] A variety of technics have been utilized to perform these pancreatograms, including cannulation of both proximal and distal ends of the main pancreatic duct, direct injection of the fistulous tract, transcystic cannulation of the duct and injection of contrast into an intact cyst. Obscure sites of leakage have been idenified by these technics, as in our Case 4, allowing for quick, accurate diagnoses, which otherwise may have been missed entirely or made only after extensive pancreatic dissection.

The management of pancreatic ascites has been varied in patients reported to date, but the majority have had surgical treatment and, on the whole, the results have been satisfactory (Table 3). It seems reasonable to withhold operation for several weeks of observation if the patient is stable or improving, since a few patients apparently seal their leaks spontaneously and proceed to resolution of the ascites, as in our Case 5.[31] We do not recommend postponing surgical intervention in the patient who continues to deteriorate, however, in anticipation of a possible eventual, spontaneous reversal. Nor do we feel that it is justifiable to defer operation for many months, as recommended by Jensen and Babior, even in the stable patient, since surgical treatment offers an opportunity for prompt, total relief of symptoms at a low risk of mortality.[20] Among the 19 patients who had no surgical treatment (including 15 with medical therapy only and four with irradiation), there were five deaths and one recurrence, for a failure rate of 32 per cent. Abdominal exploration only, without a definitive drainage procedure, also failed in a high percentage of cases. Of 10 patients who had exploration only, six had recurrence or persistence of the ascites and five of these required re-operation for relief of symptoms.

External drainage of the leaking pseudocyst or disrupted pancreatic duct was selected as definitive treatment in 15 patients with pancreatic ascites. Although subsequent follow-up on these individuals showed six treatment failures (one death and five recurrences), the method should not be condemned since it may be the treatment of choice for poor-risk patients or those with immature, fragile cysts that will not support a suture anastomosis, as was true in our Case 7.[29] Warren's experimental and clinical studies have shown that the pseudocyst must be 4 to 6 weeks in duration before sufficient fibrous tissue is present in the "wall" to permit a safe internal drainage anastomosis.[40,41] If operation must be done before the cyst wall is mature, and simple external drainage is elected, a relatively high incidence of external pancreatic fistula or recurrent pseudocyst must be expected. A second stage procedure can usually be delayed, however, until the patient has recovered from the acute, debilitating ascitic problem.

Most of the operations performed for pancreatic ascites are done electively, in relatively stable patients, permitting the surgeon to perform definitive treatment at initial operation.[4] As indicated in Table 3, the methods of treatment have varied from partial resection of the pancreas to a wide variety of internal drainage procedures. The route of drainage selected most often has been connection of the pseudocyst or the leaking duct to the stomach by means of a cystogastrostomy or pancreatogastrostomy (16 cases). This apparently is an attractive internal drainage procedure because of the proximity of the stomach to the pancreatic lesion. It

TABLE 3. *Summary of Definitive Treatment among 70 Reported Cases of Pancreatic Ascites*

Form of Therapy	Number of Patients Treated	Persistent or Recurrent Ascites and/or Pseudocyst	Hospital Deaths	References
Supportive Medical	15	1	4	1, 5, 13, 18, 19, 20, 24, 30, 34, 36, authors
Irradiation Therapy	4	0	1	10, 14, 21, 24
Abdominal Exploration Only	10	6	0	1, 5, 17, 18, 28, 30, 33, 42, authors
External Drainage	15	5	1	6, 7, 8, 16, 21, 27, 28, 30 35, 39, authors
Sphincterotomy	4	2	0	18, 23, 35
Cystogastrostomy or Pancreatogastrostomy	16	2	3	3, 5, 15, 22, 25, 27, 28 30, 31, 32, 38, 42, authors
Cystoduodenostomy or Pancreatoduodenostomy	2	0	0	2, 30
Partial Pancreatectomy Alone	3	0	0	5, 28, 35
Partial Pancreatectomy with Roux-Y Drainage*	6	0	0	4, 5, 31, authors
Roux-Y Drainage Alone	6	1	0	5, 8, 11, 12, authors
Thoracic Duct Drainage	2	1	0	11

* Includes one Puestow procedure

should be noted, however, that three deaths and two recurrences can be attributed to this procedure, for a 31 per cent failure of initial therapy. Not included among those complications and deaths are two other individuals with massive gastric bleeding after cystogastrostomy and two more who had stormy postoperative courses related to intra-abdominal sepsis, presumably from leakage of the gastric suture line. The over-all significant morbidity rate for transgastric drainage, therefore, exceeds 50 per cent. By contrast, Roux-Y internal drainage procedures, with or without concomitant partial pancreatectomy, and partial pancreatic resection alone, have been performed a total of 15 times with only one late recurrence and no deaths reported. Moreover, there have been essentially no major complications such as gastrointestinal bleeding or sepsis related to suture-line breakdown among these patients. On the basis of these accumulated data and our personal experience with six operative cases, the authors feel that Roux-Y cystojejunostomy or pancreatojejunostomy is far superior to any variety of internal drainage that utilizes the stomach for decompression. If done at all, transgastric drainage should be confined to those patients who have a thick, common wall between the pseudocyst and the posterior aspect of the stomach causing complete fusion of the two structures. Care should be exercised to avoid mobilization of the stomach away from the cyst, in order to preserve the common wall and to eliminate the need for suture coaptation. If there is discontinuity between the cyst and the stomach, or if there is no cyst at all, but simply a ruptured duct, we feel that it is contraindicated to attempt transgastric drainage, because the effectiveness of the operation depends on a tenuous suture-line. Decompression with a defunctionalized loop of jejunum is superior in those circumstances, because the integrity of the suture-line is not so critical. If the Roux-Y pancreatojejunostomy leaks, or even separates entirely, bowel continuity is maintained, and, at the worst, one might expect recurrence of the pseudocyst or ascites.

If the cyst is small and located in the tail, excision of the cyst together with a portion of the pancreas may be the preferred treatment. In those cases, as well, a pancreatogram should be performed to determine whether the duct empties into the duodenum or is obstructed somewhere along its course. If obstruction is identified, appropriate Roux-Y drainage should be established (Case 6). The same principle applies in those few patients with no cyst and no obvious duct disruption at abdominal exploration. In those individuals, a pancreatogram is essential, and internal drainage should be provided for any recognized obstructions.[5] A sphincterotomy may suffice for decompression if the obstruction is confined to the proximal end of the main duct.[18,23,35]

Intravenous hyperalimentation is an important adjunct to therapy in patients with pancreatic ascites during the periods of preoperative preparation and postoperative support. This therapy was utilized effectively in two of our patients when oral alimentation was impossible or inadequate (Cases 2 and 7).

Summary

Pancreatic ascites should be considered in any patient with a chronic peritoneal fluid accumulation, regardless of age. The diagnosis can be made simply by paracentesis and analysis of the fluid for protein and amylase content. The serum amylase is usually elevated as well, but may be within normal limits and, thus, is not a reliable index of the pancreas as a source for ascites.

Once the diagnosis has been established, the patient

may be observed on medical treatment for a few weeks if he is otherwise stable, since spontaneous resolution of pancreatic ascites occurs in a significant number of individuals. If the ascites does not resolve, or if the patient's condition deteriorates under observation, abdominal exploration is indicated. Definitive surgical treatment consists of partial pancreatectomy and/or appropriate internal drainage of the leaking pseudocyst or ruptured pancreatic duct. Based upon physiologic principles and the accumulated clinical experience to date, the technic of choice for internal drainage is the defunctionalized Roux-Y jejunostomy. Transgastric drainage is contraindicated in any patient in whom a suture anastomosis is required to connect the stomach and the pancreatic cyst or leaking duct. External drainage may be indicated as the initial procedure in poor-risk patients to achieve immediate survival.

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