Pancreatic Ascites

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Pathogenesis, diagnosis and treatment of pancreatic ascites has been greatly enlightened in recent years, allowing differentiation from other forms of ascites and more specific forms of treatment.

Leakage of pancreatic secretions into the peritoneal cavity can produce massive ascites. Although approximately one-hundred cases of pancreatic ascites have been reported, the condition is almost certainly more common than the literature would suggest. Because pancreatic ascites usually occurs in patients who are heavy users of alcohol, ascites is often ascribed to liver disease. This discussion of the diagnosis and treatment of pancreatic ascites includes two case reports.

CASE REPORT #1

A 66-year-old black man was admitted to Presbyterian Hospital in the Oklahoma Health Sciences Center on 9-27-78 with complaints of several months weight loss, abdominal pain, nausea and vomiting. The patient had lost over 40 pounds weight. He admitted to the daily consumption of one-half pint of alcohol. His abdominal girth had increased markedly during the previous three months. Physical examination revealed an acutely ill male with a massively distended abdomen. There was marked muscle wasting in all extremities. Stool guaiac was positive. Hemoglobin was 8 gm% and hematocrit was 25.9%. Serum amylase was 734 IU/dl. Ascitic fluid removed by paracentesis revealed a protein of 3.2 gm% and amylase of 9,650 IU/dl. Liver function tests were normal. Radiographic examination at the upper-gastrointestinal tract was unsuccessful because the patient repeatedly vomited the ingested barium. Sonography showed only ascites and the liver scan pattern was consistent with diffuse hepatic disease. Endoscopic retrograde cholangiopancreatography (ERCP) was unsuccessful. An acute pulmonary embolus, necessitating treatment with heparin, delayed the institution of intravenous total parenteral nutrition (TPN). After one week of TPN, the central catheter became occluded. The patient declined all medical therapy and requested an operation to relieve his ascites.

On 11-1-78, he underwent abdominal exploration. After drainage of 12 liters of ascitic fluid, an apparent abscess, eight cm in diameter, was found in the head of the pancreas. Cultures of the fluid were sterile. The tail of the pancreas was disrupted near the ligament of
operatively, the patient had resumed normal activities, gained 48 pounds and was free of abdominal complaints.

CASE REPORT #2

A 50-year-old white man was admitted to a community hospital with a two-month history of abdominal swelling and discomfort. He had been a heavy drinker of alcohol but was gainfully employed and considered himself to be in good health until the onset of symptoms. Initially he was assumed to have cirrhosis, but liver chemistries were normal. Paracentesis revealed ascitic fluid with an amylase of 15,800 Somogyi units/dl and 2.93 gm% of protein. Ultrasound examination of the abdomen showed no evidence of pseudocyst. Efforts to perform ERCP were unsuccessful. A diagnosis of pancreatic ascites was made and the patient was managed medically. He became weaker, with loss of muscle mass, and the ascites reaccumulated over a six-week period, and the patient was referred to the University Hospital in the Oklahoma Health Sciences Center.

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and a biopsy of the slightly nodular liver was done. When no obvious pancreatic rupture was seen, operative pancreatography was performed through a duodenotomy and revealed a rupture of the proximal pancreatic duct into a three cm peripancreatic cavity. This cavity had ruptured into the peritoneal cavity in the region of the foramen of Winslow. (Fig 3) Dissection in this region resulted in the demonstration of a communication between this cavity and the pancreatic ductal system. (Fig 4) The jejunum was then divided approximately 20 cm below the ligament of Treitz and the distal end of the jejunum was carefully anastomosed to the rim of the peripancreatic cavity at the superior border of the pancreas. Intestinal continuity was established by anastomosing the proximal end of the jejunum to the side of the Roux-en-Y limb 15 cm distal to the pancreatic-jejunal anastomosis. The postoperative course was uncomplicated. Liver tissue obtained by biopsy showed no evidence of cirrhosis. Five months later the patient had gained weight.

Figure 2—Chest x-ray of Patient #2 after initial thoracentesis.

Physical examination revealed a cachectic 50-year-old white male unable to lie flat because of dyspnea. There was marked abdominal distention. Neck veins were also distended. No abdominal masses could be palpated. There was moderate edema of both ankles and feet. Laboratory studies revealed normal serum electrolytes, BUN, creatinine and liver function tests. Serum amylase was 950 Somogyi units/dl (normal, 50-200 Somogyi units/dl). Chest x-ray demonstrated a massive right pleural effusion. (Fig 2) Thoracentesis yielded 2,200 cc of serous fluid with an amylase of 58,000 Somogyi units/dl and a protein of 2.7 gm/dl. Because of rapid reaccumulation of pleural fluid, a chest tube was inserted and a large quantity of it was removed. Abdominal distention lessened after the pleural drainage. Total parenteral nutrition through a central venous catheter was begun shortly after admission and laparotomy was performed one week thereafter. At operation, two liters of fluid were aspirated from the peritoneal cavity.

Figure 3—Operative cholangiogram, Patient #2, showing ductal rupture at about midpoint with extravasation of dye into the peritoneal space.
was normally active and had no recurrence of ascites.

DISCUSSION

The typical manifestations of pancreatic ascites include massive, chronic, progressive and painless ascites. This process occurs most frequently in males past the fourth decade of life, but has been reported in patients of both sexes over a wide age span. Muscle wasting, especially of the shoulder girdle and extremities, is common. Subcutaneous nodules are sometimes seen and represent fat necrosis. In the Johns Hopkins series, only 4 of 27 patients had experienced abdominal pain. One-third of patients with pancreatic ascites have pleural effusions which represent a retroperitoneal tracking of pancreatic fluid into the thorax. Such a connection evidently existed in Case #2. Patients with pancreatic pleural fluid often present little to suggest intra-abdominal pathology. In Sankaran and Walt’s series, 15% of all pseudocysts had concomitant pancreatic ascites. Alcoholism is present in up to 82% of reported cases.

The cause of pancreatic ascites is rupture of the pancreatic duct or of a pseudocyst. The latter is slightly more common. It was formerly thought that blocked lymphatics were responsible for this ascites. Most episodes of rupture occur without acute pancreatitis. Indeed, this lack of inflammation is probably responsible for the failure of the rupture to seal off. Blunt or penetrating trauma, especially in children, may result in ductal rupture and pancreatic ascites. Surgical trauma may also result in this condition. Only one case of pancreatic cancer has been reported to cause pancreatic ascites.

The differential diagnosis primarily involves cirrhosis. Since one-third of patients with cirrhosis have pancreatic disease, the diagnosis may be missed. Undoubtedly, a large number of patients with pancreatic ascites are managed as though they had cirrhotic ascites. Intraperitoneal carcinomatosis, tuberculous peritonitis, constrictive pericarditis, nephrosis, congestive heart failure and the Budd-Chiari syndrome may also be confused with pancreatic ascites.

The diagnosis is established by measuring ascitic fluid amylase and protein. The amylase is always elevated, usually strikingly so. Ascitic fluid from cirrhotic patients has a normal amylase value, and protein content is consistently over 2.0 gm%. The value of ascitic-fluid lipase as an aid in diagnosis has been championed, but it is difficult to see how this will improve the diagnostic yield since almost every case has elevated ascitic-fluid lipase. The serum amylase is frequently elevated although some patients in Donowitz’s series had normal values. Cytologic studies should be done; however, in Cameron’s series, there were two false positives. Amylase may cause distortion or metaplasia of the cells in the fluid.

TREATMENT

According to Cameron, the initial treatment is medical and should be continued for two-to-three weeks, although Munoz recommends eight weeks and Donowitz recommends two to three months. This therapy includes ap-
propriate parenteral fluid and electrolyte replacement and continuous nasogastric suction. Acetazolamide (Diamox) and atropine have been used to decrease pancreatic secretions, but they are not of proven value. Nutrition is important and TPN has been an invaluable aid in the management of these patients. Thoracentesis and paracentesis have been helpful in speeding resolution of fluid. Almost one-half the patients were cured with medical therapy in Cameron’s series. The mortality for medical management is 20-25% and this probably represents the poor condition of patients rather than the dangers of medical therapy.1,4

Before surgical treatment is considered, endoscopic retrograde cholangiopancreatography should be done. This should delineate pancreatic ductal anatomy and allow planning a rational surgical approach. If endoscopic pancreatography is unsuccessful, operative pancreatography should be done. Without appropriate knowledge of the ductal system, more than one-half of operative procedures will fail.1 With knowledge of the ductal systems, success is assured in the great majority of cases.

For those patients who do not respond to or cannot tolerate medical therapy, operative intervention is indicated. The abdominal approach should be used even when pleural fluid is present. With adequate knowledge of the ductal systems, the ruptured duct or cyst may be drained internally with a Roux-en-Y jejunostomy or through the stomach or duodenum. Resection of the distal pancreas may be done in conjunction with appropriate internal drainage if no rupture of the duct is found. Patients with thoracic pancreatic fluid fare better than those with pancreatic ascites alone.1 The operation is successful in 90% of patients reported.1 Low-dose irradiation has been reported to be successful but should be reserved for patients in whom operation is contraindicated.3 The irradiation apparently decreases pancreatic secretions temporarily and allows the ruptured duct or pseudocyst to seal.

**SUMMARY**

If more patients with pancreatic ascites are to be diagnosed and successfully treated, all patients with ascites must undergo diagnostic paracentesis and the amounts of amylase and protein in the ascitic and pleural fluids should be determined. Recognition of this condition leads to highly successful medical and surgical therapy.

**Bibliography**


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