SUPPURATIVE PANCREATITIS WITH ASSOCIATED LIVER ABSCESS*

THOMAS A. SHALLOW, M.D., SHERMAN A. EGER, M.D.,
AND
FREDERICK B. WAGNER, JR., M.D.
PHILADELPHIA, PA.

FROM THE SAMUEL D. GROSS SURGICAL DIVISION OF THE JEFFERSON MEDICAL COLLEGE HOSPITAL, PHILADELPHIA, PA.

IN ACCORD with our own experience,[1] recent trends in the literature have emphasized the decreased mortality of acute pancreatitis when treated by the conservative nonoperative method, reserving operation for the complications of this disease, namely, pseudocyst and abscess. Within the past three years 19 cases of acute pancreatitis have been encountered in the Jefferson Medical College Hospital, 12 of the edematous type and seven of the hemorrhagic type. In the hemorrhagic group, pseudocyst formation occurred in two cases, and pancreatic abscess, also, in two cases. It is the purpose of this communication to present in detail one case from this series, unusual because of suppuration followed by hepatic abscess, to discuss its pathogenesis and evaluate the factors contributing to recovery.

Case Report.—A. T., female, age 55, was admitted to the Surgical "A" Ward Service of the Jefferson Medical College Hospital, September 30, 1944, with the chief complaint of epigastric and right upper abdominal pain.

The patient had been well until three days prior to admission, at which time she first experienced moderately severe epigastric and right upper abdominal pain. The pain was constant, did not radiate, and was accompanied by nausea and frequent episodes of vomiting of greenish-yellow material. She also had diarrhea, with passage of four loose stools daily for two days. From the onset of the attack she remained confined to bed and had fever.

The past medical history revealed that the patient previously had always enjoyed excellent health. She had been free of indigestion and was able to tolerate all types of food.

Physical Examination.—The patient was a moderately obese middle-aged Italian woman who was apprehensive, slightly irrational, and appeared acutely ill. Temperature 103° F.; pulse 130; respirations 26; and blood pressure 140/90. She was slightly jaundiced and markedly dehydrated. The heart and lungs were essentially normal. On inspection, the abdomen was slightly distended and abdominal respiratory movement was restricted. In the right upper quadrant there were exquisite tenderness, marked rigidity and rebound tenderness. No mass was noted at this time. Rectal examination, as well as the remainder of the physical examination, were essentially normal.

Laboratory Data.—Examination of the blood showed hemoglobin 78 per cent; red cells 4,000,000; white cells 13,600, with polymorphonuclear cells 57 per cent (8 per cent young forms), lymphocytes 39 per cent, monocytes 3 per cent, and basophils 1 per cent. Uranalysis revealed slight albuminuria, but was otherwise negative. Wassermann and Kahn serologic reactions were negative. The blood urea nitrogen was 9.0 mg., sugar 114 mg., and serum proteins 6.5 Gm. Van den Bergh test showed a positive direct reaction, with serum bilirubin of 2.1 mg. Bromsulfalein liver function study showed 20 per cent dye retention. The prothrombin time was 68 per cent of average normal.

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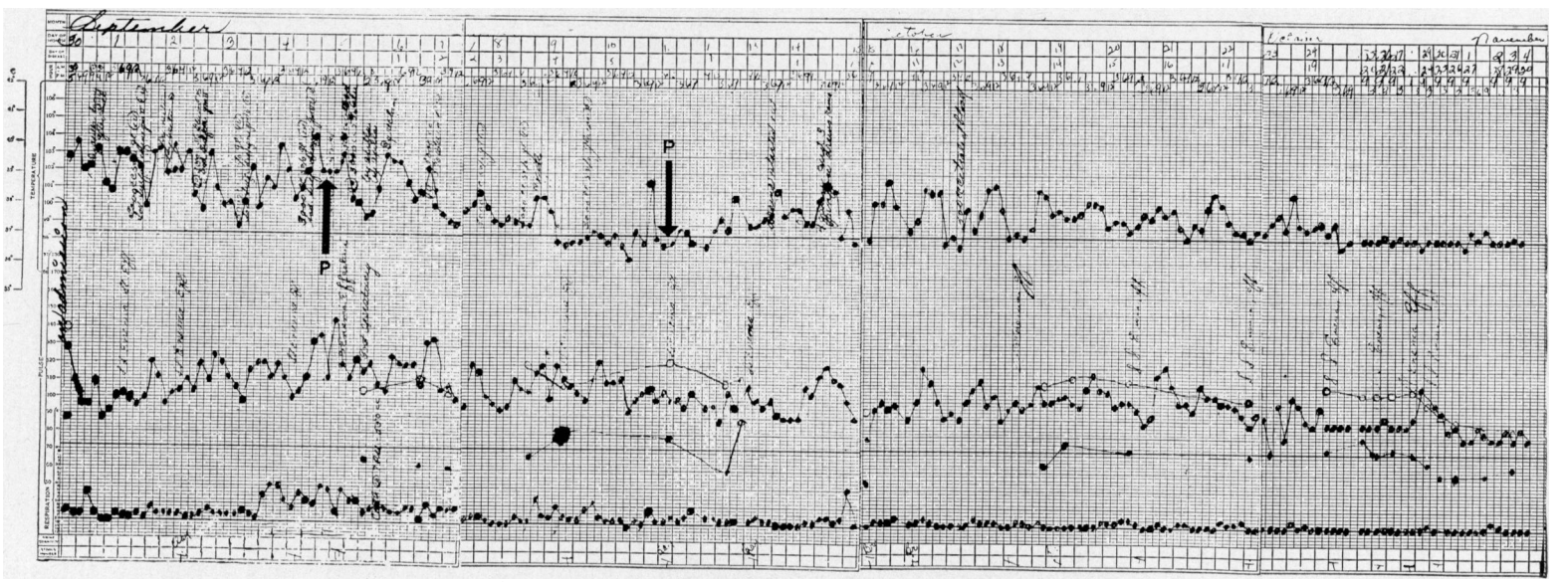

CHART 1.—Vital signs during first hospital admission. Large arrows (P) indicate start and termination of administration of penicillin.

The serum amylase, determined six days following the onset of symptoms, was normal. Blood culture revealed no growth in 48 hours. A plain roentgenogram of the abdomen was negative.

Preoperative Course.—It was thought that the patient had acute calculous cholecystitis. Accordingly, a conservative plan of treatment was instituted, consisting of Wangensteen suction; 5 per cent glucose in normal saline solution, with vitamin B_1 100 mg., vitamin C 100 mg., and vitamin K 4 mg. intravenously daily; also sodium sulfadiazine 5 Gm. intravenously daily. In spite of this therapy the patient failed to improve, and during the ensuing three days a mass became palpable in the right upper quadrant extending four fingers' breadth below the right costal margin. The temperature fluctuated daily between 100° F. and 103° F., and the pulse ranged between 100 and 120. On the fourth day of admission the patient developed grunting, shallow respirations at a rate of between 30 to 40 a minute, but there was no evidence of pneumonitis on physical examination. The leukocyte count rose to 18,000 by the third day of admission, 20,000 by the fourth, and 26,000 by the fifth day. At this time the administration of sodium sulfadiazine was discontinued and penicillin administered intramuscularly at two-hour intervals, with a total dosage of 100,000 Oxford units the first 24 hours and 50,000 units daily thereafter (Chart 1). On the sixth day the patient was operated upon, with a preoperative diagnosis of empyema of the gallbladder, and the contemplated procedure was cholecystostomy.

Operation.—Doctor Shallow, October 5, 1944: Under pontocaine spinal anesthesia, a right upper rectus muscle-splitting incision was made, and a slight excess of odorless turbid fluid was encountered in the peritoneal cavity. The gallbladder and biliary tract were free of any inflammatory process, easily compressible, and no stones were palpable. The anterior surface and lesser curvature of the stomach, as well as the duodenum, were next examined and showed no evidence of perforation. The gastrohepatic omentum was markedly congested, edematous, spotted with patchy areas of fat necrosis, and overlay a boggy mass protruding into the lesser peritoneal cavity. The gastrocolic omentum was likewise edematous and involved by scattered patches of fat necrosis. After walling-off the coils of bowel with saline gauze packs the gastrohepatic omentum was incised, and the lesser peritoneal cavity exposed. The posterior wall of the stomach and duodenum showed no evidence of perforation. Within the body of the pancreas was a fluctuant mass, which, following incision, was found to contain about eight ounces of extremely foul-smelling material of thin consistency and dish water color, in which small pieces of necrotic pancreatic tissue were suspended. Subsequent smear of this material revealed necrotic cellular débris, many gram-positive cocci, and the culture was positive for *Streptococcus hemolyticus.* Most of the body of the pancreas was destroyed by the abscess.

The quadrate lobe of the liver, on the inferior surface between the falciform ligament and the gallbladder, presented a slight bulge, suggestive of an underlying abscess. An aspirating needle was introduced and 5 cc. of pus was readily obtained. The liver was then incised over this area and a finger introduced. Two abscess cavities, in direct communication, were palpated. Four ounces of foul-smelling, thick, creamy-yellow material were aspirated, which on subsequent smear showed necrotic cellular débris, a few gram-positive cocci, and on culture were positive for *Streptococcus hemolyticus.* This hepatic abscess was entirely independent, showing no continuity or contiguity with the one in the pancreas.

Two iodoform packs and a Penrose drain were inserted into the abscess cavity of each organ and an additional Penrose drain was placed in the kidney pouch adjacent to the foramen of Winslow. These were brought out at the upper pole of the wound, which was then closed in layers. The patient received 500 cc. of citrated blood during the operation, which lasted 30 minutes, and she left in fair condition.

Postoperative Course.—The immediate postoperative response and convalescence

were essentially uneventful. She received 5 per cent glucose in saline, containing vitamins B, C, and K intravenously daily for four days; Wangensteen suction for four days; sodium sulfadiazine 5 Gm. intravenously daily for two days; penicillin 50,000 Oxford units daily in divided doses every two hours intramuscularly for six days (Chart 1); and 500 cc. of citrated blood on the seventh and twelfth days postoperatively.

Postoperative Laboratory Data.—Blood count (first day) showed hemoglobin 71 per cent; red cells 3,400,000; white cells 19,000 and 5,000 (fifteenth day). Repeated uranalyses were normal. The serum amylase was normal (first day); urea nitrogen 21.4 mg. (first day) and 18.8 mg. (fourth day); serum calcium 9.2 mg. (fourth day); positive direct van den Bergh reaction, with serum bilirubin 1.7 mg. (sixth day) and 0.5 mg. (21st day); bromsulfalein liver function study showed 5 per cent dye retention (sixth day), and no dye retention (21st day); and blood sugar 99 mg. (15th day).

The patient drained purulent material profusely through the wound during the first week, requiring change of dressings several times daily. During this time the jaundice began to subside, the temperature and pulse rate decreased, and the patient's mental state cleared. The drains were started out on the seventh day and completely removed on the tenth. The wound became mildly infected but responded to 0.8 per cent sulfanilamide irrigations and compresses. By the 20th day the jaundice had entirely subsided, the temperature and pulse had become normal and remained so until the patient was discharged, November 4, 1944, the 30th day postoperatively (Chart 1). At this time there was still slight drainage from the wound.

Second Hospital Admission.—The patient was readmitted 20 days later, November 24, 1944. Slight drainage from the wound had persisted, and three days before admission she developed fever.

Physical Examination.—Temperature 100.4° F.; pulse 110, and respirations 20. There was no jaundice, and her general appearance was the same as on discharge from the hospital. An area of tenderness and fluctuation was present in the upper half of the wound.

Laboratory Data.—Blood count revealed hemoglobin 65 per cent; red blood cells 3,900,000; and white cells 15,000. Three routine uranalyses were normal. The serum bilirubin was 0.6 mg.; bromsulfalein liver function test showed no dye retention; prothrombin time was 52 per cent of normal; and the serum proteins were 5.1 Gm. A plain roentgenogram of the abdomen was essentially negative.

Subsequent Treatment and Progress.—The tip of an hemostat was introduced into the upper pole of the wound, and about an ounce of greenish pus, positive on culture for *Streptococcus hemolyticus* and *Staphylococcus aureus*, obtained. The wound was irrigated twice daily, and compressed with 0.8 per cent sulfanilamide solution, and sulfadiazine was given orally so as to maintain a blood level around 5 mg. Vitamin B and K and bile salts were administered orally. Ferrous sulfate was given orally, and 500 cc. of citrated blood intravenously, following which the hemoglobin rose to 71 per cent. The patient was discharged, December 9, 1944, 15 days after admission, at which time drainage from the wound was almost negligible.

The patient was last seen, January 5, 1945, three months postoperatively. Her appetite was good, she had gained ten pounds in weight, was free of fever, and drainage from the wound had ceased.

COMMENT.—Suppurative pancreatitis usually occurs as a complication of hemorrhagic pancreatitis, resulting from bacterial invasion of the necrotic tissue. Less frequently it may occur as a result of ascending infection through the pancreatic duct. Pancreatic abscess has been produced in animals by injection of colon bacilli into the pancreatic duct. Ascending infection may occur when the biliary tract is the seat of inflammation, as in cholelithiasis

with cholangitis, with reflux of infected bile into the pancreatic duct in the presence of obstruction at the ampulla of Vater (cases of Mayo Robson[2]). Bevan[3] and Batchelor[4] have each reported a case associated with cholelithiasis. According to Opie,[5] pancreatic calculi are associated with pancreatic suppuration with relative frequency, and carcinoma compressing the pancreatic duct or carcinoma of the ampulla of Vater (case of Pearce[6]) may, likewise, be

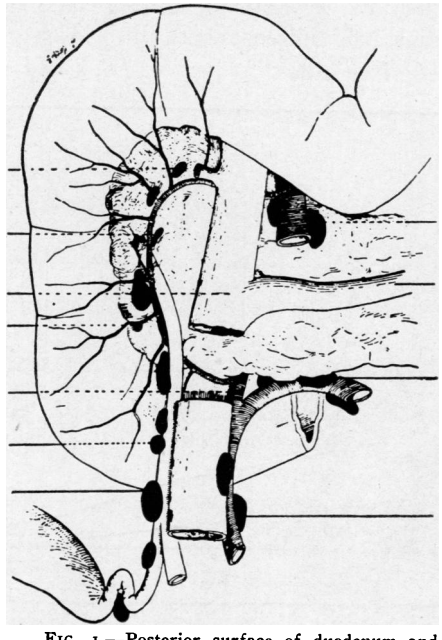

FIG. 1.—Posterior surface of duodenum and pancreas, showing relation of lymph nodes along the hepatic artery, cystic and common bile duct chains to those corresponding to the vascular arch behind the pancreas (Poirier, Cuneo, and Delamere).

associated with abscess of this gland. Pancreatic abscess may arise by extension from an adjacent organ by contiguity or through the lymphatics. Thus, abscess in the tail of the pancreas secondary to a perforated gastric malignant tumor has been reported by Ochsner.[7] Finally, hematogenous spread of infection to the pancreas may occur in rare instances.

Pancreatic abscess is usually associated with peritonitis of the lesser peritoneal cavity, and perforation into the general peritoneal cavity may occur. According to Opie[5] rupture into the stomach or intestine has followed, and vomiting or discharge from the bowel of purulent or hemorrhagic material has been reported. Associated thrombophlebitis of the splenic and portal vein may result in metastatic liver abscess. Destruction of a major portion of the gland may lead to the development of diabetes mellitus, as in the case of Beller and Nach.[8]

We believe that the sequence of events in the present case consisted of an attack of acute hemorrhagic pancreatitis followed by bacterial invasion, involvement of the pancreatic veins, and perhaps the splenic vein, by the

infection, and seeding of bacteria or septic emboli into the liver through the portal vein. The spread of infection from pancreas to liver by the lymphatics is also a possibility (Fig. 1). The finding of a normal preoperative serum amylase value on the sixth day following onset of the attack is in keeping with the well-substantiated fact, both in the literature and our own cases,[1] that the level usually falls to normal within 12 to 72 hours, and only in the minority of cases is elevated beyond this time (Chart 2).

The absence of biliary tract disease, both in the past history and operative findings, is of interest, since about 70 per cent of cases of acute pancreatitis

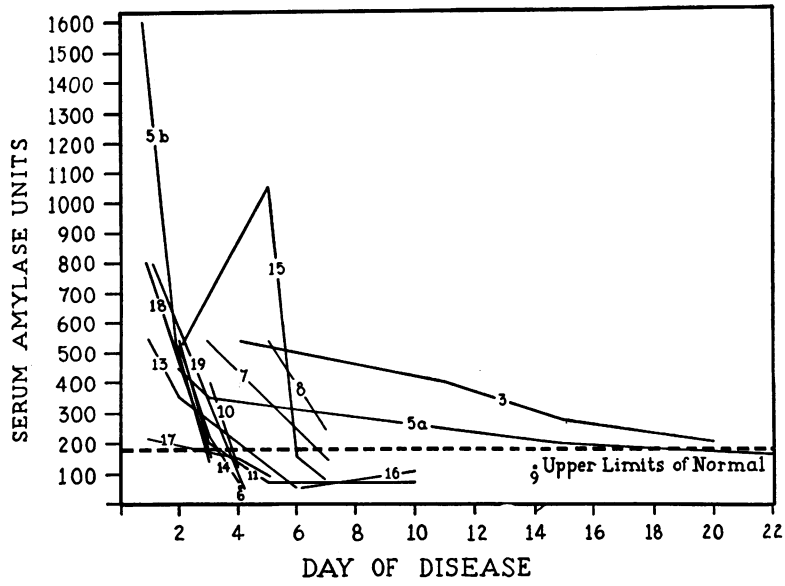

CHART 2.—Curves showing elevated serum amylase during acute attack, with fall as attack subsides. (Numbers on curves correspond to cases in authors' series.)

are associated with lesions in this system. The jaundice in this case was mainly due to hepatitis and possibly, to a lesser degree, to compression of the common bile duct in its transit through the gland by edema. Posterior perforation of a peptic ulcer into the pancreas was definitely ruled out at operation. The possibility of an hematogenous origin of the pancreatic abscess seems unlikely in view of the lack of clinical evidence of a primary focus elsewhere, and the negative blood culture.

According to the recent work of Edmondson and Berne,[9] serum calcium findings below 9 mg. per 100 cc. of blood usually occur in cases of pancreatic necrosis some time between the second and 15th day of the disease, and values below 7 mg. indicate a fatal prognosis. In keeping with this work, the value in the present case of 9.2 mg. on the 14th day following onset of the attack seemed to prognosticate a favorable outcome.

In this case the loss of pancreatic tissue did not result in diabetes mellitus. According to the work of Allen,[10] destruction of seven-eighths, or more, of the gland is necessary for a diabetic state to ensue.

The importance of scrupulous pre- and postoperative care in this serious condition is self-evident. Supportive measures, such as fluids intravenously, plasma, blood, and vitamins; Wangensteen suction to combat ileus; and chemotherapy with sulfonamides and penicillin are vital adjuncts in treatment. Because the infection was associated with gram-positive cocci, we believe that the use of penicillin for one week was an important factor in the patient's recovery.

CONCLUSIONS

1. Although the conservative nonoperative management of acute pancreatitis has been stressed in the recent literature, this case illustrates a clear-cut indication for surgical intervention, namely, abscess formation.

2. The finding of an associated liver abscess at operation is unusual.

3. Meticulous pre- and postoperative care, including the use of penicillin, are vital adjuncts to carefully timed and adequate surgical intervention.

4. Suppurative pancreatitis with associated liver abscess is a grave but not hopeless condition.

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