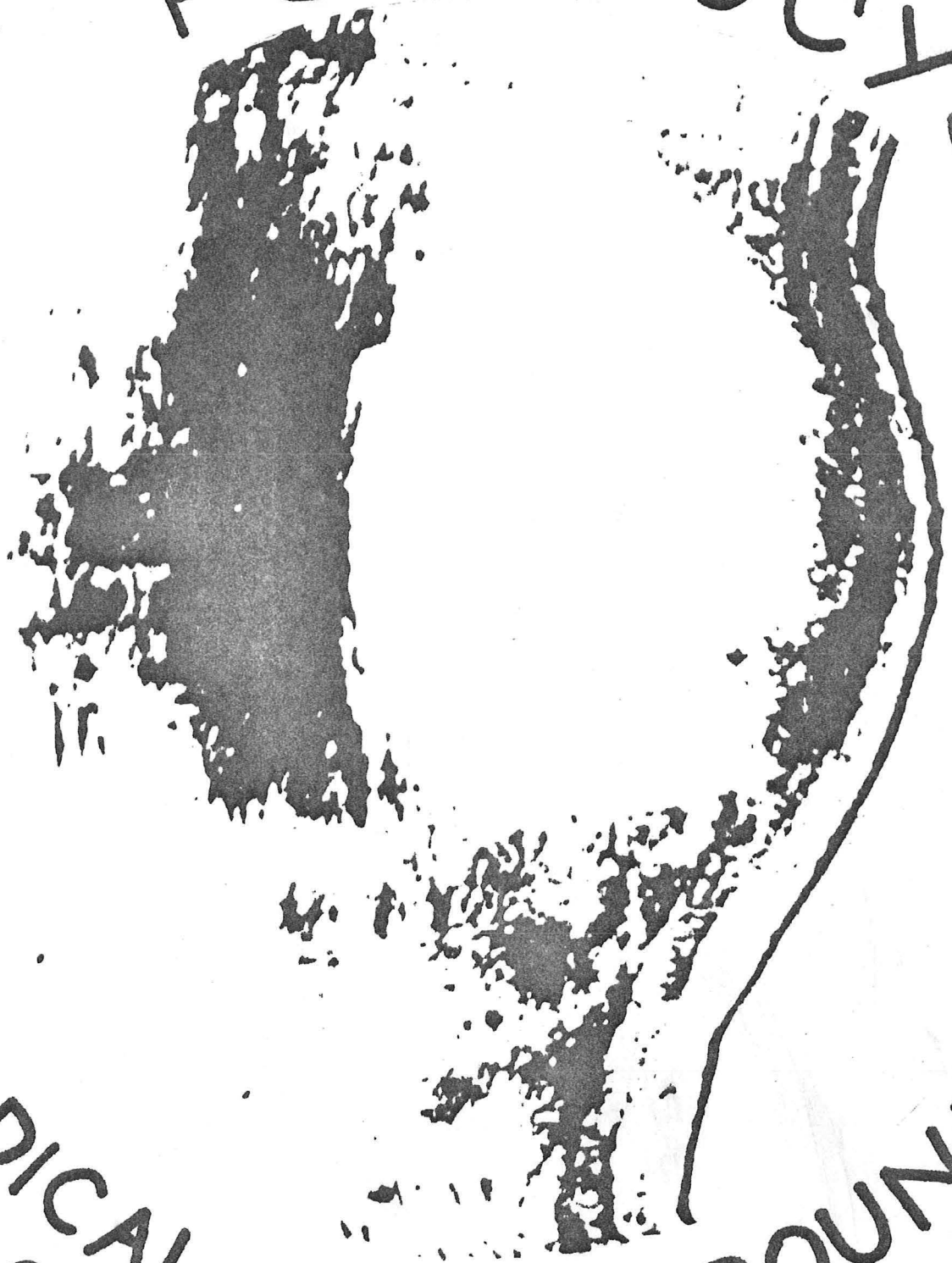
PANCREATIC PSEUDOCYST



MEDICAL GRAND ROUNDS

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TABLE 1

DIFFERENTIAL DIAGNOSIS OF ABDOMINAL CYSTS

- Pancreatic cysts
- Ovarian cysts
- Renal cyst, adrenal cyst, hydronephrosis and cystic kidney tumor
- Mesenteric cyst
- Duplication cyst
- Splenic cyst, liver cyst
- Echinococcus cyst
- Giant sigmoid diverticulum
- Abscesses
- Dissecting aneurysm

INTRODUCTION

Pancreatitis may be acute, chronic, or chronic with acute exacerbations. Each of these types may show a different natural history and resulting complications may also differ considerably.

However, in any type of pancreatitis in which an acute inflammatory process is affecting the organ as a whole or in part, pancreatic pseudocyst is one important possible complication.

Pancreatic pseudocyst is so important in the consideration of pancreatitis, in fact, that it is virtually an entity in its own right, with its own set of pathogenetic, diagnostic, complication and therapeutic features.

The purpose of this presentation is to focus on these features of pancreatic pseudocyst.

TABLE 2

PANCREATIC CYSTS

- Pseudocyst (80% of all pancreatic cysts)
  - Inflammatory
  - Traumatic
- Retention cyst
- Neoplastic cyst
  - Cystadenoma
  - Cystadenocarcinoma
- Miscellaneous
  - Congenital cyst
  - Dermoid cyst
  - Angiocyst

HISTORICAL ASPECTS

Andreas Vesalius (1514-1564) is credited for describing the pancreas (De Humani Corporis Fabrica, 1555). Wirsung in 1642 in Padua described the ductal anatomy. In 1867, Lucke (1), a German gynecologist, reported drainage of a pancreatic pseudocyst by insertion of a trochar in a patient who was thought to have an ovarian cyst. Unfortunately, this treatment resulted in peritonitis and death. In 1882, Bozeman (2) (see page 22), also a gynecologist, excised a large pancreatic pseudocyst and presented the specimen in a meeting of the New York Pathological Society. Marsupialization of a pancreatic pseudocyst was performed by Gussenbauer in 1883 (3). In 1911, Ombredanne was the first to use internal drainage of a pseudocyst via the duodenum (4).

CASE #1 (PMH #24-28-83) ["*After all, a typical case of pseudocyst*"]

42 y/o black man. Heavy drinker. Treated for pulmonary tuberculosis in past. Five years prior to admission, a splenectomy was performed following a gunshot wound to the abdomen. Although there was a history of occasional epigastric pain, it was not severe enough to seek medical attention. Thus, there was no documented history of pancreatitis when he presented with a LUQ abdominal mass to the hospital.

On multiple determinations, amylase was either normal or borderline elevated.
KUB and chest x-ray: Abnormal configuration of gastric bubble which was pushed medially. The left dome of the diaphragm was considerably higher than the right.
IVP: Left kidney pushed downward and poor filling of calyceal system.
UGI: Large round fundic mass.
Sonography: Mass free of echos (cystic).
Gastroscopy: Large mass in fundus.
Celiac arteriogram: Large avascular mass in LUQ displacing the stomach to the left in the region of the gastric fundus. No tumor vessels. Read as probable large necrotic leiomyoma of the body of the stomach.

After a 4 week observation period, the patient underwent laparotomy. The pre-operative diagnosis was necrotizing gastric leiomyoma versus pancreatic pseudocyst. A huge mass was found adherent to the fundus, left hemi-diaphragm and left lobe of the liver. An anterior gastrostomy was done and on needling the mass, yellow fluid was obtained (amylase concentration: 6400). A cell block obtained on the cyst fluid showed multiple neutrophils, scant macrophages and fibrin, but no malignant cells. Histology revealed pancreatic pseudocyst wall. A gastrocystostomy was performed and the patient made an uneventful recovery. No pancreatic problems so far during a 1 year follow-up.

Discussion of Case #1: The patient presented without a history of pancreatitis and the intragastric position of the cystic mass suggested necrotic leiomyoma. Although many diagnostic tests were performed, the definite diagnoses was only established at laparotomy.

PATHOGENESIS

Figure 1 organizes and summarizes our knowledge of the mechanisms of tissue damage in acute pancreatitis, a process out of which pseudocyst may develop (5). Across the top of the scheme are displayed the etiologic factors in the development of pancreatitis; the big three are alcoholism, biliary tract disease and trauma. These etiologic factors have in common two important features, namely, the ability to injure the pancreatic acinar cell and/or to activate pancreatic enzymes.

The pancreas is normally protected from the action of its own enzymes by the fact that the potentially destructive enzymes are formed, stored and secreted in the precursor state. Activation of the precursor to their proteolytic and lipolytic enzymes ordinarily takes place at the appropriate site of enzyme action, that is, in the gut lumen. The occasion of pancreatic damage, however, may be as yet uncertain mechanisms resulting in activation of these enzymes within the substance of the pancreas, which results in a process of autodigestion and further tissue destruction (6).

The enzymes involved are trypsin, the proteolytic enzyme first recognized by Pavlov. Trypsinogen is activated by enterokinase and converted to trypsin, trypsin itself further activates trypsinogen as well as most of the other enzyme precursors to their respective enzymes. Chymotrypsin is another such proteolytic enzyme.

Elastase is particularly important since it breaks down collagen and is believed to be responsible in great part for vascular damage with resultant hemorrhage - in other words, the hemorrhagic part of hemorrhagic pancreatitis is thought to be due to the action of elastase.

Kallikrein and phospholipase and their importance in pancreatitis have been emphasized only in recent years. Kallikrein induced edema and vascular permeability, and may be implicated in the shock which sometimes is a component of acute pancreatitis; activated phospholipase causes marked pancreatic necrosis and converts lecithin to lysolecithin which is present in increased amounts in severe or fatal pancreatitis. Lipase is responsible for fat necrosis in pancreatic and surrounding tissue during acute pancreatitis. Lipase changes insoluble neutral fat to fatty acids and glycerol.

The net result of these enzymatic actions may therefore be viewed as a process of autodigestion of the gland, and the stage is set for a variety of complications, including the development of a pancreatic pseudocyst.

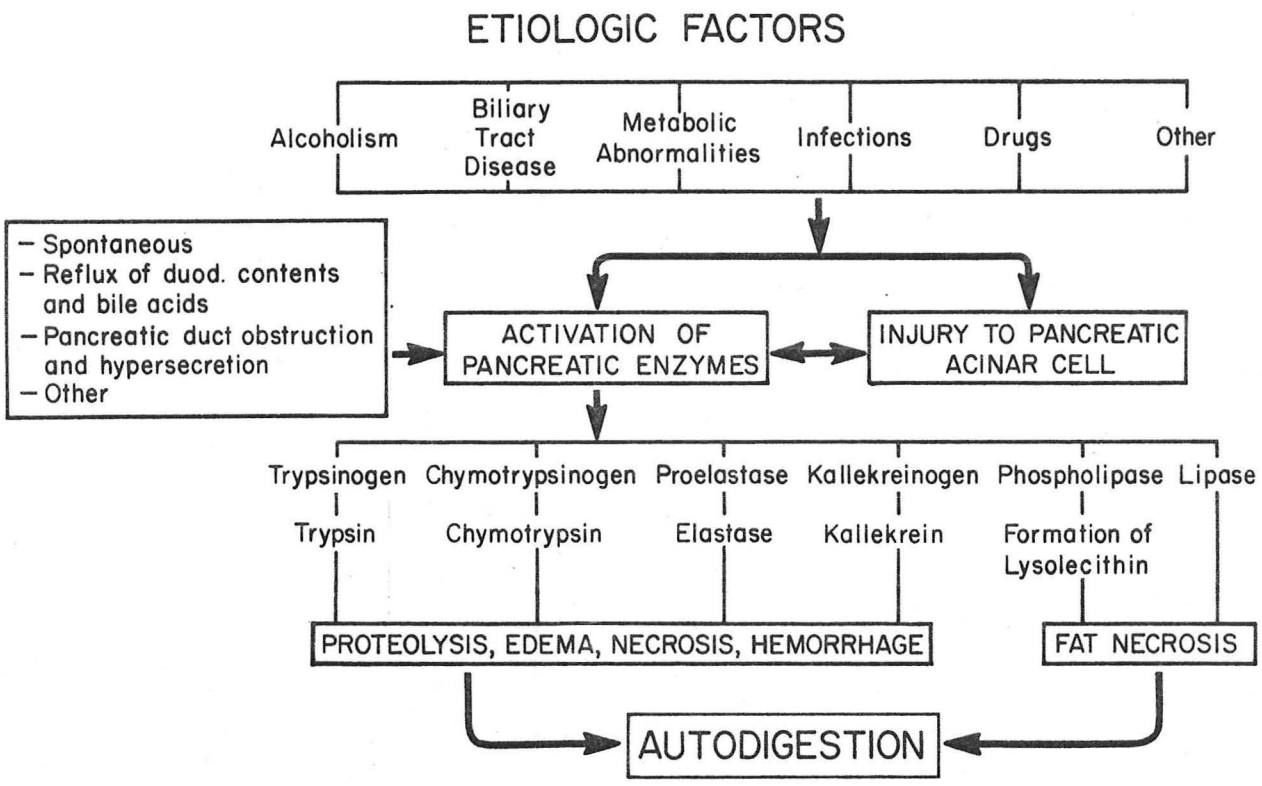

Figure 1.: Etiology and pathogenesis of pancreatitis [Modified from (5) and (6)]

LOCATION OF PSEUDOCYSTS

Pancreatic pseudocysts may develop in or from the head, body or tail of the pancreas (Figure 2) and they consist of collections of blood, pancreatic enzymes, tissue fluid and debris from the pancreas, which develop over a period of days or

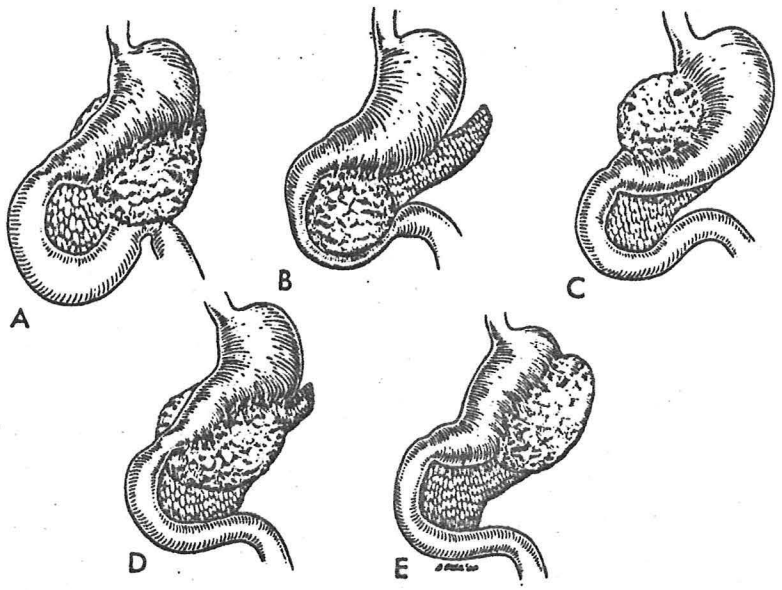

Figure 2.: Pseudocysts may: (A) push against the ligament of Treitz, obstructing the distal duodenum, (B) impress the duodenal C-loop, (C) push the stomach to the left or downward, (D) upwards, or (E) to the right. [*Elliott, 1975, (7)*].

weeks after the onset of acute pancreatitis, and which accumulated in a necrotic portion of the pancreas or in the lesser sac (7). The wall of the pseudocyst consists of necrotic tissue, granulation tissue and fibrous tissue, but no epithelium, hence, the term "pseudocyst".

While disruption of the pancreatic ductal system is a prerequisite for cyst formation, the cystic spaces often connect with ducts draining the part of the gland distal to the cyst. Thus, while drainage or outflow is impaired or interrupted, secretion of some or all of the remaining functional glandular tissue enters the cystic space but cannot escape. This process enhances growth or persistence of the pseudocyst.

Figure 2 illustrates how the pseudocyst may push the stomach and duodenum in a number of different directions. Other adjacent structures or organs may be affected. The colon may be displaced or changes in the mucosal pattern of a segment of transverse colon may be observed. Mucosal irritation, edema and sometimes stenosis may be difficult to differentiate from colon malignancy. A pseudo-

cyst may invade the spleen (8-10) or displace it. Occasionally, the left kidney is affected by a pseudocyst leading to downward displacement, compression or distortion (11). Expansion of the cyst can go in every direction, some extend as far as the groin (12), the porta hepatis (13) and the mediastinum (14,15).

EXPERIMENTAL PSEUDOCYST

In 1957, Warren and coworkers succeeded to construct a pseudocyst model in dogs (16). The procedure was difficult and had an overall mortality of more than 50%. In the first step, an artificial cavity was created in the lesser sac by inserting a sponge-filled polyethylene bag. Six to eight weeks later, the polyethylene bag was removed through an anterior incision. Then a 2 cm ellipse of duodenal wall surrounding the major pancreatic duct was excised. This segment of the duodenum was then anastomosed to a 2 cm opening in the right posterolateral wall of the sac. The duodenum was closed. In a third operation, drainage of the cyst was performed either by cystogastrostomy or cystojejunostomy.

SYMPTOMS

The clinical features of pancreatic pseudocyst are highly variable and therefore frequently misinterpreted. Incorrect pre-operative diagnoses have been reported in up to 50% of cases (17,18). The incidence parallels that for pancreatitis which is common between 30 and 50 years of age. In about 5% of patients, there is no history or clinical evidence of pre-existing pancreatitis. In those with a history of pancreatitis, the interval to pseudocyst manifestation is usually 6 weeks but ranges from about 3 weeks to 14 months (13).

Most patients experience pain which usually is located in the epigastrium or left upper quadrant of the abdomen, and which radiates to the back in approximately one third. Nausea and vomiting occur in about 60% of patients with pseudocyst. Weight loss is manifested in slightly more than half the patients. A low grade fever is less common and generally parallels the degree of active pancreatitis present. Diarrhea may develop in some patients. Rarely patients with pseudocyst are asymptomatic (Table 3).

TABLE 3

SYMPTOMS IN PANCREATIC PSEUDOCYST

Pain	95%
Nausea and vomiting	60%
Weight loss	55%
Fever	25%
Diarrhea	15%
Asymptomatic	5%

SIGNS

A palpable abdominal mass is universally accepted as the most prominent feature on physical examination. In many patients, upper abdominal fullness was misinterpreted as being "liver" and frequently a mass is not palpable until after it has been outlined on roentgenologic examination. Abdominal tenderness, usually mild, is noted in most of those who suffer pain. Transient pleural effusion or left lower lobe pulmonary changes or both are encountered in about 30%. Icterus is noted in about 20% of patients. Pancreatic ascites and pancreatic pleural effusion (due to internal fistulas) once thought to be extremely rare are now recognized more frequently (see page 13).

DIAGNOSIS

Laboratory Tests

Mild leucocytosis is frequent. Only one half of patients demonstrate elevated serum amylase or lipase values (13,19). Urinary excretion of amylase and its relation to creatinin clearance might be more useful. In acute pancreatitis and in pancreatic pseudocyst more amylase is delivered to the urine by way of clearance from the serum than is normally the case (20). The cause for this increased urinary excretion of amylase in pancreatitis is unknown, but the mechanism by which it occurs has been shown to be a reduced tubular reabsorption of amylase in patients with pancreatitis (21).

X-ray Studies

KUB: The supine radiograph of the abdomen, properly performed and interpreted, may be one of the most helpful of all the diagnostic modalities (22-24). Pancreatic calcifications may indicate chronic pancreatitis. Signs of retroperitoneal disease will include obliteration of one or both ileopsoas shadows, loss or displacement of a renal shadow, loss of the flank or fat stripe laterally, and mass effect on a hollow, air-filled viscus.

The colon cut-off sign is a valuable indicator of upper abdominal inflammatory disease; in this sign, gas will be seen to be present in the colon proximal to a point from mid-transverse to splenic flexure of the colon. Distal to that point, no gas is seen. A so-called sentinel loop of gas-filled small bowel is often observed in the upper abdomen, which is actually a localized ileus of a short segment of small intestine secondary to a focus of inflammatory disease such as pancreatitis. If pancreatic abscess is present, a typical x-ray finding may be extraluminal gas in the lesser sac or the "soap bubble" pattern characteristic of retroperitoneal sepsis.

UGI: Upper gastrointestinal series with barium meal is particularly valuable in both pancreatitis and pseudocyst (22-24). The changes of adjacent inflammation include spiculation of the wall of the stomach and duodenum, retained gastric secretions, thick and edematous rugae of stomach and duodenum, widening of the duodenal C-loop with effacement of the inner border and retraction at the ampullary region to produce the so-called "inverted-3" sign, abnormalities of the papilla, outlet obstruction or duodenal obstruction, deviation of portions

of the stomach, duodenum or other parts of the bowel by mass. Those signs which indicate the presence of a mass are particularly helpful in recognizing a pseudocyst, but the observer must always know that a given mass seen in the patient with pancreatitis may be either cystic or a phlegmone in nature. The larger and more persistent the mass, the more likely it is to be a pseudocyst, but this rule is by no means absolute, and certainly all pseudocysts are not large. Hypotonic duodenography may be of particular value in selected cases in which the character of the duodenal changes from either adjacent inflammation or pressure from a pancreatic mass may be better delineated (24).

Other radiographic studies may provide limited information. The gallbladder is often not visualized or is poorly visualized during an acute bout of pancreatitis and often in the presence of pseudocyst as well. However, recently in a study on 42 patients, it was shown that on OCG the gallbladder visualizes in 90% of patients at the time of resumption of a solid diet following alcoholic pancreatitis (25). Nevertheless, it is important to remember that nonvisualization of the gallbladder during acute pancreatitis does not in itself mean biliary tract disease is present.

Radiographic examination of the biliary tract by other means such as IVC is important, however, since stones are a possible etiologic factor in pancreatitis. In rare cases, particularly if jaundice is a major symptom, percutaneous cholangiogram may be helpful to identify the site and shape of the obstruction. The intravenous pyelogram may reveal displacement of one kidney, usually the left, from a pseudocyst. In rare cases, one kidney will be nonfunctional secondary to acute pancreatitis or pancreatic pseudocyst.

Angiography: Selective celiac and superior mesenteric and subselective pancreatic angiography may reveal vascular changes suggesting pancreatitis and pseudocyst (26,27). In acute pancreatitis, no diagnostic changes may be appreciated, but later, changes in vessel size, increased vascularity, increased tissue "staining" may be seen. With chronicity arterial beading is observed as well as narrowing of adjacent hepatic and splenic vessels. In pseudocyst mass effects are demonstrable and are the typical findings. Stretching of arteries and veins around the lesion is characteristic, stretching of intrapancreatic arteries almost always means pseudocyst.

ER[C]P (Endoscopic Retrograde [Cholangio]Pancreatography): In this procedure, insertion of a fine catheter into the papilla of Vater is accomplished by fiberendoscopic manipulation and a contrast agent is injected directly into the pancreatic (and/or common bile) duct. In a small series, Silvis et al (28) demonstrated that 12 out of 16 pseudocysts could be filled by this technique. However, whenever a cyst starts to fill, injection of the contrast medium should be discontinued to avoid infection. Other findings may be compression or abrupt stop of a duct due to pseudocyst. Such an obstruction of the main pancreatic duct is of course an unspecific finding and can also be caused by tumor. In my opinion, ERP is not indicated for the diagnosis of "uncomplicated" pseudocyst (Pancreatic Ascites, see page 13). However, since ERP can demonstrate other abnormalities such as caliber irregularities, stenosis or dilatation, small pseudocysts will occasionally be found by this method in the patient with intractable chronic pancreatitis in whom visualization of the ductal system is important with respect to possible future surgery. ERP is difficult to perform and despite great experience, it may be impossible to cannulate the papilla or to visualize the pancreatic duct. The risk of the procedure is not small enough to recommend its wide use. It has a mortality of about 0.2% and such complications as duodenal perforation,

acute pancreatitis and septicemia have been reported (29-31). If a surgical lesion is found by this technique, such as stenosis and distal duct dilatation, or cysts that qualify for drainage, then the interval between ERCP and surgery should be short (not exceeding 24 hours).

ULTRASOUND

The accuracy of the B-mode ultrasonic scanning for evaluation of fluid filled masses in the abdomen has made it the method of choice for evaluating suspected pancreatic pseudocysts (32,33). In order to recognize pseudocyst formation, sonography has become a routine test for every patient with acute or exacerbated chronic recurrent pancreatitis. Advantages of the procedure are the possibility to be able to distinguish cystic from solid masses, to be noninvasive and relatively cheap. Cysts as small as 2 cm in size can be identified. Only a very few studies of ultrasound in pancreatitis are available at this time (34,35). However, the following interesting results have been reported: First, cyst formation was found as frequent as in 50% of patients. Second, cysts were found to be multilocular in about 15%. Finally, the false positive as well as false negative rate of the test (with reference to surgery or autopsy) was about 8%, which is much better than any other test.

CAT-SCAN

Computerized axial (or computer assisted) tomography (CAT) whole body scanning is in the process of being added to the armamentarium of noninvasive radiographic procedures (36,37). It is yet too early to know the true value of this method. It may be no better than ultrasonography in the diagnosis of pancreatic disease (38). Disadvantages are the high costs and the radiation exposure. CAT-scanning, however, appears to be useful in patients where excessive bowel gas prevents successful visualization with ultrasound and also in obese patients in whom the degree of sound attenuation is great enough to limit diagnostic accuracy. The advent of the fast scanners (5 seconds) utilizing a 360 degree rotational fan beam has reduced patient radiation exposure and virtually eliminated motion artifacts. This is important in severely ill patients who cannot hold their breath for more than a few seconds. Like with ultrasound, solid and cystic lesions can be easily discriminated. Dilute water soluble contrast material by mouth is routinely employed to opacify the stomach, duodenum, and proximal small bowel loops to more clearly separate the pancreas from these structures.

FREQUENCY

Pseudocysts occur in an estimated 10% of cases of chronic pancreatitis (17). New figures obtained by sonography suggest that cyst formation might be demonstrable in about 50% of cases (34). Thus, pseudocysts are seen with a frequency of 2 to 20 per 100,000 hospital admissions.

At Parkland Memorial Hospital, 130 cases were registered in the Department of Surgery between 1962 and 1976 (10 cases per year). In 1975 and 1976, a total of 15 cases per year was seen on the Medical and Surgical Service combined.

NATURAL HISTORY

The following questions about the natural history of pancreatic pseudocysts will be discussed:

- Do they regress spontaneously?
- What are the hazards of watchful following a patient with pseudocyst?
- How often do they rupture?
- How often does bleeding occur?
- Significance of pancreatic ascites and pleural effusion?

CASE #2 (VA #463-54-7846) *["After all, spontaneous pseudocyst regression"]*

42 y/o black man. The patient had been admitted several times for pancreatitis in the past. One month following his last episode of pancreatitis, he returned to the hospital (December 25, 1976) with LUQ pain and serum amylase was found to be elevated. After a short period of NG-suction, he became asymptomatic but his amylase remained elevated. UGI series suggested a retrogastric mass. On sonography, a cyst (6 cm in diameter) was found. The patient remained asymptomatic. Once on a weekend, he had some pain and the amylase spiked high (he obviously had gotten hold of some alcohol) but was again without symptoms a day later. After 4 weeks, the cyst had not changed in size and some pressure to subject the patient to surgery was communicated from the physicians on the ward at GI-Attending Follow-up Rounds. There was no change seen on sonography at 5 weeks. At 6 weeks, the cyst had disappeared and the pancreas appeared normal when sonography was repeated as an outpatient after the 10th week.

Discussion of Case #2: Spontaneous regression of this 6 cm cyst occurred 6 weeks after the diagnosis of pseudocyst had been made. Watchful waiting in an asymptomatic patient with pseudocyst that is not enlarging is probably reasonable for even as long as 3 months (see page 18).

SPONTANEOUS REGRESSION

Spontaneous regression may occur if a pancreatic duct becomes available for drainage of the cyst, or rarely the cyst may spontaneously drain into the stomach, duodenum, common bile duct, or colon. Since most pseudocyst literature is surgical literature and since surgeons used to be more concerned about time and type of operation, relatively little information is available on spontaneous regression of pseudocysts. Before ultrasound became available, regression was difficult to follow and was reported to occur in about 7% of cases (39). Like most other statistics about pseudocyst that need to be revised after introduction of ultrasonography, spontaneous regression is now observed in about 20% of cases (34). It appears, however, that this

increased regression rate is due to recognition of smaller cysts that in the past were not diagnosed and which obviously do not need specific treatment. Thus, from a clinical point of view, really nothing was missed in this group of patients. Although it has been noted that spontaneous regression is not dependent on the size of the cyst (34), it appears to be more reasonable that the smaller the pseudocyst, the more likely it is to resolve (40). It is unlikely that a real large pseudocyst - cysts up to 22 liters have been reported (17) - or else, a very thick-walled cyst (which might calcify) can ever resolve spontaneously.

RUPTURE

Rupture of a pseudocyst is probably the complication many physicians fear most. There are about 100 cases of acute rupture reported in the literature and from these reports, it appears that perforations have occurred with equal frequency into a hollow viscus and into the peritoneal cavity.

In an effort to obtain better appreciation of the incidence of pseudocyst rupture, a small survey of the local experience was performed. Five staff gastroenterologists and five staff surgeons at Parkland Memorial Hospital, Dallas, were asked how often they had seen a patient with acute intraperitoneal rupture of a pseudocyst. None of the 5 gastroenterologists had seen such a patient (no case per 74 cumulative years in the profession), while 8 cases have been seen by the 5 surgeons (8 per 68 cumulative years in the profession, 1 case per 8.5 years). Thus, for practical purposes, rupture of a pseudocyst appears to be very rare.

In the cases reported, rupture of a pseudocyst has generally been spontaneous. This probably followed increased intracystic pressure either due to hemorrhage or continuous communication with a major pancreatic duct. Palpation by the surgeon before laparotomy has ruptured several cysts (41) and one rupture resulted incidentally during liver biopsy (42). In another case, blunt abdominal trauma sustained in an automobile accident resulted in rupture of a pre-existing cyst (43). Intraperitoneal rupture of a cyst presents as acute abdomen. Clinically the patient is usually in shock and the abdomen is rigid with generalized tenderness. If the patient had a known cyst that has disappeared, the diagnosis would be obvious. Otherwise, most commonly the clinical picture is that of a perforated viscus.

Absence of pneumoperitoneum may be an important factor in the differential diagnosis. An abdominal tap is helpful if blood or free fluid is obtained from the peritoneal cavity. This may reveal a very high amylase out of proportion to that seen in perforated peptic ulcers or other conditions in which there is an elevated amylase response. One third of patients reported with intraperitoneal perforation of a pseudocyst died as a result of massive hemorrhage or peritonitis found at autopsy (43).

Rupture into a hollow viscus may happen spontaneously. As indicated, perforation may occur into the stomach, the duodenum, common bile duct and colon. Rarely, the cyst may rupture into the esophagus (39,44) or involve multiple segments (45). While rupture into the alimentary tract may lead to cure with disappearance of the cyst associated bleeding from the site of rupture may be a severe problem.

CASE #3 (STZ 1473/74) ["After all, a typical complication of pseudocyst: hemorrhage"]

43 y/o white female. Alcoholic, heavy smoker. During admission to a sanatorium for treatment of pulmonary tuberculosis, she experienced sudden severe epigastric pain followed by vomiting. She was pale and in acute distress. Her Hb dropped to 6 g%. She was referred with the diagnosis of acute abdomen and hemorrhagic shock.

KUB: Gastric bubble with downward convexing at some distance from the diaphragm.
UGI (gastrographin): Huge round filling defect in the fundus. The NG aspirate was positive for blood, but only small amounts could be aspirated, stools were guaiac positive but melena was not present.
Celiac and mesenteric angiogram: Hepatic artery slightly pushed to the right and the gastroduodenal and gastroepiploic artery were pushed downward. SMA-root slightly dislodged to the right. No extravasation.
Liver scan: Filling defect in the area of the left lobe, or left lobe pushed to the right.
Peritoneoscopy: "Prominent stomach", normal liver.
Gastroscopy: Huge round mass in the fundus with mucosal ulcerations.

At surgery, a large pseudocyst was found that contained 1.5 liters of partly clotted blood. A cystogastrostomy was performed, the cyst was irrigated, and no further bleeding was seen. The postoperative course was uneventful.

Discussion of Case #3: Presenting symptoms were sudden epigastric pain and hemorrhagic shock. Since she was in poor general condition, surgical exploration was delayed when no repeat Hb drop occurred. Subsequent tests were not diagnostic of pseudocyst which ultimately was found at surgery and was filled with blood.

HEMORRHAGE

Hemorrhage from pseudocysts may well be the most serious complication of the condition with a mortality rate which approaches 60 per cent (39). The occurrence of bleeding has been estimated to be about 10% in patients with pancreatic pseudocyst. In one half of cases with hemorrhage, the bleeding occurs into the cyst itself. It arises from a major artery (e.g., splenic, gastroduodenal, superior or inferior pancreaticoduodenal, and gastroepiploic artery), eroded by the basic autodigestion process underway in the formation of the pseudocyst. In this situation, the pseudocyst is converted into a false aneurysm or pseudoaneurysm (42). The wall of the pseudoaneurysm is subjected to arterial pressure and may either perforate into the peritoneal cavity (46) or into an adjacent segment of the GI-tract (47), or finally into the pancreatic ductal system leading to blood discharge from the Papilla of Vater into the duodenum (48,49). As opposed to hemobilia, the name for bleeding from the pancreatic duct is "Hemosuccus pancreaticus", an expression that so far obviously has not found wide acceptance (50). The other half of cases in which bleeding occurs into the cyst shows multiple bleeding sites in the cyst wall (42,47).

Signs of bleeding into a pseudocyst are a sudden increase in the size of the mass, possibly with a pulsatile component (although this probably rather presents aortic pulsations transmitted through a cyst), a localized bruit over the mass

and a drop in hemoglobin and hematocrit. Angiography may directly show bleeding into a pseudocyst that thus becomes a pseudoaneurysm, or at endoscopy, blood may come out of the papilla. The bleeding may occur external to the pseudocyst as well, either because of rupture of the pseudocyst, or due to other causes such as concomitant peptic ulcer, acute gastric erosions, or rarely from esophageal varices secondary to portal vein compression by a cyst (7,17). Esophageal varices may also result from splenic vein thrombosis (51). Thus, in a rare case bleeding from esophageal varices may rather reflect pancreatic disease than liver disease.

ABSCESS

Pancreatic abscess is another serious complication of pseudocyst and represents an immediate danger to the patient. The pseudocyst may be converted to an abscess spontaneously, for example, due to communication with the colon and subsequent invasion of coliform bacteria, or more likely, from some much more obscure route of contamination. These abscesses generally occur from the second week to the sixth month after an attack of pancreatitis (52). Sixty percent occur within the first 6 weeks. Some pancreatic abscesses may be iatrogenic, following, for example, inadequate surgical drainage, needling of a pseudocyst or possibly ERCP.

The characteristic signs of abscess are fever, leucocytosis of greater than 18,000 per cu mm, and paralytic ileus in a patient who appears to be recovering from pancreatitis (7,53). The initial fever, leucocytosis and ileus associated with uncomplicated pancreatitis ordinarily subsides within a few days, persistence or recurrence of these signs should therefore be viewed with some alarm. Multiple blood cultures provide very useful laboratory help. Multiple organisms are present in the abscess in about 50 per cent of cases and cultures often yielf mixed bacterial flora, including staphylococci, coliforms, proteus and pseudomonas. In most instances, the varieties are characteristic of the GI-tract population since more than two-thirds are coliform organisms (54). Not all pancreatic abscesses necessarily originate as cystic complications of pancreatitis, but this sequence pertains in a majority (55). Prolonged maintenance of nasogastric suction and avoiding early operation for acute pancreatitis was found to be associated with a reduction in the frequency of pancreatic abscess (56).

PANCREATIC ASCITES AND PANCREATIC PLEURAL EFFUSION

Pancreatic ascites has only been recognized in recent years; the first patients were reported in the 1950's (57-59). Thereafter, identification became more frequent and in 1974, 85 cases were reported in the literature (60). As awareness of pancreatic ascites has increased, its recognition has also increased to the point where its relative frequency in some series approaches that of classical pseudocysts (61). The recognition of patients with chronic massive pancreatic pleural effusions is even more recent. In 1973 Anderson (62) reported five patients with this entity and was able to document an additional 11 cases from the literature.

As far as the pathogenesis is concerned, there can be no doubt now, that pancreatic ascites is secondary to a pancreatic duct disruption with the leakage of pancreatic secretions directly into the peritoneal cavity. Pancreatic duct disruptions in the face of inflammatory pancreatic disease is common. As indicated

earlier, the inflammatory reaction results in a walling off of the ductal leak which leads to pseudocyst formation. Occasionally, however, such a duct disruption occurs in the face of chronic inflammatory disease, in the absence of acute inflammation. In this setting, the leak may only be partially walled off, so that a small pseudocyst with a leak results; or the leak is not walled off at all and the ductal hole is in direct communication with the surrounding environment. If the duct disruption is anterior, an internal pancreatic fistula into the peritoneal cavity is formed and pancreatic secretions flow freely into the abdomen. Since the enzymes are not activated, an acute peritonitis does not occur and painless ascites results. Some peritoneal irritation does occur, however, because large quantities of albumin are found in the ascitic fluid. Since there is no albumin in pancreatic secretions, it has to be from a subacute or chronic peritoneal exudative reaction. In this same setting, if the duct disruption is posterior, the pancreatic secretions flow into the retroperitoneum. Once in the retroperitoneum they track along the path of least resistance, which usually is along the esophagus or aorta up into the mediastinum (62).

In the mediastinum, they can be contained and present as a mediastinal pseudocyst (14), or more commonly, they can penetrate one of both pleural surfaces and present as unilateral or bilateral pleural effusions. In such instances, the patient has an internal pancreatic fistula between his pancreatic duct and one or both pleural cavities. Although these internal fistulae usually pass through the esophageal or aortic hiatus, instances have been reported in which the fistula passes directly from a pseudocyst up though the dome of the diaphragm into the right or left pleural cavities (63). These effusions are not to be confused with the small left-sided pleural effusions seen with attacks of acute pancreatitis, which are sympathetic in nature, self-limited, and require no treatment. Chronic pancreatic pleural effusions are the result of an internal pancreatic fistula into the pleural cavity and are massive, recur rapidly if tapped, and require specific treatment.

The clinical presentation of patients with internal pancreatic fistulas often does not suggest chronic inflammatory pancreatic disease and only in 40% of cases described by Cameron (64) was there a history of an episode of acute pancreatitis and in most instances it was months or years prior to presenting with ascites or a pleural effusion. Patients with pancreatic ascites present primarily with a history of a slowly increasing abdominal girth (Table 4).

TABLE 4

INTERNAL PANCREATIC FISTULAS

Presenting Complaint	Ascites	Pleural Effusion	Both
Abdominal swelling	18		4
Abdominal pain	3	1	1
Shortness of breath		4	
Cough		2	
Weight loss	1		
Total	22	7	5

(From Cameron, 1977 [61])

Since most patients have a significant history of alcoholic intake, cirrhosis with ascites is often the admitting diagnosis. Tuberculous peritonitis, constrictive pericarditis, the Budd-Chiari Syndrome and intraperitoneal carcinomatosis are other diagnoses that can be suggested by the clinical presentation. Abdominal pain can be present but usually is not a prominent part of the clinical presentation, Weight loss, despite the massive ascites, is common (60) but rarely is the presenting symptom.

Patients with pancreatic pleural effusion present even more of a clinical paradox. Generally their symptoms are that of dyspnoea, chest pain, or a cough, and there is little to suggest adominal pathology. Pulmonary parenchymal or pleural disease is usually the admitting diagnosis. Internal pancreatic fistulas into the peritoneal and pleural cavities may be present concomitantly in the same patient (15-30% of patients with internal pancreatic fistulas) (64,65).

In the past, patients with pancreatic ascites presented a difficult clinical diagnostic challenge. The amylase in the ascitic fluid of a patient with pancreatic ascites is always markedly elevated, often up in the thousands. In addition, the ascitic fluid albumin is usually elevated to the range of 3 mg% or above (64,65). Elevated lipase levels have also been reported in the ascitic fluid (66). Cell blocks of ascitic fluid may be read as positive for malignant cells. However, pancreatic enzymes are apparently capable of inducing metaplasia of serosal cells in pancreatic ascites such that they can be mistaken for malignant cells (61). It should be emphasized that there is only one patient in the literature with documented pancreatic malignancy that also had criteria for the diangosis of pancreatic ascites (67). The great majority of patients with the clinical and laboratory picture of pancreatic ascites will have benign disease, and even in the face of a positive ascitic fluid cell cytology, management should proceed as if the underlying disease is treatable and curable.

When it became evident that internal fistulas are the source of pancreatic ascites and pleural effusions, the ductal or pseudocyst leaks have been demonstrated with increasing frequency grossly at the time of surgery or with pre-operative retrograde pancreaticography (64,65,68-70).

CASE #4 (MHD 1279/77) ["*After all, traumatic pseudocyst following automobile accident*"]

69 y/o white farmer. In January 1977, he ran into a parked car on a Dallas freeway. He was not wearing his seat belt and the steering wheel hit the middle of his abdomen. On admission, some epigastric tenderness was noted but not severe enough to suggest surgical exploration. A short bout of icterus resolved spontaneously during observation and he was discharged. He presented again one month later with epigastric tenderness and on PE, a grapefruit size mass was felt in the midepigastrium.

Chest x-ray: Two nodular lesions in right lung.
UGI: WNL except for a slight pad on the greater curve.
BE: Narrow segment in the transverse colon.
Sonography: Cystic mass anterior to pancreas.

At surgery, a 10 cm pancreatic pseudocyst filled with thick and turbid reddish-brown colored fluid was found. The cyst was irrigated. Histology showed fibroblastic cyst wall and a gastrocystostomy was performed. The nodes in the right lung were subsequently found to be vascular abnormalities on thoracotomy. The patient made an uneventful revovery.

Discussion of Case #4: Although a history of blunt abdominal trauma suggested pseudocyst, the pulmonary nodes added the possibility of metastatic abdominal tumor to the differential diagnosis.

TRAUMATIC PSEUDOCYST

Traumatic pseudocysts were first reviewed by Lloyd in 1892 (71) and in 1903 Mikulicz reported 24 cases with a 78% mortality (72). The only survivors were from a group managed surgically with drainage of the lesser sac.

A history of forcible compression of the upper abdomen and contusion of the anterior abdominal wall, especially with disruption of the recti muscles, indicates possible intra-abdominal injury. However, the diagnosis is often missed until a pseudocyst develops weeks and months later (73).

The etiology is evident. Blunt pancreatic trauma causes ductal disruption and intraparenchymal haematoma. Pancreatic secretions into the damaged area then causes pseudocyst formation in the same way, as discussed earlier in the setting of inflammatory pancreatic disease. Traumatic pseudocysts almost never resolve spontaneously (74).

RETENTION CYSTS

True retention cysts are much less frequently seen than pseudocysts. Retention cysts are gross enlargements of pre-existing pancreatic ducts. They contain a lining of epithelial cells thereby distinguished from pseudocysts whose walls consist of fibrous tissue and no cellular lining. While both cyst types arise from obstructive processes within the pancreas, retention cysts more commonly occur with chronic pancreatitis (75), possibly due to the greatly increased incidence of stone formation. Other than by stones, obstruction of normal ducts may be secondary to inflammation more proximally, or tumor. It should be remembered, that both retention cyst and pseudocyst can occur in the presence of solid pancreatic carcinoma.

Sometimes it may be difficultto differentiate retention cyst from pseudocyst since the latter may have isolated areas of endothelial lining and may communicate with pancreatic ducts (76). This indicates that in what may have begun as a retention cyst, the columnar epithelium may become disrupted and autolyzed and transition into a pseudocyst may occur. Retention cysts are usually smaller than pseudocysts and only rarely exceed 5 cm in diameter. Thus a palpable mass is only found in one-third of patients with retention cyst (75).

Patients with small retention cysts may be asymptomatic. Or else, pain suggestive of chronic pancreatitis may be present. Other symptoms such as vomiting are less frequent than in pseudocyst and the history is usually longer (rarely less than 6 months). Positive roentgenograms indicative of pancreatic cyst are only found in 50% of cases as opposed to more than 90% in pseudocyst. Ultrasound and endoscopic retrograde pancreaticogram appear to be the best diagnostic modalities for retention cyst. Bleeding from a retention cyst is extremely rare (77).

NEOPLASTIC CYSTS

Cystadenoma of the pancreas is a benign tumor, but malignant transformation has been reported (78,79). Cystadenocarcinoma is a slow growing tumor with a low degree of malignancy. Expansion beyond the borders of the pancreas is slow and early metastases are rare. The opened cyst has a thick, irregular wall and is often multilocular. The relative proportion of solid tissue and cyst content is variable. Carcinoma may be present in only a small portion of the cyst, the remainder being a cystadenoma, so that biopsy of a limited area may be misleading. These tumors are more frequent in females.

CONGENITAL PANCREATIC CYSTS

These cysts may be single, multiple, unilocular or multilocular and can be associated with polycystic disease of the kidney, liver or spleen. The cysts are usually in the body or tail and may contain pancreatic tissue and a cloudy yellow fluid (80). These cysts are rarely symptomatic.

MEDICAL MANAGEMENT

The possibility to monitor cyst development by sonography, the observation that about 20% of cysts resolve spontaneously, and the time (about 6 weeks) needed for maturation of a cyst wall strongly suggest initial medical treatment. Exceptions of course are patients that already present with severe complications of pseudocyst that might need immediate surgery such as rupture or continuous severe bleeding.

Once the diagnosis of pseudocyst is made close serial observation for several weeks is necessary. The purpose of this careful observation is to determine what is happening to the patient and the pseudocyst, to recognize complications as they arise and to be constantly apprised of the need for surgical intervention. Serial observation should consist of frequent clinical evaluations, since over 50% of pseudocysts are palpable, serial serum amylase determinations and most importantly, serial sonography, since this procedure is accurate, relatively inexpensive, it is noninvasive and therefore can be repeated even daily if desired (81). Retrograde pancreatography is too time consuming, expensive and dangerous to be used as a means of following a pseudocyst serially (31). Similarly, computerized axial tomography is too expensive and the radiation hazard is probably too great for serial use (38).

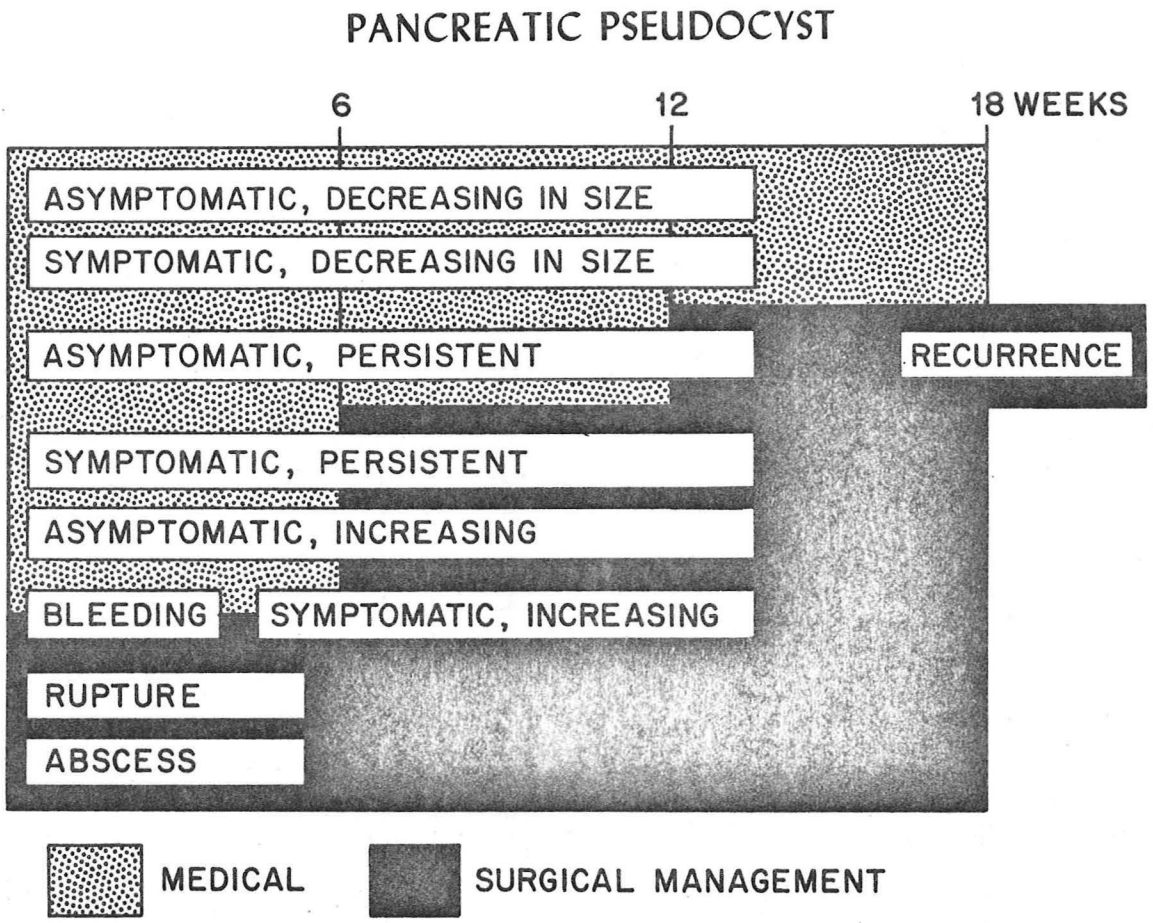

Figure 3.: Indications for medical and/or surgical treatment of pancreatic pseudocysts in relation to time course.

Conservative management should, if possible, be carried out for 6 weeks. The mortality of intervention earlier than 6 weeks approaches 60 per cent, when this intervention is for the purpose of definite surgical management of the pseudocyst, that is, internal drainage. This unsatisfactory risk is related, at least in part to the need to allow the wall of the cyst to mature before it can be handled surgically (7). When carried out after 6 weeks, the mortality was 9%. Cerilli and Faris (82) published these figures some 10 years ago and surgeons have claimed that they may do better today. Moreover, the total number of cases was only 16 in this study. Of course, in some patients it will be difficult or even impossible to know from when to count these 6 weeks. In others, however, cyst formation may be observed in the hospital following an acute episode of pancreatitis and timing is then no problem.

Appropriate medical therapy must include treatment of the underlying disease leading to pseudocyst formation. For instance, treatment of pancreatitis with nasogastric suction is performed according to symptoms and serum amylase values like in cases not complicated by pseudocyst. Continuation of nasogastric suction to maintain the pancreas "at rest" has not been shown to promote resolution of pseudocysts, although in some patients this might appear a reasonable approach. In others, like asymptomatic patients with pseudocyst, nasogastric suction might be quite unreasonable and unpractical for prolonged time. The administration of antibiotics in the course of acute pancreatitis has now been shown in four prospective studies to have no effect on the course of acute pancreatitis (52,83-85). Prophylactic use should be abandoned.

Hypoalbuminemia and loss in body weight resulting from accelerated catabolism, sequestration of protein and caloric deficit are among the serious metabolic and hemodynamic consequences of chronic pancreatitis and pseudocyst. Parenteral hyperalimentation is necessary in a number of these patients, particularly before surgery. In Figure 3, an attempt is made to show the indications for medical and/or surgical treatment in the course of pseudocyst. Only medical treatment is indicated for asymptomatic and symptomatic pseudocyst which is decreasing in size and ultimately resolving. For an asymptomatic persistent cyst, I suggest surgery only after prolonged observation, such as after 12 weeks. The patient may even be followed as an outpatient. For symptomatic persistent pseudocyst as well as asymptomatic pseudocyst increasing in size, surgery is suggested around the 6th week after cyst formation. Symptomatic pseudocysts increasing in size may need drainage before the 6th week.

Bleeding from a pseudocyst often needs surgery although a few nonsurgical approaches are noteworthy. Bleeding has been stopped by controlled hypotension (86,87), pitressin infusion and such extreme techniques as selective emolization or vascular occlusion by cauterization using a wire directed via an angiography catheter (88). Rupture into the peritoneal cavity and abscess formation always urge the surgeon to early intervention. Without surgical drainage of pancreatic abscess, death is virtually inevitable (89,90); even with the appropriate use of antibiotics and adequate drainage, the mortality averages 50 per cent (7,54). Furthermore, the abscess may recur or be loculated such that a second operation is required in some 30 per cent of patients, simply to achieve adequate drainage to the outside (91).

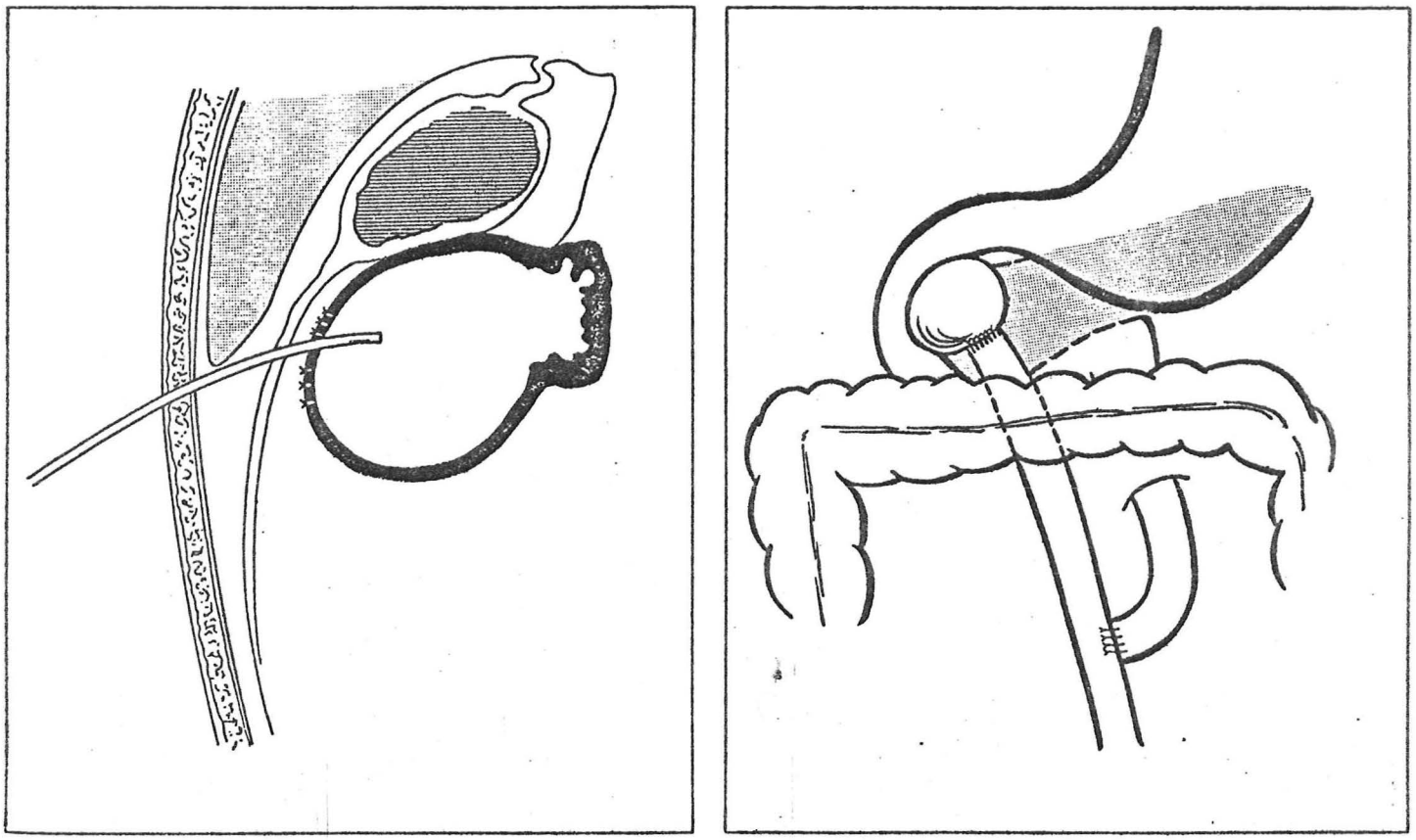

Figure 4.: External drainage (left) and internal drainage (right) of pancreatic pseudocyst (From Fux, H.D. et al., 1977)

Recurrent cyst formation is generally considered an indication for surgery. Jaundice occurs secondary to common bile duct obstruction in 10 per cent of cases of pseudocyst. Obstructive jaundice and obstruction of the upper gastrointestinal tract by pseudocyst may require surgical intervention if persistent or complicated by cholangitis or sepsis, decompression by pseudocyst drainage is indicated (50). Before coming to the surgical drainage procedures, it should be stated that endoscopists have performed transgastric puncture of a pseudocyst and created a communication to facilitate drainage. This might be possible and appropriate in a very, very exceptional case (92).

SURGICAL MANAGEMENT

External Drainage (Figure 4)

Most surgeons now agree that external drainage has considerable disadvantages and should be reserved for cases that need surgery before maturation of the cyst wall, for severely ill patients with a high operative risk (in whom a relatively short procedure should be done). Disadvantages of external drainage are fluid and electrolyte loss, skin irritation, persistent fistula and a remarkably high rate of recurrence (as high as 25%) (74,93).

Internal Drainage (Figure 4)

This operation is the procedure of choice. It is accomplished by anastomosis of the cyst to the stomach, duodenum or proximal jejunum. Anastomosis to a Roux-en-Y segment of jejunum is preferred by many surgeons over gastrocystostomy. The latter may be more prone to bleeding and to become infected. Of course position of the cyst plays a major role and a cyst densely adherent to the posterior wall of the stomach suggests cystogastrostomy. Although the Roux-en-Y anastomosis is theoretically superior to cystogastrostomy, it is a more complicated operation. Anastomosis of a pseudocyst to the duodenum is more hazardous than either cystogastrostomy or Roux-en-Y cystojejunostomy (73,74,94,95).

Excision or Resection

Occasionally, it is possible to enucleate or excise a superficial cyst. This form of treatment should not be elected, however, when the cyst is deep within the substance of the pancreas or when it appears to involve the main pancreatic ducts. Cysts confined to the tail of the pancreas with marked destruction of the pancreatic parechnyma lend themselves to distal pancreatectomy. If there is no obstruction to the pancreatic duct at the papilla of Vater, the distal divided end of the pancreas is closed.

Since the first excision of a pancreatic pseudocyst was performed on a woman from Texas (2), I thought you might enjoy reading the original communication:

NEW YORK PATHOLOGICAL SOCIETY

Stated Meeting, December 14, 1881

Removal of a cyst of the pancreas weighing twenty and one-half pounds:

Dr. T.E. Bozeman presented a specimen accompanied by the following history: it was interesting with reference to three particulars: first, as having been removed from the pancreas of a living woman; second, as having been mistaken for an ovarian cyst; and third, as being the first operation of the kind upon record. The patient was the wife of a prominent physician of Texas, forty-one years of age, tall and robust, weighing nearly two hundred pounds, and perfectly healthy up to seven years ago, except for occasional attacks of dyspepsia. Seven years ago she had, for the first time, pain in the right iliac region, extending down the right thigh and occasionally attended with numbness. Five years ago the abdomen began to enlarge, slowly at first, but gradually increased in size upon the left side, with a corresponding flatness upon the right side. The point at which the enlargement was first noticed was higher than would naturally be expected for an ovarian cyst. At that time no special importance was attached to the enlargment of the abdomen, either by herself or husband, who frequently examined the tumor. It progressed in the ordinary way up to six or seven months ago, when it suddenly began to grow rapidly, and finally the entire abdomen was distended symmetrically. At the same time, the patient began to lose flesh. The case was diagnosticated as one of ovarian cyst by Professor Richardson, of New Orleans, who advised the patient to consult Dr. Bozeman. On November 19, 1881, the patient having entered the Woman's Hospital, Dr. Bozeman examined her and diagnosticated ovarian cyst. She was also examined by his colleagues, Dr. Thomas and Emmet, both of whom confirmed his diagnosis. An operation was decided upon and it was performed on the second day of December, under Listerism. Nothing unusual presented itself in the early stage of the operation. When the tumor was reached through an incision below the umbilicus, its appearance was nearly that presented by an ordinary unilocular ovarian cyst, except perhaps it had a little deeper pearlish color. It was tapped and two and one-half gallons of fluid were removed. After the greater part of the fluid was drawn off, about two-thirds of the cyst was drawn through the abdominal opening, and then for the first time, Dr. Bozeman suspected that it was not ovarian. He then passed his hand into the peritoneal cavity and found the uterus and both ovaries, and also determined that the cyst had an origin somewhere in the upper part of the abdomen. The abdominal incision was extended upward two inches above the umbilicus. The stomach was then found crowded against the diaphragm, and the bowels were deep in the abdominal cavity below the cyst. The cyst had an extensive attachment, apparently to the transverse mesocolon. After some manipulation he finally reached the pancreas, where he discovered a large vein, subsequently determined to be the splenic, which was very tortuous, and offered considerable obstruction to the operation, owing to its close relationship to the pedicle. Finally he traced the cyst down until he reached the tail of the pancreas, which was turned up on the side of the cyst, and firmly adherent to it to the extent of two inches. He then proceeded to separate the extremity of the pancreas from the cyst by dissection, and when completely separated the pancreas spread out and presented its natural appearance. The attachment of the cyst was at the junction of the outer with the inner tow-thirds of the organ, and it had a pedicle three-fourths of an inch in length and about three-fourths of an inch in diameter. The veins of the pedicle were very large. Having fairly reached the pedicle, he transfixed it with a needle,

ligated it in the usual way, and cut it off. The result was that he cut out the bottom of the cyst, as shown in the specimen. The portion of the cyst, however, which remained attached to the pedicle was subsequently completely removed by dissection. The artery which supplied the growth was doubtless a branch of the splenic, and it had attained a very large size - as large as the brachial. The loss of blood was small, and not a single bleeding vessel required a ligature. The fluid which the cyst contained was of a light brownish color, its specific gravity was 1020, and it had an acid reaction, in that respect differing from the fluid removed from the ordinary ovarian cyst, which is alkaline. The girth of the patient before the operation was forty-one inches, and both oblique measurements, from the anterior superior spinous processes of the ilia to the umbilicus, were the same - nine inches. The tumor, with the fluid weighed twenty and one-half pounds.

The specimen was also interesting in another respect, namely: with reference to the point of attachement, which was almost precisely in the position occupied by the bullet in the late case of our deceased President. The patient underwent special preparation for the operation. She took salicin, fifteen grains three times a day for two weeks. On the morning of the day on which the operation was performed she received fifteen grains of quinine with one of opium, and when she went upon the table she was thoroughly cinchonized. The patient rallied from the anaesthetic and from the operation without any shock whatever. After the operation she took by the rectum, at intervals of six hours, ten grains of quinine with two ounces of beef-juice, half a drachm of liquor opii comp., and two drachms of brandy. On the third day the temperature reached its highest point, 101.5°F., but the pulse never rose above 98. Subsequently, the pulse fell to 80, and the quantity of quinine was gradually lessened, but on the eighth day after stopping the quinine the temperature rose to 102.8°F. The quinine was again resumed, ten grains every six hours, and the temperature in the course of thirty-six hours fell to 99.5°F., and subsequently the patient had progressed in the most satisfactory manner, and there was every prospect of a complete recovery.

RESULTS OF SURGERY

Table 5 summarizes the results of 1442 patients operated on for pseudocyst (73,75,95,96). There are two striking observations, namely the high mortality in the group with excision and the high recurrence rate with external drainage.

TABLE 5

RESULTS OF SURGERY FOR PANCREATIC PSEUDOCYST

	CASES	RECURRENCE (%)	MORTALITY (%)
Excision, Resection	168	2	12
External Drainage	594	22	9
Internal Drainage	680	5	6
Total	1442		

RETENTION CYSTS

Retention cysts can be treated either by drainage or if located in the distal pancreas by resection.

NEOPLASTIC CYSTS

These cysts always need to be resected. If there is doubt about the differential diagnosis, and particularly if there is a multiloculated cyst, surgery should be performed. On the other hand, in each pseudocyst a wall biopsy should be done not to overlook a neoplastic cyst which rather needs resection than drainage.

MANAGEMENT OF PANCREATIC ASCITES AND PANCREATIC PLEURAL EFFUSION

Once the diagnosis has been confirmed by ascitic or pleural fluid amylase and protein elevations, the treatment probably should be non-operative first. In an attempt to decrease pancreatic secretion to a minimum and thus encourage the sealing of the pancreatic duct or pseudocyst leak, the following regimen is suggested by Cameron (61). (1) NPO and nasogastric suction; (2) Diamox and atropin are administered to further decrease pancreatic secretion; (3) Paracenteses are performed as needed to keep the peritoneal cavity free of fluid, and thus encourage the approximation of peritoneal surfaces and the sealing of the leak; (4) Intravenous hyperalimentation is often indicated, since most of these patients are in poor nutritional status.

Ascites will clear in about 40% of cases on this described medical regimen. If after 3 or 4 weeks the ascites continues to recur, most patients should be operated upon. If a patient were a prohibitive operative risk, low dose irradiation of the pancreas might be considered, since there have been five cases reported with resolution of ascites in each instance following radiation (57,97,98,99,100).

The success of operative management of pancreatic ascites depends upon adequate knowledge of the pathologic anatomy. ERP should be attempted pre-operatively in all patients being considered for surgery. Multiple case reports document the ability of ERP to define the pathology and at times the site of the ductal leak. If ERP is unsuccessful, operative pancreatography should be performed, at the time of surgery. The type of surgical procedure employed will depend upon the information gained by pancreatography. If a direct duct leak is demonstrated with no pseudocyst, a direct anastomosis between the leak and a Roux-en-Y jejunal loop will be preferable. If the leak is distal in the tail, a distal pancreatectomy could also be considered. If a small cyst is present in the distal portion of the gland, as is frequently the case with pancreatic ascites, a distal resection removing the tail of the gland and cyst is indicated. If the cyst is large or in a region of the pancreas not amenable to resection, drainage of the cyst into a Roux-en-Y loop or the stomach is preferable.

In patients with internal pancreatic fistulas into the chest presenting with chronic massive pleural effusions conservative treatment is very successful. In

addition to parenteral hyperalimentation, nasogastric suction and diamox and atropin administration, patients have been treated with multiple paracenteses or a chest tube. Since the patients have a longer fistulous tract than do those with pancreatic ascites, the fistula may be more apt to closure without surgical intervention. If, however, surgery is employed then operative management of pancreatic pleural effusion is identical to that of pancreatic ascites. It is absolutely unnecessary to enter the chest, and a total abdominal approach should be planned (61).

SUMMARY

Pancreatic pseudocyst can be a consequence of inflammation, trauma or proximal duct obstruction by tumor. It is a serious condition with such life threatening complications as bleeding, abscess formation and rupture. At the present time, ultrasonography is the optimal method to measure cyst size and to closely follow each patient with pseudocyst. Twenty per cent of pseudocysts show spontaneous regression. If the cyst persists or even increases in size, then surgery is indicated. About six weeks are necessary for maturation of the cyst wall in order to make internal drainage either by cystogastrostomy or preferably by cystojejunostomy a safe procedure. External drainage is only advocated when surgery is needed early in the course before cyst wall maturation has occurred. The recurrence rate of pseudocysts after external drainage is high. Pancreatic ascites and pancreatic pleural effusion respond to medical treatment in about 40%, if surgery is required the leaking duct or pseudocyst need to be drained.

CASE #5 [*"After all, a wrong operation in a misdiagnosed case"*]

69 y/o white man with a history of moderate alcohol intake and one episode of pancreatitis in the past. The patient was admitted for epigastric pain, vomiting, fever, and weight loss. On physical examination, a mass was palpated in the right epigastrium which was shown to be cystic on sonography. On UGI, the C-loop was widened and anteriorly dislodged. Laboratory results showed leucocytosis and slightly elevated amylase. The patient's condition did not improve on medical management. A pancreatic pseudocyst, possibly infected, was suspected and the patient underwent laparotomy. The cyst was found and appeared to be arising from the pancreas. The wall was dense enough to be suitable for internal drainage. A cystojejunostomy was performed. The patients condition improved after surgery, he became afebrile and slowly started to gain weight. A barium study before discharge showed normal small bowel but barium also filled the complete calyceal system of the right kidney. Obviously a cyst-like distended renal pelvis in a case of pyonephrosis had been drained into the small bowel. The patient refused further surgery.

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