Pancreatic Ascites, Producing Abdominal Distension

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A 44-years-of age female presented herself on January 27, 1970, with an illness characterized by diffuse upper abdominal pain, abdominal distension, protracted diarrhea, weakness, vomiting, loss of weight, low grade fever, no jaundice, and a poor appetite. A history of heavy alcohol intake over a period of several years was obtained. She was anemic, dehydrated and emaciated. Her abdomen was moderately distended with clinically detectable ascitic fluid. There was no history of trauma. These symptoms and signs were documented as those of pancreatic ascites and effectively treated by an appropriate surgical procedure.

Pancreatic ascites is a distinct clinical entity and is increasingly being separated from cirrhosis of the liver with ascites.[1] Most frequently patients with cirrhotic liver ascites and patients with pancreatic ascites, both have a well established history of alcoholism. Largely because of this common history of alcohol being a great causative factor in each disease, pancreatic ascites has been clearly and emphatically separated in diagnosis and treatment from cirrhotic liver ascites-, only in the past decade.[1] Cameron, Brawley et al.[1] reported a comprehensive study of the diagnosis and treatment of pancreatic fluid in the peritoneal cavity of large quantity in a series of nine patients within a three-year period, at the Johns Hopkins Hospital and the Loch Raven Veterans Administration Hospital. The number of cases gives some concept of the infrequency of this syndrome.

CASE REPORT

R.P., No. 59779, is a 44-years-of-age female who was admitted (to The Hughes Spalding Pavilion), initially, on January 27, 1970, because of upper abdominal pain, distension, tarry stools, and a loss of weight of 15 pounds over a four month period of time. Her alcoholic intake had been heavy for many years. She also had some purulent para-rectal drainage. The admitting impressions were:—pancreatitis, duodenal ulcer, cirrhosis of liver with ascites, and peritoneal carcinomatosis possibly due to ovarian or pancreatic carcinoma. She also had, incidentally, multiple fistulae-in-ano. She was emaciated, dehydrated, and presented a distended abdomen with ascites, but no abnormal mass. Neither the spleen nor liver was palpable. The admission hematocrit was 24%; W.B.C. 14,800 with 88% polys. Urinalysis: albumin-negative, sugar-neg. (I.V. fluids) and microscopic-normal. E.K.G.-normal. Serum cholesterol-125; calcium-8.4 mgm%; inorganic phosphorus-2.8 mgm%; total bilirubin-0.6 mgm%; albumin-2.0; total-protein-6.2 gm.; uric acid-6.9; B.U.N.-16; glucose-80 mgm; L.D.H.-90; alkaline phosphatase-22; S.G.O.T.-27. Serum amylase-352 units and 323 units (normal 40-150 units) V.D.R.L.-N.R.; B.S.P.-1.8%; Ceph. floc.- neg. in 24 hours; thymol turbidity-10.5. Serum sodium-136; serum chloride-105; serum potassium-4.4 and CO_2-26.9. X-ray chest-normal. Barium enema-a few diverticula of the colon with some shortening of the colon. Upper G. I. series-small duodenal ulcer.

The patient was placed on an anti-ulcer regime, hydrated, given whole blood transfusions and anti-infectives. Sigmoidoscopic was normal to 25 cm. and fistulectomy was performed. The patient improved and was discharged on March 2, 1970, with a less distended but by no means flat abdomen.

She rapidly grew worse at home with an increase in abdominal distension, loss of weight, anorexia, weakness, dehydration, and emaciation with a weight of 84½ pounds. She was in a critical condition on re-admission on March 23, 1970. Re-admission hematocrit was 30; W.B.C. 9,810. Urinalysis: albumin-negative; sugar-negative; microscopic-negative; serum amylase-512 units. Serum sodium-135.5; chloride-108; potassium-4.6; CO_2-21.9. E.K.G.-normal. Chest x-ray-fluid in both pleural spaces-, with more on the right (Fig 1). A scout x-ray of the abdomen revealed a "ground glass" haziness of the entire abdomen-, highly suggestive of ascites (Fig 2). Serum cholesterol-120; calcium-8.5; inorganic phosphorus-3.75; total bilirubin-0.6; albumin-1.7; total protein-6.5; uric acid-9.8; B.U.N.-20; serum glucose-95; L.D.H.-97. Alkaline phosphatase-13; S.G.O.T.-42.

The patient was hydrated, transfused and prepared for laparotomy. Thoracentesis was performed prior to laparotomy and 950 cc of brownish pleural fluid was aspirated with an amylase determination of 772 units.

On laparotomy on March 30, 1970, over 4,300 cc of brownish ascitic fluid was aspirated from the peritoneal cavity with an amylase value of 4,830 units. The liver was firm and showed evidence of early nodulation. The gall-bladder, common bile duct, stomach, intestines,

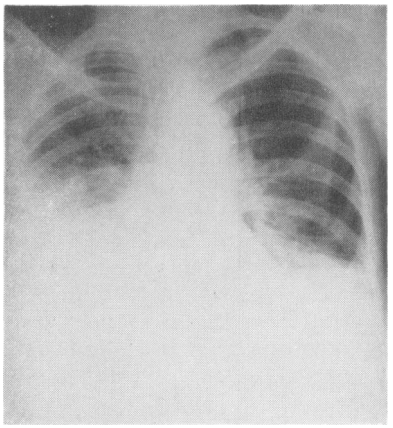

Fig. 1. Brownish fluid was in both pleural spaces with an amylase value of 772 units.

ovaries, and kidneys were normal. The spleen was slightly enlarged. The pancreas was edematous and there was an orange-size pseudocyst in the body of the pancreas—to the left of the vertebral column- and pos-

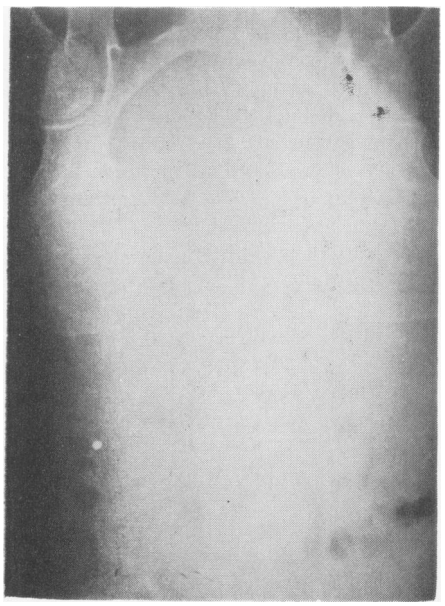

Fig. 2. Over 4,300 cc of brownish ascitic fluid was in the peritoneal cavity with an amylase value of 4,830 units.

terior to the stomach. Edema and adhesions obliterated the lesser omental sac with the pseudocyst being densely adhered to the posterior gastric wall. Outstandingly though, there were no adhesions in the general peritoneal cavity in the face of fluid with such a high amylase value. The intestinal serosa did show some mild irritation and thickening-, probably accounting for the patients' protracted and persistant diarrhea. The liver was biopsied and the abdomen closed. Pathological diagnosis on the liver specimen was "severe fatty metamorphosis with early cirrhosis."

With the findings of brownish ascitic fluid whose amylase content was 4,830 units, a pancreatic pseudocyst;- and a liver—though damaged—but not severely so by irreversible scarring, the diagnosis of pancreatic ascites was established.

After the initial laparotomy, the fever persisted, the diarrhea continued and slowly the ascitic fluid began to re-accumulate. The pleural fluid, right, recurred and was aspirated again.

A second laparotomy was performed on April 10, 1970, and over 2,000 cc of reaccumulated pancreatic ascitic fluid was aspirated. The pancreatic pseudocyst was about orange-size and was 2½ inches to the left of the vertebral column. A duodenotomy was done for the purpose of performing a pancreatogram to establish the presence of a ruptured pancreatic duct. The initial attempt at canulating the pancreatic duct produced a cholangiogram (Fig 3). This cannula was allowed to remain in the common bile duct so as to inhibit the

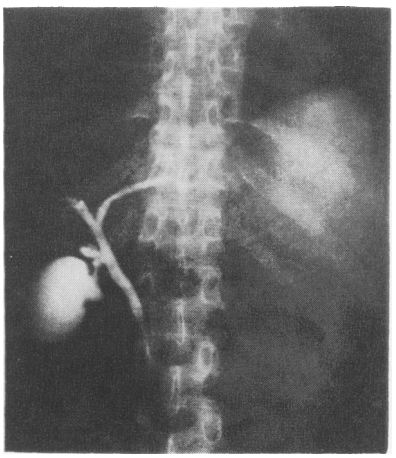

Fig. 3. The polyethylene tubing on first insertion produced a cholangiogram and the tubing was allowed to remain in place and a second portion of tubing was easily inserted in the pancreatic duct for the first canula inhibits a second insertion in the undesired bile duct.

entrance, of a second attempt, in the undesired duct. A second polyethylene cannula was thusly easily and correctly inserted in the pancreatic duct. X-ray opaque material was gently injected by gravity and documentary evidence of a ruptured pancreatic duct into the palpated pancreatic pseudocyst was obtained (Fig 4). The vertical duodenotomy incision was transversely closed. Then a 6 cm. opening was made between the posterior gastric wall and the pancreatic pseudocyst, at

the site of the synechia. The stomal edges of the stomach and cyst were sutured together. Telescoping mushroom and Foley catheters were placed in the pancreatic pseudocyst and in the stomach, and brought out *via* a gastrostomy and a stab wound. The pathology report on the pseudocyst contents was old blood clot and fibrofatty tissue. The patient's weight was at its lowest of

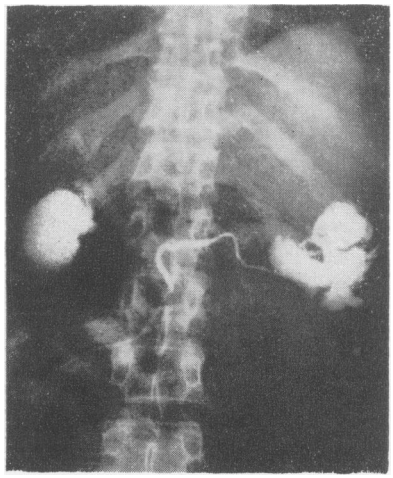

Fig. 4. Hypaque was injected in the pancreatic duct which shows filling of the pancreatic pseudocyst (left of the vertebral column) due to rupture of the pancreatic ductal system.

84½ pounds at the time of surgery (Fig 5). After the performance of cystogastrostomy, her fever subsided, the diarrhea ceased, the ascites has not re-accumulated, her pleural fluid has cleared and not recurred, and her weight has increased to 105 pounds as of July 9, 1970. This is a weight gain of 20 pounds. The patient was discharged from the hospital on May 2, 1970. Though we did not attempt to demonstrate that the pseudocyst was leaking into the general peritoneal cavity by the use of a colored dye, the old bloody contents of the pseudocyst and the associated brownish ascitic fluid in addition to the patient's favorable convalescence are excellent evidence that the pseudocyst leaked into the peritoneal cavity-, producing pancreatic ascites.

DISCUSSION

An amylase determination and a total protein should be gotten on all ascitic fluid in order to establish a precise diagnosis. Our patient had mild cirrhosis of the liver but the ascites was being produced by a pancreatic duct leaking into a pancreatic pseudocyst; which cyst, was in turn, leaking into the general peritoneal cavity. A high amylase value may occur in ascites due to cirhosis [2], but the value is usually *not* so high as in pancreatic ascites. The protein content of cirrhosis fluid is from 0.5 to 1.5 gm%; whereas in pancreatic ascites, the range is from 2.9 to 5.7 gm/100 ml. Also about 20 per cent of patients with cirrhosis or acute hepatitis will show an elevated serum amylase.[4]

Inactivated pancreatic juice can collect insidiously in the peritoneal cavity without great tissue reaction.[5] This out-pouring can continue for months provided that patient takes in sufficient replacing water and electrolytes. Inactivated pancreatic juice can also flow insidiously retroperitoneally. More complicated problems arise when for some reason, the pancreatic juice is activated. It is not entirely clear how activation of trypsinogen is accomplished when no enterokinase is present. Trypsin cannot digest a healthy living cell, some

Fig. 5. Immediately after definitive surgery, the patient weighed 84⅓ pounds. She gained 20 pounds in three months after cystogastrostomy.

injury must exist to the cell. Bacteria and necrotic tissue are assumed to activate the trypsinogen. For example, following trauma to the pancreas,[6] with ductal rupture, shock and prostration do not occur with the frequency seen in acute pancreatitis.

Some of the other causes of ascites in addition to cirrhosis of the liver and pancreatic ascites are: —peritoneal carcinomatosis, polyserositis, tubercu-

lous peritonitis, cardiac failure, thrombosis of the portal vein, renal failure etc.[8] Pancreatic ascites is curable and hence its differential diagnosis is significant. An occasional patient with pancreatic ascites may be cured by abdominal paracentesis for the opening in the pancreatic duct or cyst may spontaneously close. This apparently occurred in the patient, R.M.W., C-148123, aged 43 years, of Grady Memorial Hospital (courtesy of Grady Memorial Hospital Staff). This patient had a laparotomy on March 7, 1963, and 4,000 cc of ascitic fluid was aspirated with a total protein of 3.54 gm% and an amylase value of 560. The liver was grossly normal and biopsy revealed mild fatty metamorphosis. The pancreas was edematous but contained no abnormal mass. No further surgery was performed. No reaccumulation of ascitic fluid had occurred when patient was last seen on June 27, 1967. The patient thusly was relieved of ascites for over four years-, which represents the total duration of the follow-up on this patient. Peritoneal irritation and blocked lymphatic vessels may have caused this patient's fluid accumulation.[7] Twiss and Oppenheim state that almost one-third of patients with cirrhosis have chronic pancreatic disease at autopsy, each on a common etiologic basis, the heavy ingestion of alcohol, frequently.[7]

CONCLUSION

Pancreatic ascites can be promptly controlled by adequate internal drainage of a leaking pancreatic duct and pseudocyst; or by use of whatever form of pancreatic drainage is indicated after operative pancreatography,[7] in the absence of a pancreatic cyst.

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BOSTON UNIVERSITY INTERDISCIPLINARY PROGRAM

Boston University has initiated a unique interdisciplinary Center for Law and Health Sciences to engage faculty and students from several Boston area institutions in the exploration of complex societal problems involving the provision of health services and the advancement of medical technology. The Center's pilot program last summer, financed by the U.S. Public Health Service, was designed to begin research and explore the feasibility of an educational program bringing together faculty and students from different diciplines. The participating graduate students and faculty were selected from Harvard, Massachusetts Institute of Technology, Brandeis and Boston University, and represent such diverse fields as medicine, law, economics, bioengineering, political science, sociology and theology.

The students, most of them divided into interdisciplinary research teams, have been raising such questions as: What effect does health insurance such as Blue Cross/Blue Shield have on the quality and cost of health care? What policy question arise for society from the discouragement or prohibition of the birth of children because of the probability of genetic defects? What is the need, if any, for multiservice centers—including health facilities—in impoverished areas of Boston?

Michael Baram, assistant dean of the Graduate School at MIT, and John P. Wilson, assistant dean of Boston University School of Law, were co-directors of the summer program and of the Center. The Center's chairman is Judge David L. Bazelon, Chief Judge of the U.S. Court of Appeals for the District of Columbia and a well-known expert on the legal aspects of medicine.