The Treatment of Pancreatic Ascites

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PANCREATIC ascites is a clinical entity being recognized with increasing frequency. Since the first patient in this hospital with ascites secondary to benign chronic pancreatic disease was recognized 3 years ago,[2] we have diagnosed the entity in eight additional patients. Six patients in the present series have been treated surgically and in five the ascites has not recurred. Because of the manner in which pancreatic ascites presents, however, it may frequently be mistaken for cirrhotic ascites. Because of the effectiveness of surgical therapy we believe a concerted effort should be made to differentiate these patients with pancreatic ascites from those with ascites secondary to cirrhosis.

Clinical Material

Of the nine patients with pancreatic ascites in the present series, seven were patients at The Johns Hopkins Hospital and two at The Loch Raven Veterans Administration Hospital (Table 1). All nine patients had painless ascites. In eight of the patients the ascites was massive, and in seven it was the only reason medical help was sought. There was no history of trauma in any of the nine patients. The duration of the ascites ranged from 1 week to 18 months and averaged 3 to 4 months (Table 1). Six of the nine patients had a history of heavy alcoholic intake and most were suspected initially of having cirrhosis. During the course of the workup, however, seven were found to have no liver disease and the remaining two had only minimal liver impairment. Two patients (Patients 5 and 8) had documented episodes of acute pancreatitis 1 and 7 months prior to the development of painless ascites. The remaining seven patients, however, had no prior history suggestive of pancreatic disease. There was no significant abdominal tenderness or palpable abdominal masses in any of the patients, even after paracentesis. Many diagnostic procedures such as gastrointestinal series, barium enemas, celiac axis arteriograms, and liver scans were carried out and these were all nondiagnostic.

The ascites was straw colored in eight patients and serosanguineous in one (Table 2). The protein content of the ascites ranged from 2.2 to 5 Gm./100 ml. The ascitic fluid amylase was also elevated, often ranging into the thousands (Table 2). The serum amylase value was generally much lower than the corresponding ascitic fluid amylase, but was elevated in all but one patient (Patient 9). The triade of an elevated serum amylase and an elevated ascitic fluid amylase and protein was therefore present in all but one patient (Table 2).

Treatment

Six of the nine patients with pancreatic ascites have been operated upon (Table

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TABLE 1. *Summary of Nine Patients with Pancreatic Ascites*

	Patient		Duration of Ascites	History of Pancreatitis	History of Alcoholic Intake	Liver Disease
1	E. P. JHH	30NF	4 months	No	Heavy	No
2	P. S. JHH	29NM	1 week	No	Heavy	No
3	C. W. LRVAH	38NM	6 months	No	Heavy	Minimal
4	E. B. JHH	57NF	1 week	No	Light	No
5	J. T. LRVAH	44WM	5 months	Yes	Heavy	Minimal
6	P. M. JHH	37WF	2 months	No	Light	No
7	G. M. JHH	32NM	3 weeks	No	Heavy	No
8	C. B. JHH	26NM	1 week	Yes	Heavy	No
9	B. S. JHH	4WF	18 months	No	No	No

3). At surgery three of the six patients were found to have pseudocysts. Patient 1 was operated upon after 30 days of medical management during which multiple paracenteses were performed with rapid reaccumulation of fluid. At laparotomy a firm mass was present in the tail of the pancreas (Fig. 1A). The head and body of the gland were small, indurated, and nodular. An operative pancreatogram was performed which showed a proximal duct stricture and a small pseudocyst present in the tail of the gland (Fig. 2). A sphincterotomy was performed and the pseudocyst resected. The distal pancreas was drained with a Roux-en-Y loop (Fig. 1B). Postoperatively the patient did well and has remained free of ascites for 3 years. Patient 2 was treated medically with paracenteses and diuretics for 2 weeks prior to surgery. At operation the distal body and tail of the pancreas were indurated, edematous, and enlarged (Fig. 3A). The duodenum was opened and an operative pan-

TABLE 2. *Serum and Ascitic Fluid Values for Patients with Pancreatic Ascites*

Patient	Serum Amylase (mg./100 ml.)	Ascitic Fluid		Character
		Amylase (mg./100 ml.)	Protein (gm./100 ml.)	
1	1380	4365	3.5	Straw colored
2	785	8250	3.5	Straw colored
3	1060	16,800	4.3	Straw colored
4	1970	4035	3.9	Straw colored
5	544	360	5.0	Straw colored
6	340	510	2.9	Straw colored
7	1000	8000	3.0	Serosanguineous
8	595	2875	4.4	Straw colored
9	120	630	2.2	Straw colored

TABLE 3. *Treatment of Nine Patients with Pancreatic Ascites*

Patient	Pathology	Treatment	Operative Pancreatogram	Result
1	Pseudocyst with proximal duct stricture	Excision of cyst with Roux-en-Y drainage: sphincterotomy	Proximal duct stricture with distal pseudocyst	Asymptomatic and free of ascites 3 years
2	Pseudocyst	Excision of cyst	Extravasation of contrast media from pseudocyst	Asymptomatic and free of ascites 5 months
3	Pseudocyst	Cystogastrostomy	—	Died 2 weeks post-operatively
4	Disruption of pancreatic duct	Roux-en-Y drainage of duct leak	Extravasation of contrast media from duct	Asymptomatic and free of ascites 2 years
5	Duct strictures	Puestow procedure	—	Asymptomatic and free of ascites 2 years
6	Disruption of pancreatic duct	Exploratory laparotomy with operative pancreatogram	Retroperitoneal extravasation of contrast media	Asymptomatic and free of ascites 2 years
7	—	Medical	—	Asymptomatic and free of ascites 1 year
8	—	Medical	—	Asymptomatic and free of ascites 3 months
9	—	None	—	Asymptomatic with ascites

creatogram performed. The study revealed a small pseudocyst in the distal pancreas with free extravasation of contrast media from the pseudocyst into the peritoneal cavity (Fig. 4). The distal edematous gland and pseudocyst were resected and the proximal end over sewn with horizontal mattress sutures (Fig. 3B). A sump drain was left in the area of the resection. The postoperative course was benign and the patient has remained free of ascites for 5 months. Patient 3 was initially admitted to the hospital with the diagnosis of cirrhotic ascites. When liver functions were found to be only minimally impaired he was started on INH for suspected tuberculous peritonitis. After several weeks of paracenteses in an effort to control the ascites and obtain a positive culture or smear, the diagnosis of pancreatic ascites was made. At surgery a pseudocyst was found in the tail of the gland, firmly adherent to the posterior wall of the stomach. A cystogastrostomy was performed. Postoperatively the patient developed massive atelectasis and had a respiratory arrest. He was resuscitated but was decerebrate and died 2 weeks after surgery.

The remaining three patients who were operated upon had pancreatic duct disruptions without pseudocysts. Patient 4 was treated medically for several weeks with paracenteses. Because the ascites was not controlled she was operated on and found to have a nodular, indurated pancreas. Inspection of the anterior surface of the body of the gland revealed an indurated area surrounding a 2–3 mm. hole (Fig. 5A). The duodenum was opened and an operative pancreatogram revealed free extravasation of contrast media from the ductal system into the peritoneal cavity through the hole in the anterior surface of the gland (Fig. 6). A Roux-en-Y loop was anastomosed end-to-side to the pancreas over the pancreatic leak (Fig. 5B). Postoperatively the patient did well and has remained free of ascites for 2 years. Patient 5 was initially

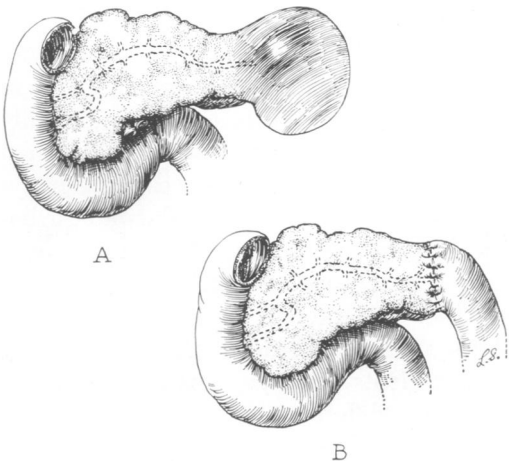

FIG. 1A. Small mass present in the distal pancreas of Patient 1. The rest of the gland was nodular and indurated. B. Pseudocyst resected with drainage of the distal gland with a Roux-en-Y loop.

admitted to the hospital with acute pancreatitis. One month after the pancreatitis subsided he developed painless ascites which persisted despite repeated paracenteses over a 5-month period. At surgery a calcified duct was palpated in an indurated, shrunken gland and a Puestow procedure was carried out. Over a 2 year follow-up he has been free of ascites. Patient 6 was transferred from another hospital with a 2-month history of painless ascites. She had been operated upon 2 weeks prior to transfer but no cause for the ascites was found. After admission she was believed to have constrictive pericarditis but tests including a cardiac catheterization were negative. Subsequently the diagnosis of pancreatic ascites was made and at operation an edematous pancreas with massive retroperitoneal edema was found. An operative pancreatogram revealed retroperitoneal extravasation of contrast media from the ductal system in the head of the gland (Fig. 7). No definitive surgery was performed. After a stormy postoperative course with staphylococcus peritonitis and septicemia, the retroperitoneal leak apparently sealed and the patient has been free of ascites for 2 years.

The remaining three patients were treated medically. One patient (Patient 7) was hospitalized for 3 months, much of the time on nasogastric tube suction with no oral intake. Multiple paracenteses and thoracenteses were performed with the removal of many liters of protein rich fluid from the peritoneal cavity and right chest. Finally, after 3 months the patient remained free of both peritoneal and pleural fluid and was discharged. He lost 50 pounds of weight during hospitalization. One year after discharge he had regained his pre-admission weight, was asymptomatic, and was free of ascites. A second patient (Patient 8) was treated for 2 weeks by the medical service with atropine, diamox, nasogastric tube drainage and no oral intake after being tapped dry at paracentesis. There was no reaccumulation of fluid. Over a 3-month follow-up he remained free of ascites. The final patient, a juvenile diabetic child (Patient 9), was known to have pancreatic ascites for 18 months. The ascites was only moderate in amount, however, and her pediatricians chose to study her with no specific treatment.

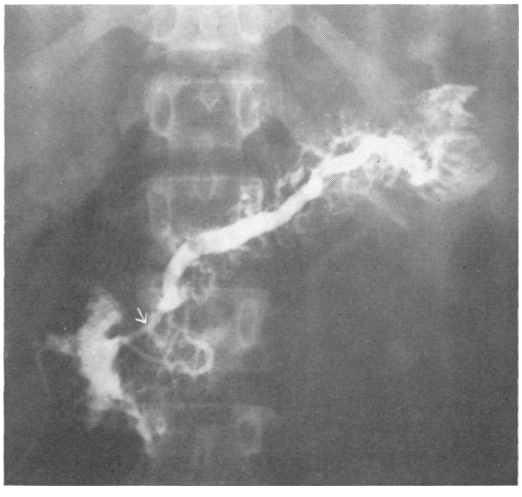

FIG. 2. Operative pancreatogram of Patient 1 showing proximal duct stricture (arrow) and distal pseudocyst filled with contrast media. Pancreatic duct is ectatic distal to the stricture.

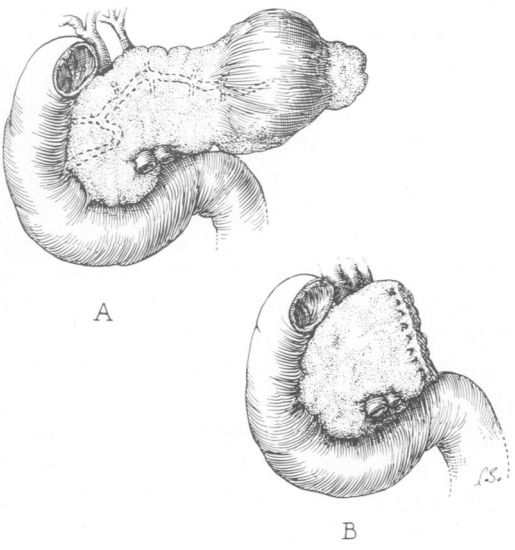

FIG. 3A. Small mass present in the tail of the pancreas of Patient 2 with surrounding edema and induration. B. Distal pancreas including pseudocyst resected and end of gland oversewn.

Discussion

Ascites secondary to benign chronic pancreatic disease may occur with relative frequency. The recognition of this clinical entity may be difficult, however, because of its similarity to alcoholic cirrhosis with ascites. Most of the patients in the present series were initially considered to have alcoholic cirrhosis with ascites. All but three of them had a history of heavy alcoholic intake. Only two of the nine patients had a history suggestive of pancreatitis, and in those two ascites developed 1 month (Patient 5) and 7 months (Patient 8) after their last attack of acute pancreatitis. Only because of a high index of suspicion was the diagnosis of pancreatic ascites made. If one considers the entity, however, the diagnosis can be made with relative ease. In an earlier report from this clinic the serum amylase and the ascitic fluid amylase and protein were shown to be elevated in patients with pancreatic ascites.[2] This triade was present in all patients in the current series except for one (Patient 9) who had a normal serum amylase. In ascitic fluid secondary to cirrhosis the protein content is usually low and the amylase content is normal. The serum amylase in patients with cirrhosis can be minimally elevated but is usually in the normal range.[3] Pancreatic ascites can so easily masquerade as ascites secondary to cirrhosis that any patient with ascites should have a serum amylase and an ascitic fluid amylase and protein content determined. With the aid of these three laboratory tests patients with pancreatic ascites can be readily differentiated from patients with ascites secondary to cirrhosis. It is also possible that some patients may have both cirrhosis and pancreatic ascites, since in alcohol they have a common etiologic precursor. It is conceivable that the ascites in some patients with cirrhosis may actually be the result of concurrent pancreatic disease. In these patients with both hepatic and pancreatic disease the specific etiology of the ascites may be elucidated with the aid of these three laboratory determinations.

Previously pancreatic ascites was thought to be secondary to chronic peritoneal irritation and blocked abdominal lymphatics.[1,7] In a review of the previously reported cases of pancreatic ascites, 11 of 13

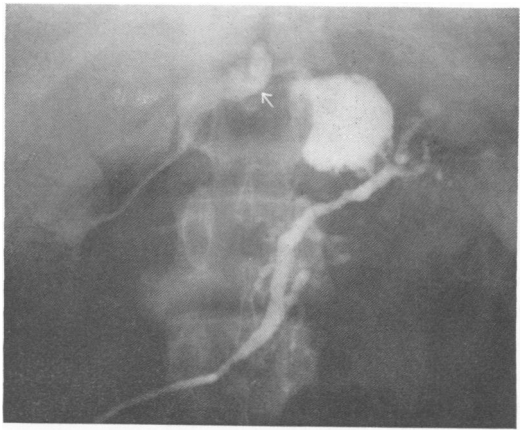

FIG. 4. Operative pancreatogram of Patient 2 demonstrating distal pseudocyst. Contrast media is seen extravasating free into the peritoneal cavity (arrow) from the cyst.

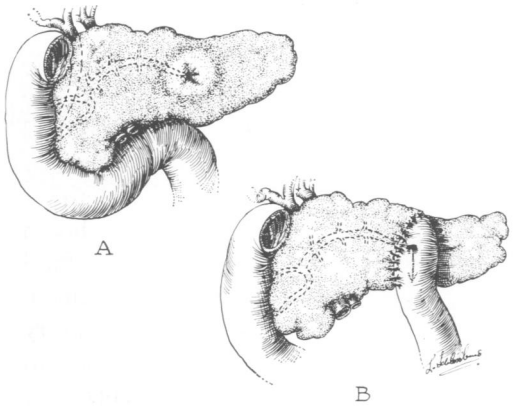

FIG. 5A. Pancreas of Patient 4 showing an indurated and nodular pancreas. An indurated area in the body of the gland surrounds a small hole which was found to communicate with the ductal system. B. Roux-en-Y loop anastomosed end-to-side over the hole leading to the pancreatic duct.

patients were found to have small pseudocysts.[2] This fact indicates that pancreatic duct disruption is of etiologic significance. Operative pancreatograms in three of the present patients demonstrated duct disruptions (Figs. 4, 6, 7,) with escape of contrast media into the peritoneal cavity. The usual response to pancreatic duct disruption is the formation of a pseudocyst. Adjacent tissues and organs become inflamed by the extravasated pancreatic juices and contain the leak by the formation of a pseudocyst. When the duct disruption is incompletely contained pancreatic secretions are released free into the peritoneal cavity. Pancreatic secretions even when not activated are irritating to peritoneal surfaces and albumin is exuded with a resultant ascitic fluid rich in amylase and protein. Ascites secondary to traumatic duct disruption has been reported and in those instances the ascites has also been high in amylase content.[4, 5, 6, 8] Why pancreatic duct disruption occurs in patients with no history of trauma or acute pancreatitis is unclear. In the present series, all six patients operated upon had gross and histologic evidence of chronic pancreatitis even though only two of the nine patients in the series had a history suggestive of pancreatic disease. All but one of the patients had elevated serum amylase levels, but these probably were secondary to absorption of amylase from the ascitic fluid rather than to active pancreatitis. The process leading to duct disruption in the pancreas may often be secondary to alcohol ingestion and seemingly leads to the histologic changes of chronic pancreatitis, but in many instances it apparently occurs without symptoms.

The correct mode of therapy for all patients with pancreatic ascites is not fully established. Many undoubtedly will require surgery. Some, however, such as Patient 8 may be cleared quickly by medical therapy and will not need surgery. Others who require multiple paracenteses over prolonged periods with rapid reaccumulation of fluid should be operated upon. Possibly a 2- to 3-week period of nasogastric tube suction and atropine or diamox administration should be tried along with paracenteses in an attempt to decrease pancreatic secretion and allow the leak to seal. If this program fails to control the ascites surgery should be undertaken. A prolonged course of medical management with a resultant excessive weight loss such

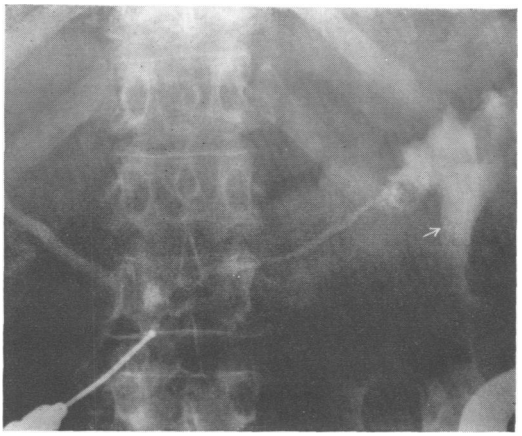

FIG. 6. Operative pancreatogram of Patient 4 showing free extravasation of contrast media through the hole in the pancreatic duct into the peritoneal cavity.

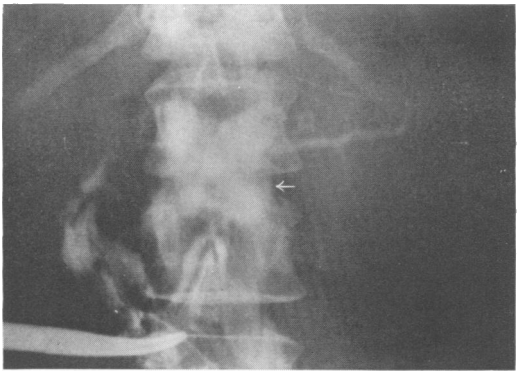

FIG. 7. Operative pancreatogram of Patient 6 showing retroperitoneal extravasation of contrast media (arrow) from the pancreatic duct.

as one patient (Patient 7) in the present series experienced is probably unwise.

When surgery is undertaken operative pancreatography is of prime importance. Since pancreatic duct leakage appears to be the cause of the ascites, the source of the disruption should be identified if possible. Three of the patients operated upon in this series had pseudocysts. If a pseudocyst is present it probably will be the site of the pancreatic leak as was demonstrated in one of our patients (Fig. 4). If the cyst is small and can be resected and there is no evidence of proximal duct stricture, resection alone probably suffices (Fig. 3B). If there is evidence of proximal duct stenosis, however, the distal gland should be drained with a Roux-en-Y loop (Fig. 1B). Most of the pseudocysts associated with pancreatic ascites seem to be small, probably because they are decompressed into the peritoneal cavity. If the cyst is large, however, and cannot be resected internal drainage can be carried out. This decompression should allow the pancreatic leak to seal.

If a pseudocyst is not present, as in three of the present operative cases, a direct pancreatic duct leak must be assumed. If operative pancreatography identifies the disruption, drainage can be carried out with a Roux-en-Y loop (Fig. 5B). If the pancreatic duct leak cannot be identified, a duct drainage procedure such as Duval or Puestow probably should be performed. This decompression of the duct should allow the leak to seal.

The surgical alternatives for the treatment of pancreatic ascites are multiple. They should all be attempts, however, to resect or drain any pancreatic leak that is identifiable, or to decompress the ductal system so that an unidentifiable or inaccessible leak may seal. If operative pancreatography is used and these principles followed, the results of surgical therapy should be successful.

Summary

Painless ascites secondary to benign chronic pancreatic disease is being diagnosed with increasing frequency. A series of nine patients recognized over a 3-year period is presented. Pancreatic duct disruption with direct leakage of pancreatic juice into the peritoneal cavity appears to be the cause of the ascites. The triade of an elevated serum amylase and an elevated ascitic fluid amylase and protein is diagnostic of pancreatic ascites. Its medical and surgical management are discussed.

References

1. Barua, R. L., Villa, F. and Steigmann, F.: Massive Ascites Due to Pancreatitis. Amer. J. Dig. Dis., 7:900, 1962.
2. Cameron, J. L., Anderson, R. P. and Zuidema, G. D.: Pancreatic Ascites. Surg. Gynec. Obstet., 125:328, 1967.
3. Cummins, A. J. and Bockus, H. L.: Abnormal Serum Pancreatic Enzyme Values in Liver Disease. Gastroenterology, 18:518, 1951.
4. Doubilet, H. and Mulholland, J. H.: Surgical Management of Injury to the Pancreas. Ann. Surg., 150:854, 1959.
5. Hardy, J. D. and Bowlin, J. W.: Some Complications of Pancreatic Disease. Ann. Surg., 145:848, 1957.
6. Parrish, R. A., Humphries, A. L. and Moretz, W. H.: Massive Pancreatic Ascites. Arch. Surg., 96:887, 1968.
7. Schmidt, E. H. and Whitehead, R. P.: Recurrent Ascites as an Unusual Complication of Chronic Pancreatitis. JAMA, 180:533, 1962.
8. Sulamaa, M. and Viitanen, I.: Treatment of Pancreatic Rupture. Arch. Dis. Child., 39:187, 1964