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1

Acute Biliary Pancreatitis

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1. Introduction

Acute pancreatitis is an inflammatory disease of the pancreas. The etiology and pathogenesis of acute pancreatitis have been intensively investigated for centuries worldwide. It can be initiated by several factors, including gallstones, alcohol, trauma, infections and hereditary factors. About 75% of pancreatitis is caused by gallstones or alcohol. In this chapter we discuss the causes, diagnosis, imaging findings, therapy, and complications of acute biliary pancreatitis.

2. Anatomy and physiology

The pancreas is perhaps the most unforgiving organ in the human body, leading most surgeons to avoid even palpating it unless necessary. Situated deep in the center of the abdomen, the pancreas is surrounded by numerous important structures and major blood vessels. Surgeons that choose to undertake surgery on the pancreas require a thorough knowledge of its anatomy. However, knowledge of the relationships of the pancreas and surrounding structures is also critically important for all surgeons to ensure that pancreatic injury is avoided during surgery on other structures.

The pancreas is a retroperitoneal organ that lies in an oblique position, sloping upward from the C-loop of the duodenum to the splenic hilum. In an adult, the pancreas weighs 75 to 100 g and is about 15 to 20 cm long. The fact that the pancreas is situated so deeply in the abdomen and is sealed in the retroperitoneum explains the poorly localized and sometimes ill-defined nature with which pancreatic pathology presents.

Surgeons typically describe the location of pathology within the pancreas in relation to four regions: the head, neck, body, and tail. The head of the pancreas is nestled in the C-loop of the duodenum and is posterior to the transverse mesocolon.

Most of the pancreas drains through the duct of Wirsung, or main pancreatic duct, into the common channel formed from the bile duct and pancreatic duct. (Figure 1) The length of the common channel is variable. In about one third of patients, the bile duct and pancreatic duct remain distinct to the end of the papilla, the two ducts merge at the end of the papilla in another one third, and in the remaining one third, a true common channel is present for a distance of several millimeters.

The main pancreatic duct is usually only 2 to 3 mm in diameter and runs midway between the superior and inferior borders of the pancreas, usually closer to the posterior than to the

anterior surface. Pressure inside the pancreatic duct is about twice that in the common bile duct, which is thought to prevent reflux of bile into the pancreatic duct. The main pancreatic duct joins with the common bile duct and empties at the ampulla of Vater or major papilla, which is located on the medial aspect of the second portion of the duodenum. The muscle fibers around the ampulla form the sphincter of Oddi, which controls the flow of pancreatic and biliary secretions into the duodenum. Contraction and relaxation of the sphincter is regulated by complex neural and hormonal factors.
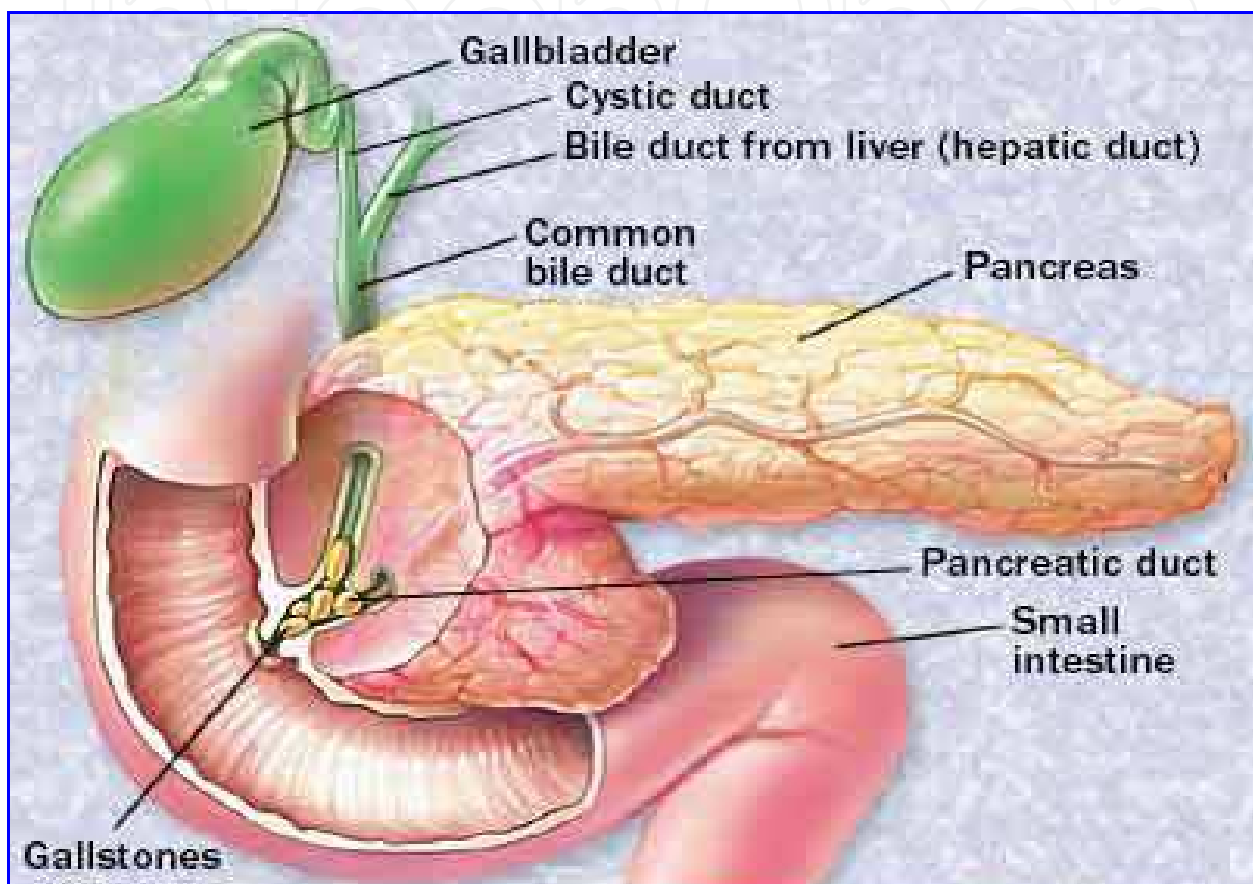

Fig. 1. Pancreas and biliary system anatomy

The exocrine pancreas accounts for about 85% of the pancreatic mass; 10% of the gland is accounted for by extracellular matrix, and 4% by blood vessels and the major ducts, whereas only 2% of the gland is comprised of endocrine tissue.

The pancreas secretes approximately 500 to 800 mL per day of colorless, odorless, alkaline, isosmotic pancreatic juice. Pancreatic juice is a combination of acinar cell and duct cell secretions. The acinar cells secrete amylase, proteases, and lipases, enzymes responsible for the digestion of all three food types: carbohydrate, protein, and fat. The acinar cells are pyramid-shaped, with their apices facing the lumen of the acinus. Near the apex of each cell are numerous enzyme-containing zymogen granules that fuse with the apical cell membrane.

Pancreatic amylase is secreted in its active form and completes the digestive process already begun by salivary amylase. Amylase is the only pancreatic enzyme secreted in its active form, and it hydrolyzes starch and glycogen to glucose, maltose, maltotriose, and dextrins.

These simple sugars are transported across the brush border of the intestinal epithelial cells by active transport mechanisms. Gastric hydrolysis of protein yields peptides that enter the intestine and stimulate intestinal endocrine cells to release cholecystokinin (CCK)-releasing peptide, CCK, and secretin, which then stimulate the pancreas to secrete enzymes and bicarbonate into the intestine.

The proteolytic enzymes are secreted as proenzymes that require activation. Trypsinogen is converted to its active form, trypsin, by another enzyme, enterokinase, which is produced by the duodenal mucosal cells. Trypsin, in turn, activates the other proteolytic enzymes. Trypsinogen activation within the pancreas is prevented by the presence of inhibitors that are also secreted by the acinar cells. Chymotrypsinogen is activated to form chymotrypsin. Elastase, carboxypeptidase A and B, and phospholipase are also activated by trypsin. Trypsin, chymotrypsin, and elastase cleave bonds between amino acids within a target peptide chain, and carboxypeptidase A and B cleave amino acids at the end of peptide chains. Individual amino acids and small dipeptides are then actively transported into the intestinal epithelial cells. Pancreatic lipase hydrolyzes triglycerides to 2-monoglyceride and fatty acid. Pancreatic lipase is secreted in an active form. Colipase is also secreted by the pancreas and binds to lipase, changing its molecular configuration and increasing its activity. Phospholipase A2 is secreted by the pancreas as a proenzyme that becomes activated by trypsin. Phospholipase A2 hydrolyzes phospholipids and, as with all lipases, requires bile salts for its action. Carboxylic ester hydrolase and cholesterol esterase hydrolyze neutral lipid substrates like esters of cholesterol, fat-soluble vitamins, and triglycerides. The hydrolyzed fat is then packaged into micelles for transport into the intestinal epithelial cells, where the fatty acids are reassembled and packaged inside chylomicrons for transport through the lymphatic system into the bloodstream.

The centroacinar and intercalated duct cells secrete the water and electrolytes present in the pancreatic juice. About 40 acinar cells are arranged into a spherical unit called an *acinus*. Centroacinar cells are located near the center of the acinus and are responsible for fluid and electrolyte secretion. These cells contain the enzyme carbonic anhydrase, which is needed for bicarbonate secretion.

The acinar cells release pancreatic enzymes from their zymogen granules into the lumen of the acinus, and these proteins combine with the water and bicarbonate secretions of the centroacinar cells. The pancreatic juice then travels into small intercalated ducts. Several small intercalated ducts join to form an interlobular duct. Cells in the interlobular ducts continue to contribute fluid and electrolytes to adjust the final concentrations of the pancreatic fluid. Interlobular ducts then join to form about 20 secondary ducts that empty into the main pancreatic duct. Destruction of the branching ductal tree from recurrent inflammation, scarring, and deposition of stones eventually contributes to destruction of the exocrine pancreas and exocrine pancreatic insufficiency.

There are nearly 1 million islets of Langerhans in the normal adult pancreas. Alpha cells that secrete glucagon, Beta cells that secrete insulin, Delta cells that secrete somatostatin, Epsilon cells that secrete ghrelin, and PP cells that secrete PP.[1]

3. Incidence

Acute pancreatitis is a relatively common disease that affects about 300,000 patients per annum in America with a mortality of about 7%. Acute pancreatitis is mild and resolves

itself without serious complications in 80% of patients, but it has complications and a substantial mortality in up to 20% of patients despite the agressive intervention[1]. The incidence of alcoholic pancreatitis is higher in male, and the risk of developing acute pancreatitis in patients with gallstones is greater in male. However, more women develop this disorder since gallstones occur with increased frequency in women[2].

4. Etiology and pathophysiology

The pathogenesis of acute pancreatitis has not been fully understood. The general belief today is that pancreatitis begins with the activation of digestive enzymes inside acinar cells, which cause acinar cell injury. The Factors in Acute Pancreatitis can be classified as:

Metabolic

Alcoholism
Hyperlipoproteinemia
Hypercalcemia
Drugs (e.g., thiazide diuretics)
Genetic
Scorpion poison

Mechanical

Trauma
Gallstones
Iatrogenic injury
Perioperative injury
Endoscopic procedures with dye injection
Pancreas divisium
Pancreatic duct obstruction(tumors, ascariasis, ampullar stenosis)
Pancreatic duct bleeding
Duodenal obstruction

Vascular

Shock
Atheroembolism
Vasculitis (Polyarteritis nodosa)

Infectious

Viral

Mumps
Coxsackievirus
EBV
HIV (Human Immunodeficiency Virus)

Bacterial

Mycoplasma pneumonia
Campylobacter
Legionella

Parasites

Ascaris
Clonorchis sinensis

Of note, 10% to 20% of patients with acute pancreatitis have no known associated processes. Although this condition is currently termed idiopathic.

The underlying reason of gallstone disease and other conditions causing acute pancreatitis is ductal hypertension resulting from ongoing exocrine secretion into an obstructed pancreatic duct. Elevated intraductal pressure, due to ongoing exocrine secretion, causes rupture of the smaller ductules and leakage of pancreatic juice into the parenchyma. Pancreatic tissue favors activation of proteases when transductal extravasation of fluid occurs.

In the normal pancreas, the inactive digestive zymogens and the lysosomal hydrolases are found separately in discrete organelles. However, in response to ductal obstruction, hypersecretion, or a cellular insult, these two classes of substances become improperly colocalized in a vacuolar structure within the pancreatic acinar cell. Coalescence of zymogen granules with lysosome vacuoles resulting in intrapancreatic activation of proteolytic enzymes. Small amounts of trypsin can be countered by endogenous pancreatic trypsin inhibitor. However, large amounts of trypsin release can overwhelm the serological defense mechanism (α-1-antitrypsin and α-2-macroglobulin) and activate other enzymes resulting in destruction of acinar cells, local and systemic complications commonly seen in the course of the disease. Activation of the enzyme phospolipase A2 has important consequences like destruction of pulmonary surfactant that can result in ARDS and liberation of prostaglandins and leucotriens that may be important in the pathogenesis of the systemic inflammatory response which can lead to multi organ failure. More than that, inflammatory mediators may be used as predictors of disease severity in the near future. Also, trypsin activates and complements kinin, kallikrein, possibly playing a part in disseminated intravascular coagulation, shock, renal failure and vascular instability. [3], [4].

5. Diagnosis

A detailed history and careful physical examination are the first step towards making the diagnosis. The diagnosis of gallstone pancreatitis should be suspected if the patient has a prior history of biliary colic. [5], [6] Acute pancreatitis typically presents with severe upper abdominal pain which may radiate through to the back and be associated with nausea and vomiting.

On physical examination, the patient may show tachycardia, tachypnea, hypotension, and hyperthermia. The temperature is usually only mildly elevated in uncomplicated pancreatitis. Voluntary and involuntary guarding can be seen over the epigastric region. The bowel sounds are decreased or absent. There are usually no palpable masses. The abdomen may be distended with intraperitoneal fluid. There may be pleural effusion, particularly on the left side. With increasing severity of disease, the intravascular fluid loss may become life-threatening as a result of sequestration of edematous fluid in the retroperitoneum.

6. Biochemical markers

Due to the destruction of acinar cells, the levels of the enzymes that they contain (e.g., amylase, lipase, trypsinogen, and elastase) are found elevated in the serum of most pancreatitis patients. Serum amylase concentration increases almost immediately with the onset of disease and peaks within several hours. It remains elevated for 3 to 5 days before returning to normal. There is no significant correlation between the magnitude of serum amylase elevation and severity of pancreatitis. [7,8]

Lipase is more specific for pancreatitis. Serum lipase has a longer half life than amylase and therefore tends to remain elevated for longer.

Urinary clearance of pancreatic enzymes from the circulation increases during pancreatitis; therefore, urinary levels may be more sensitive than serum levels.

Several tests can help differentiate biliary pancreatitis from other causes of pancreatitis. Aspartate aminotransferase (AST), alanine aminotransferase (ALT), gamma-glutamyl Transpeptidase (GGT), alkaline phosphatase and serum bilirubin are the so-called liver function tests; they should be reviewed before making a confident diagnosis.

Several recent research studies have suggested additional markers that may have prognostic value, including C-reactive protein (CRP), alpha$_2$-macroglobulin, polymorphonuclear neutrophil–elastase, alpha1-antitrypsin, and phospholipase A2. [9],[10] Although CRP measurement is commonly available, many of the others are not. Therefore, at this time, CRP seems to be the marker of choice in clinical settings. The measurement of IL-6 has recently been shown to distinguish patients with mild or severe forms of the disease. Another prognostic marker under evaluation is urinary–trypsinogen activation peptide (TAP). It has a good correlation between the severity of pancreatitis and concentrations of TAP in urine.

Currently, these new markers have limited clinical availability, but there is significant interest in better understanding markers of immune response and pancreatic injury because these could be valuable tools for reliably predicting the severity of acute pancreatitis and supplementing imaging modalities. [10],[11],[12]

7. Radiologic imaging

Ultrasound: Abdominal ultrasound (US) examination is the best way to confirm the presence of gallstones in suspected biliary pancreatitis. It also can detect extrapancreatic ductal dilations and reveal pancreatic edema, swelling, and peripancreatic fluid collections. But abdominal ultrasonography seldom visualizes the pancreas in patients with acute pancreatitis due to air in the distended loops of the small bowel. [13] (**Figure 2**)

Computed Tomography Scan (CT): A CT allows identification of pancreatic edema, fluid or cysts, and the severity of pancreatitis to be graded, detects complications including development of pseudocysts, abscess, necrosis, hemorrhage, and vascular occlusion. The finding of gallstones and dilatation of the extra-hepatic biliary tree on cross-sectional abdominal imaging further support to the diagnosis of gallstone pancreatitis. [14]

Currently the best method to stage the acute pancreatitis is CT. Specific CT findings can be categorized into pancreatic and peripancreatic changes. Pancreatic changes include diffuse or focal parenchymal enlargement, edema, or necrosis with liquefaction. Peripancreatic involvement includes blurring or thickening of the surrounding tissue planes. An approximate correlation exists between the degree of CT abnormalities and the clinical course and severity of acute pancreatitis.

An early discrimination between mild edematous and severe necrotizing forms of the disease is of the utmost importance to provide optimal care to the patient. CT has become the gold standard for detecting and assessing the severity of pancreatitis. Although clinically mild pancreatitis is usually associated with interstitial edema, severe pancreatitis is associated with necrosis. The presence of air bubbles on a CT scan is an indication of infected necrosis or pancreatic abscess. [15]
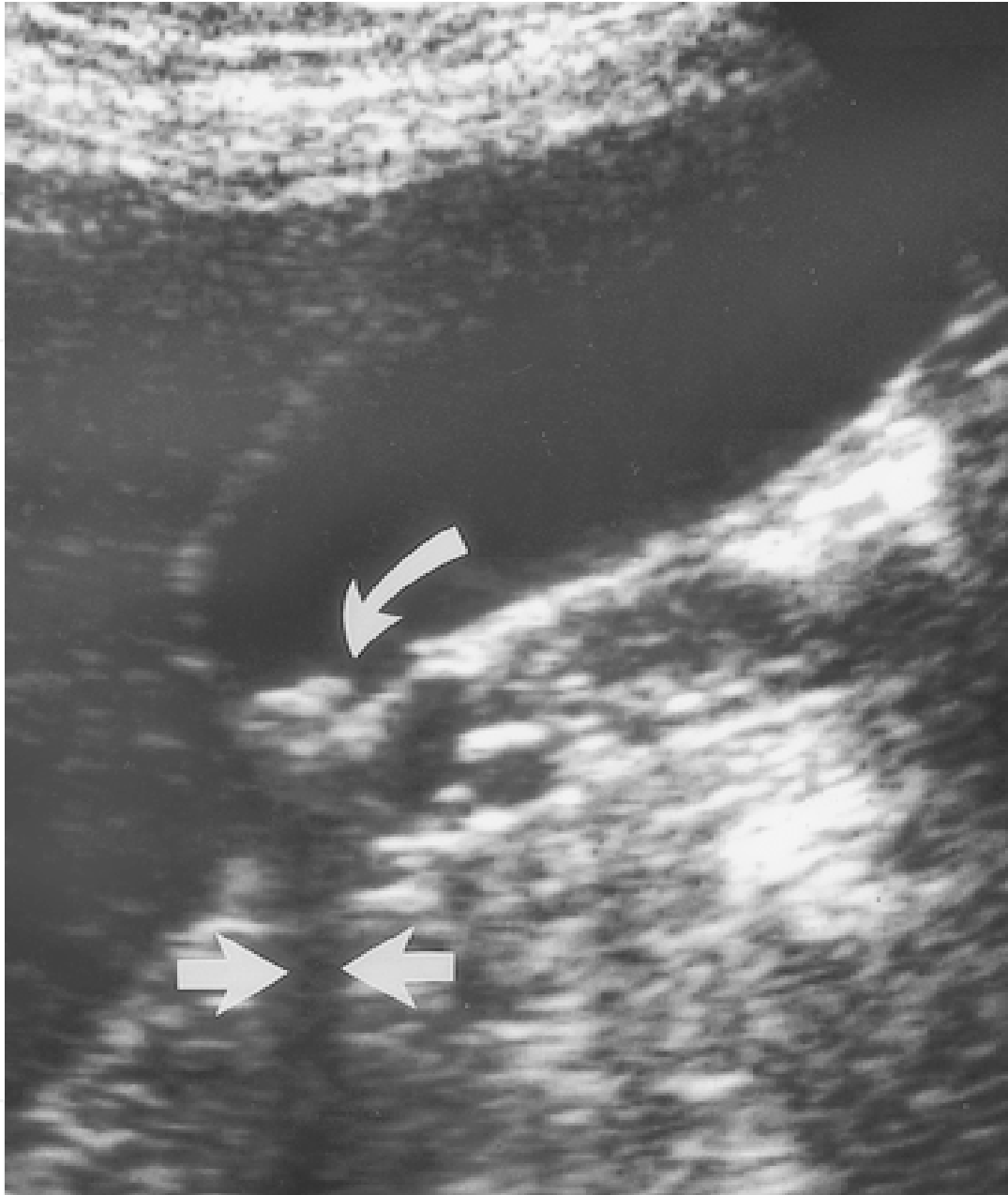
Fig. 2. Ultrasound image of the gallbladder demonstrates multiple dependent gallstones (curved arrow) with acustic shadowing (straight arrows). The patient had elevated pancreatic enzyme levels and underwent cholecystectomy because of gallstone pancreatitis.

Magnetic Resonance Cholangiopancreatography (MRCP): MRCP has been found to be as accurate as contrast-enhanced CT in predicting the severity of pancreatitis and identifying pancreatic necrosis but is less sensitive for detection of small stones.

Endoscopic Ultrasonography: It is useful in obese patients and patients with ileus, and can help determine which patients with acute pancreatitis would benefit most from therapeutic ERCP. [16], [17]

Fig. 3. Acute biliary pancreatitis with a thickened pancreas and an effusion around the pancreatic tail and around the spleen - CT scan

Fig. 4. Sigmoid configuration of the main pancreatic duct with distal dilation of both main and dorsal ducts, suggesting the presence of an obstructive condition at the level of both major and minor papillae.

ADMISSION	INITIAL 48 HOURS
Gallstone Pancreatitis	
Age > 70 yr	Hct fall >10
WBC >18,000/mm^3	BUN elevation >2 mg/100 mL
Glucose > 220 mg/100 mL	Ca^{2+} <8 mg/100 mL
LDH >400 IU/L	Base deficit >5 mEq/L
AST >250U/100 mL	Fluid sequestration >4 L
Nongallstone Pancreatitis	
Age >55 yr	Hct fall >10
WBC >16,000/mm^3	BUN elevation >5 mg/100 mL
Glucose >200 mg/100 mL	Ca^{2+} <8 mg/100 mL
LDH >350 IU/L	Pa$_{O_2}$ <55 mm Hg
AST >250U/100 mL	Base deficit >4 mEq/L
	Fluid sequestration >6 L

Adapted from Ranson JHC, Rifkind KM, Roses DF, et al: Prognostic signs and the role of operative management in acute pancreatitis. Surg Gynecol Obstet 139:69-81, 1974; and Ranson JHC: Etiological and prognostic factors in human acute pancreatitis: A review. Am J Gastroenterol 77:633, 1982. (AST, aspartate transaminase; BUN, blood urea nitrogen; Ca^{2+}, calcium; Hct, hematocrit; LDH, lactic dehydrogenase; Pa$_{O_2}$, arterial oxygen; WBC, white blood cell count.)

8. Scoring systems in acute pancreatitis

A variety of scoring systems have been proposed for accurate assessment of the severity of acute pancreatitis. These include the clinical scoring scales as Ranson criteria, Glasgow scales, simplified acute physiology (SAP), score and acute physiology and chronic health evaluation II (APACHE II) score. The CT severity index (CTSI) derived by Balthazar grading of pancreatitis and the extent of pancreatic necrosis is now widely used in describing CT findings of acute pancreatitis and serves as the radiological scoring system. [18]

Ranson identified a series of prognostic signs for early identification of patients with severe pancreatitis. Out of these 11 objective parameters, five are measured at the time of admission, whereas the remaining six are measured within 48 hours of admission. Morbidity and mortality of the disease are directly related to the number of signs present. It is important to realize that Ranson's prognostic signs are best used within the initial 48 hours of hospitalization and have not been validated for later time intervals.

Another set of criteria often used to assess the severity of pancreatitis is the *a*cute *p*hysiology *a*nd *c*hronic *h*ealth *e*valuation (APACHE-II) score. This grading system assesses severity on the basis of quantitative measures of abnormalities of multiple variables, including vital signs and specific laboratory parameters, coupled with the age and chronic health status of the patient. The main advantage of the APACHE-II scoring system is the immediate assessment of the severity of pancreatitis. A score of eight or more at admission is usually considered indicative of severe disease. APACHE II, although complicated, ensures the highest positive predictive value up to 69%. [19]

The risk of severe acute pancreatitis is increased at Glasgow's or Ranson's score ≥3 in 48 hours, APACHE II on admission ≥8, Balthazar's score ≥4.

In 1985, Balthazar and colleagues introduced a scoring system based on radiological findings by means of a 5- grade scale: the presence of pancreatic and peripancreatic inflammation and fluid accumulation. [20].

Grade CT findings:

Grade A Normal pancreas

Grade B Pancreatic enlargement

Grade C Pancreatic inflammation and/or peripancreatic fat

Grade D Single peripancreatic fluid collection

Grade E Two or more fluid collections and/or retroperitoneal air.

A correlation was shown between the grade on CT performed within 10 days of admission and the clinical follow-up finding, morbidity, and mortality. Therefore, CT was appreciated as a useful prognostic indicator for outcome in Acute pancreatitis. The study showed a morbidity of only 4% and no mortality in patients with Acute pancreatitis and a CT grade of A, B, or C. In patients with CT grade D or E, the morbidity rate was 54% and the mortality 14%. The Balthazar radiological prognostic score was easy to assign without the need of contrast-enhanced CT. Unfortunately, this score did not assign any value to pancreatic necrosis as a prognostic parameter and did not make the distinction between Acute fluid collections and pseudocysts vs. post-necrotic fluid collections and walled-off pancreatic necrosis. With the introduction of newer CT-based scoring systems, some authors question the value of Balthazar's score in predicting prognosis and severity in Acute Pancreatitis [21].

9. Treatment

Gallstones are the most common cause of acute pancreatitis worldwide. According to the physical examination, radiological findings and labarotory results the etiology of the acute pancreatitis is diagnosed as biliary or non-biliary. The most important initial treatment of biliary pancreatitis is conservative intensive care with the goals of oral food and fluid restriction, replacement of fluids and electrolytes parenterally as assessed by central venous pressure and urinary excretion, and control of pain. [22], [23]

After stabilizing the patient, specific treatment and timing of the intervention have to be planned. The issue of when to intervene for clearance of gallstones is controversial. General consensus is either urgent intervention (cholecystectomy) within the first 48 to 72 hours of admission, or briefly delayed intervention (after 72 hours, but during the initial hospitalization) to give an inflamed pancreas time to recover. Cholecystectomy and common duct clearance is the best treatment of biliary acute pancreatitis. Patients who have persistent impacted stone in the distal common bile duct or ampulla should have confirmation by radiologic imaging (CT, magnetic resonance cholangiopancreatography, or endoscopic ultrasonography) before intervention. If common duct stone are diagnosed, stones are cleared and endoscopic sphincterotomy is done by ERCP and then laparoscopic cholecystectomy is performed.[24]

Routine ERCP for examination of the bile duct is discouraged in cases of biliary pancreatitis, as the probability of finding residual stones is low, and the risk of ERCP-induced pancreatitis is significant. But in the case of acute biliary pancreatitis in which analytical studies suggest that the obstruction persists after 24 hours of observation, emergency ERCP has to be done to prevent biliary sepsis.

Although ERCP with Endoscopic Sphincterotomy (ES) and stone extraction has been shown to be useful for early treatment of severe biliary pancreatitis, the incidence of bile duct stones at elective surgery is low and most of these ERCP are unnecessary. For this reason accurate predictors of common bile duct stones are required; studies have shown that the sensitivity of preoperative abdominal US for predicting common bile duct stones is 42% and specificity is 86% [25]. Furthermore, an endoscopic approach is unable to fully resolve the patient's biliary pathology with one procedure and one anesthesia. This adds substantial risk of morbidity and even mortality. Concern remains also regarding the potential long-term risks of ES. Although the immediate complications of ES are well documented, the long-term effects are less defined. Stricture formation and stone recurrence account for nearly all longterm complications. Although most of the authors prefer the endoscopic to the surgical treatment of CBD stones, there is still some minor discussion on it[26].

Timing of laparoscopic surgery in acute biliary pancreatitis depends upon the severity of the disease. In the case of mild pancreatitis it doesn't matter when, within 1 week, laparoscopic cholecystectomy is performed. However, in patients with severe pancreatitis, laparoscopic cholecystectomy, when performed within the 1st week after the onset of symptoms, as other authors have observed [27], places patients at increased risk of operative morbidity and technical complications. In these patients, the management of complications of pancreatitis is strongly advisable before cholecystectomy.

Delaying surgery for more than a week after hospitalization, in our experience, does not adversely affect technical difficulty. Delaying surgery for several weeks in severe acute pancreatitis allows acute inflammation to settle down and might allow stones in the

common bile duct to clear spontaneously. However, studies showed that approximately one-quarter of patients have symptomatic recurrence within 6 weeks if gallstones are untreated, and it increases with time [28], [29]

Cholangiogram of good quality during laparoscopic cholecystectomy, since the risk of common bile duct stones is 14–20% . [30] This strategy minimizes the need for common bile duct exploration and still achieves the goal of a limited hospital stay and the prevention of recurrence of pancreatitis. If common bile duct stones are found at cholangiogram they should be treated laparoscopically if at all possible. In most instances, it should be possible to retrieve the stones via the cystic duct, since acute pancreatitis is usually caused by the migration of small stones. If this is not feasible, one alternative is to perform a laparoscopic choledochotomy. These cases have a rather long hospital stay and delayed return to work, but their level of pain is diminished. Our current impression is that this procedure is possible though technically demanding. In case of failure, traditional exploration is mandatory.

In severe acute pancreatitis, or when signs of infection are present, most experts recommend broad-spectrum antibiotics (e.g., imipenem) and careful surveillance for complications of the disease.

10. Complications of acute pancreatitis

Acute pancreatitis complications may be divided as systemic and local. Pancreatic phlegmon, pancreatic abscess, pancreatic pseudocyst, pancreatic ascites and involvement of adjacent organs, with hemorrhage, thrombosis, bowel infarction, obstructive jaundice, fistula formation, or mechanical obstruction are local complications. Systemic complications are classified as hematologic (Hemoconcentration, Disseminated intravascular coagulopathy), cardiovascular (Hypotension, Hypovolemia, Sudden death, Nonspecific ST-T wave changes, Pericardial effusion), pulmonary (Pneumonia, atelectasis, Acute respiratory distress syndrome, Pleural effusion), renal (Oliguria, Azotemia, renal artery/vein thrombosis), metabolic (Hyperglycemia, Hypocalcemia, Hypertriglyceridemia, Encephalopathy, Sudden blindness (Purtscher's retinopathy), central nervous system (Psychosis, Fat emboli, Alcohol withdrawal syndrome), gastro intestinal system (Peptic ulcer, Erosive gastritis, Portal vein or splenic vein thrombosis with varices)

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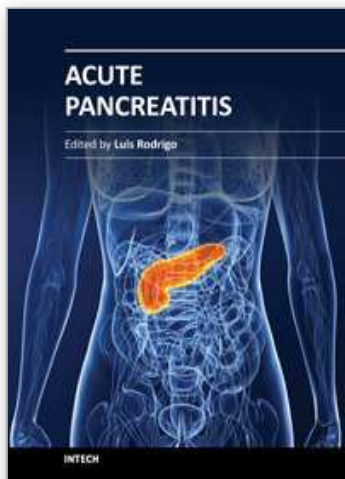
Acute Pancreatitis

Edited by Prof. Luis Rodrigo

Acute Pancreatitis (AP) in approximately 80% of cases, occurs as a secondary complication related to gallstone disease and alcohol misuse. However there are several other different causes that produce it such as metabolism, genetics, autoimmunity, post-ERCP, and trauma for example... This disease is commonly associated with the sudden onset of upper abdominal pain that is usually severe enough to warrant the patient seeking urgent medical attention. Overall, 10-25% of AP episodes are classified as severe. This leads to an associated mortality rate of 7-30% that has not changed in recent years. Treatment is conservative and generally performed by experienced teams often in ICUs. Although most cases of acute pancreatitis are uncomplicated and resolve spontaneously, the presence of complications has a significant prognostic importance. Necrosis, hemorrhage, and infection convey up to 25%, 50%, and 80% mortality, respectively. Other complications such as pseudocyst formation, pseudo-aneurysm formation, or venous thrombosis, increase morbidity and mortality to a lesser degree. The presence of pancreatic infection must be avoided.

How to reference

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