Chapter 11
Acute Pancreatitis

Ari Leppäniemi

11.1 Etiology and Pathogenesis

Alcohol and gallstone disease are the two commonest etiological factors for acute pancreatitis comprising about 70–80 % of the patients. The other causes are much rarer and include hypercalcemia, hypertriglyceridemia, trauma, a variety of drugs, infections, postoperative conditions (e.g., cardiac surgery), endoscopic retrograde cholangiopancreatography (ERCP), developmental anomalies (such as pancreas divisum), tumors, and hereditary and autoimmune diseases. In about 10 % of the cases, the etiology remains unknown.

The main pathogenic determinant in acute pancreatitis is the excessive activation of a systemic inflammatory response cascade leading to multiple organ dysfunction. At first a triggering factor is needed to initiate the pancreatic acinar cell injury. After several intracellular events, pancreatic proenzymes (zymogens) become activated intracellularly, resulting in acinar cell injury. This is fol-

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lowed by local inflammation of the pancreas resulting in activation of several inflammatory cells and release of inflammatory mediators. If this inflammation cannot be controlled locally, excessive uncontrolled activation of inflammatory cells and mediators leads to a systemic inflammatory response syndrome (SIRS) that is similar to other SIRS-associated conditions, such as sepsis or severe trauma, for example. Leaking microvessels cause a loss of intravascular fluid and in conjunction with vasodilatation lead to hypotension and shock. Accumulation of inflammatory cells in tissues, increased interstitial fluid, and activation of coagulation with microvascular thrombosis further impair oxygen supply of tissues. Clinical manifestation of all this is a multiple organ dysfunction syndrome (MODS), characterized by dysfunction or failure of the respiratory, cardiovascular, renal, hepatic, hematological, gastrointestinal, and central nervous system functions. MODS usually develops early during the course of the disease, and over half of the patients with severe acute pancreatitis have signs of organ dysfunction on hospital admission.

Recently, increased intra-abdominal pressure (IAP) and the development of abdominal compartment syndrome (ACS) have been recognized as significant contributors to the development of early MODS in severe acute pancreatitis. If the patient survives the initial inflammatory insult, a second critical phase usually follows 2–4 weeks later with the appearance of septic, local, and other complications. Infection of the pancreatic and peripancreatic necrosis occurs in about 20–40 % of patients with severe acute pancreatitis and is associated with worsening MODS.

According to the updated Atlanta classification 2012, the peripancreatic collections associated with necrosis are acute necrotic collection (ANC) and walled-off necrosis (WON). In the early phase, poorly demarcated “acute peripancreatic fluid collections” are commonly seen on CT scan. They are homogenous, are confined to normal fascial planes, can be multiple, usually remain sterile, and resolve spontaneously without intervention. A “pancreatic pseudocyst” refers to a well-defined fluid

collection containing no solid material. The development of pancreatic pseudocyst is extremely rare in acute pancreatitis and is often confused with ANC. However, it may form many weeks after operative necrosectomy due to localized leakage of a disconnected duct in the necrosectomy cavity.

ANC is a collection seen during the first 4 weeks and containing variable amount of fluid and necrotic tissue involving the pancreatic parenchyma and/or peripancreatic tissues (Fig. 11.1). WON is a mature, encapsulated collection of pancreatic and/or peripancreatic necrosis with a well-defined, enhancing inflammatory wall (Fig. 11.2). The maturation takes usually 4 weeks or more after the onset of acute pancreatitis.

11.2 Diagnosis and Estimation of Severity

Previous medical history can consist of previous episodes of acute pancreatitis; previously known gallstone disease or symptoms typical for biliary colic; chronic pancreatitis; metabolic disorders, such as hyperparathyroidism or hyperlipemia; history of a recent abdominal trauma; surgical or endoscopic procedures; new drugs; infections; and family history of acute pancreatitis. Sudden pain in the epigastrium, often radiating into the back and feeling like a belt around the upper abdomen, is the most common symptom and is usually constant rather than colicky. Nausea and vomiting are frequent. Fever is common in patients with accompanying cholangitis.

In severe form with unstable vital signs, securing airways and adequate ventilation and starting fluid resuscitation in hypovolemic shock should precede any diagnostic work-up. In addition to the assessment of hemodynamic, pulmonary, and renal functions, abdominal examination is crucial and should consist of inspection noting abdominal distension (caused by ileus, ascites, visceral edema) and possible discolorations around the

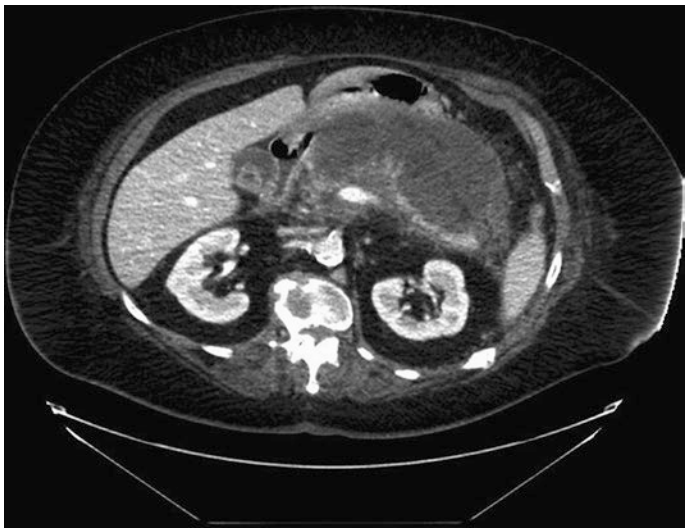

Fig. 11.1 Acute necrotic collection (ANC)

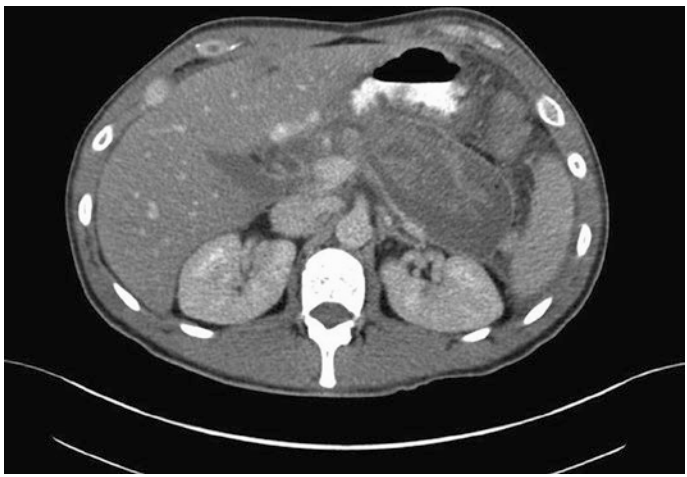

Fig. 11.2 Walled-of necrosis (WON)

umbilicus (Cullen's sign) or in the flanks (Grey Turner's sign). Palpation shows epigastric or generalized tenderness, percussion can reveal significant amount of ascites, and auscultation detect the absence of bowel sounds if the patient has paralytic ileus. Furthermore, general findings indicative of alcohol abuse, hyperlipemia, and other general disorders can help in determining the etiology.

Laboratory examinations usually show elevated plasma amylase (or lipase) levels, but the amylase levels may have returned to normal, if several days have passed from the onset of symptoms. C-reactive protein level (CRP) is a useful clinical marker of the severity, but it lags 24–48 h behind and can be completely normal in the initial phase of even a severe form of the disease. Blood count, liver function tests, electrolyte and glucose levels, as well as creatinine should be taken routinely, and in severe cases, arterial blood gas analysis and serum lactate measurements show the extent of cellular hypoperfusion. Triglyceride levels should be measured if known or suspected to be the cause.

The most reliable diagnostic method for acute pancreatitis is the CT scan. Except for differential diagnosis (free intra-abdominal air) when CT is not available or is too time consuming, plain abdominal radiographs are not needed and chest radiographs may be obtained to evaluate pulmonary status. In performing the CT scan, oral contrast can be administered (but is not necessary), whereas intravenous contrast material should be used with caution and only after confirming adequate circulating volume and urine output. CT scan without intravenous contrast is sensitive in detecting acute pancreatitis. Later on in patients with necrotizing pancreatitis, the contrast enhancement and patency of the pancreas itself can be evaluated using intravenous contrast CT scan.

Ultrasound is useful in identifying gallstones in the gallbladder and a dilated common bile duct when duct stones or cholangitis is suspected. In some cases, magnetic resonance cholangiopancreatogram (MRCP) can be used for suspected

bile duct stones and is sometimes helpful to confirm that a common bile duct stone has passed through to the duodenum, thus saving an unnecessary ERCP examination. However, ERCP is needed when ultrasonography reveals dilated common bile duct and there is a suspicion of a persistent stone or the patient has signs of cholangitis. Endoscopic sphincterotomy with clearance of the common duct from stones and/or drainage of pus (in cholangitis) is justified, even if it does not change the natural course of the pancreatitis itself.

The amount or progression of amylase levels do not correlate with severity; CRP >150 mg/L is better but manifests only 24–48 h later, and other markers such as procalcitonin are not in everyday clinical use. Clinical scoring systems such as those described by the late Ranson or Imrie are inaccurate and not used anymore. APACHE II score >8 demonstrates fairly accurately the acuity of the disease indicating significant physiological derangement, but probably the best way to monitor and quantify the organ dysfunction is by using the Sequential Organ Failure Assessment (SOFA) score and especially its cardiovascular, pulmonary, and renal components to determine if the patient should go to the ICU directly from the emergency room.

Although there is no reliable single marker to differentiate between edematous and necrotizing acute pancreatitis, the combination of clinical evaluation, CRP, CT scan, and the presence or absence of organ dysfunctions are usually sufficient. If severe form of acute pancreatitis is suspected or anticipated and especially if the patient already has signs of organ dysfunction, early admission to an intensive care or high dependency unit is mandatory in order to be able to monitor and support vital organ functions.

The most common differential diagnoses include diseases presenting with acute epigastric or mid-abdominal pain and include perforated peptic ulcer, biliary colic, acute cholecystitis, ruptured abdominal aortic aneurysm, reflux esophagitis, acute mesenteric ischemia, intestinal obstruction, acute hepatitis, inferior myocardial infarction, and basal pneumonia. It is particularly important to differentiate between secondary peritonitis

caused by hollow organ perforation usually requiring urgent surgery and acute pancreatitis where early surgery is usually harmful. Therefore, when in doubt, a CT scan is important provided that it does not delay the initiation of treatment in critically ill patients, whether having pancreatitis or peritonitis.

11.3 Treatment

11.3.1 Mild Acute Pancreatitis

The treatment of mild or edematous pancreatitis is mainly supportive consisting of fluid resuscitation and therapy, pain medication, and sometimes the management of accompanying delirium tremens in patients with alcohol-induced pancreatitis. Urine output should be monitored, usually with the placement of a Foley catheter (goal 0.5–1.0 ml/kg/h), and adequate volume restoration secured. Nasogastric tube is not routinely indicated, but is helpful in patients with dilated stomach or paralytic ileus. Oral feeding should be started as soon as it is tolerated. Any signs of severe pancreatitis should be noted early (clinical condition, CRP, organ dysfunctions) and evaluated for the need to admit the patient to the ICU.

In patients with mild biliary pancreatitis, laparoscopic cholecystectomy can be performed before discharging the patient.

11.3.2 Severe Acute Pancreatitis

11.3.2.1 Fluid Resuscitation

Aggressive fluid therapy during the early phase of acute pancreatitis used to be one of the cornerstones in the early treatment phase of severe pancreatitis, but gradually the negative effects of

excessive fluid resuscitation have been recognized, and a more measured and moderate policy of fluid resuscitation has become the standard. No doubt, the rationale behind aggressive fluid resuscitation was sound, that is, to correct hypovolemia caused by third-space fluid loss. However, excess volume loading may increase intra-abdominal pressure (IAP) and cause intra-abdominal hypertension (IAH) or even abdominal compartment syndrome (ACS). Unfortunately, there are no good resuscitation end points for specific severe acute pancreatitis, and one has to rely on the more common end points similar to other diseases causing severe physiological derangement, such as severe sepsis or septic shock. The principles of early goal-directed resuscitation including monitoring of central venous pressure (CVP), mean arterial pressure (MAP), and either central venous oxygen saturation or mixed venous oxygen saturation can be used. In addition, IAP should be monitored and the abdominal perfusion pressure (APP=MAP-IAP) calculated. The APP could also serve as a good resuscitation end point, at least in patients with IAH. Maintaining APP above 50–60 mmHg is needed in order to provide sufficient perfusion to the abdominal organs.

Base deficit and blood lactate levels should be monitored, and resuscitation should be targeted to normalize the lactate level. As soon as the set resuscitation end points are reached, the infusion rate should be slowed down in order to avoid fluid overloading.

11.3.2.2 Enteral Nutrition

Fasting does not help, and it does not alleviate the inflammatory response. Enteral feeding is superior to parenteral feeding, and the only contraindication is poor motility of the gastrointestinal tract. Enteral nutrition prevents bacterial overgrowth in the intestine and reduces bacterial translocation and reduces the risk of systemic infections, organ dysfunction, and mortality.

Besides, all critically ill patients are at risk of malnutrition, and therefore enteral nutrition of patients with severe acute pancreatitis should be started as soon as possible.

The route of enteral feeding can be either gastric or postpyloric. Most patients tolerate gastric feeding via a nasogastric tube, but the residuals should be monitored every 6 h. If gastric feeding is not possible because of impaired gastric emptying and not relieved with the use of erythromycin or other prokinetics, a nasojejunal feeding type should be inserted either with the help of endoscopy or using self-advancing tubes.

Tube feeding should be started slowly, 10 ml/h, for example, and increased by 10 ml/h every 6 h providing that gastric residual volume is below 250 ml. This should be continued until the target volume of enteral nutrition is achieved. Volumes should not exceed 60 ml/h to avoid the rare but catastrophic complication of bowel necrosis. If the patient does not tolerate enteral nutrition in sufficient volumes, parenteral nutrition can be combined with enteral nutrition to fulfill the nutritional requirements.

11.3.2.3 Antibiotics

About 25 % of the patients with acute pancreatitis suffer from an infectious complication, and they are more common in patients with severe acute pancreatitis. The majority of infections in patients with severe acute pancreatitis are extrapancreatic, such as bacteremia or pneumonia, and half of them develop during the first week after admission. Infection of the pancreatic or peripancreatic necrosis comes usually later and peaks at about week 3–4. The risk factors for infected necrosis include early bacteremia, organ failure, and extent of necrosis.

The diagnosis of infected necrosis is controversial, and the earlier reliance on fine needle aspiration (FNA) of the necrosis, usually performed with ultrasound guidance, has been ques-

tioned, as it has been shown to have a false-negative rate of 20–25 %.

Clinical signs of sepsis are too unspecific for definitive diagnosis, although a new increase in the CRP value without any other good explanation might alert you to look for the infected necrosis. Gas bubbles in the CT scan are reliable signs of infection, but they are present only in less than 10 % of patients with infected necrosis.

There are many randomized controlled trials showing that prophylactic antibiotics do not benefit patients with acute pancreatitis. However, when looking at the studies more carefully, there has been a nonsignificant trend for lower mortality and reduced number of infections, especially extrapancreatic infections in patients treated with prophylactic antibiotics. The randomized trials have been conducted with small sample sizes, and some studies included a substantial number of patients with mild pancreatitis with minimal risk of mortality and low risk of infectious complications. Acknowledging the limitations of the trials and that patients with organ failure are susceptible to infections, some surgeons use prophylactic antibiotics in patients with severe pancreatitis at least when they have organ dysfunctions and are admitted to the ICU. Clinical judgment taking into account the presence of SIRS, the presence of IAH, hyperglycemia, low plasma calcium, high creatinine, or other signs of organ dysfunction can be used to guide the decision-making.

If prophylactic antibiotics are not given, empiric use of antibiotics is appropriate in patients who develop organ dysfunctions, because of the high risk of bacteremia during the first week. After the end of the second week, empiric antibiotics may be needed for treatment of infected pancreatic necrosis if sepsis continues or the patient does not recover. The antibiotics should cover gram-negative rods and gram-positive cocci. The role of empiric antifungals is not clear. FNA for

microbiological samples should be taken if infected necrosis is suspected, although negative samples do not rule out infection. Positive samples help in the selection of antimicrobials and initiation of possible antifungal therapy. Whatever the reason for starting antibiotics, they should be discontinued when the patient recovers from organ dysfunctions, and there is no evidence of infection.

The principles of early management of acute pancreatitis are summarized in Table 11.1.

11.3.2.4 Surgical Management

In addition to surgical or endoscopic interventions required for gallstone-associated pancreatitis, there are a few reasons to operate on patients with severe acute pancreatitis, and the majority of patients never develop these complications.

Table 11.1 Early management principles in severe acute pancreatitis

Early and timely admission to an intensive care or high dependency unit
Fluid resuscitation goals:
MAP > 65 mmHg
SvO_2 > 65 % (requires pulmonary artery catheter)
Normal lactate level
Urine output >0.5–1.0 ml/kg/h
IAP measurement every 4–6 h of IAP
Vasoactive support (norepinephrine and dobutamine if cardiovascular failure)
Goal: APP (MAP-IAP) > 60 mmHg
Analgesia, sedation, lung-protective ventilation
Normoglycemia
Thrombosis prophylaxis
Early enteral feeding
Prophylactic antibiotics
Early biliary decompression, if obstruction (especially, if cholangitis)

11.3.3 Abdominal Compartment Syndrome

The combination of excessive fluid resuscitation and capillary leakage lead to tissue edema of the abdominal and retroperitoneal organs, and ascites formation. Intestinal paralysis usually adds to the increase of the intra-abdominal volume. The extra need for space can partly be compensated by the increase in the abdominal domain, but at some stage, the reserve capacity is used, and the intra-abdominal pressure (IAP) starts to increase leading to intra-abdominal hypertension (IAH). The incidence of IAH in patients with acute pancreatitis admitted to ICU is about 60 %, and the incidence of the clinical syndrome of abdominal compartment syndrome (ACS) comprising of IAP >20 mmHg and a new-onset organ dysfunction can be as high as 27 % as reported in the largest published series. All patients treated for severe acute pancreatitis should undergo repeated and routine measurement of the IAP, usually via a urinary bladder catheter. Already IAP levels of 12 mmHg impair renal function. In patients with IAH, the abdominal perfusion pressure (APP = MAP-IAP) should be calculated because patients in shock can easily have inappropriately low APP (<50–60 mmHg) even with moderate IAH. Poor perfusion increases bowel mucosal injury which is associated with infectious complications and organ failure. In addition, IAH may play significant role in ischemic bowel complications, especially colonic necrosis or even small bowel ischemia.

Although adequate fluid resuscitation is important in the early phase of severe acute pancreatitis, excessive volumes should be avoided. Prevention and management of gastric dilatation with a nasogastric tube and percutaneous drainage of excessive pancreatic ascites are useful adjuncts to nonoperative management. Short-term use of neuromuscular blockers may also be considered. Removal of fluid by extracorporeal techniques is effective in rapidly removing excess fluid.

When nonsurgical interventions fail to change the progressive deterioration of organ dysfunctions in the presence of fulminate ACS, surgical decompression should be considered. The most commonly used method is midline laparostomy, where all abdominal wall layers are divided through a vertical midline incision extending from the xiphoideum to the pubis with a few centimeters of fascia left intact at both ends to facilitate subsequent closure or late reconstruction. An alternative method utilizes a bilateral subcostal incision few centimeters below the costal margins. A less invasive technique is the subcutaneous linea alba fasciotomy (SLAF) where the fascial alone is divided through three short horizontal skin incisions leaving the peritoneum intact. The aim of surgical decompression, whatever method is used, is to achieve adequate APP of >60 mmHg. Opening the abdomen to reduce IAP is associated with severe morbidity most commonly associated with the management and complications of the open abdomen, such as enteric fistulas and giant ventral hernias. The best temporary abdominal closure technique seems to be the vacuum-assisted wound closure combined with mesh-mediated fascial traction. It has the highest fascial closure rate (80–90 %) and lowest enteric fistula rate when compared with the other currently available techniques.

11.3.4 Infected Pancreatic Necrosis

According to the updated Atlanta classification 2012, the peripancreatic collections associated with necrosis are acute necrotic collection (ANC) and walled-off necrosis (WON). In the early phase, poorly demarcated "acute peripancreatic fluid collections" are commonly seen on CT scan. They are homogenous, are confined to normal fascial planes, can be multiple, usually remain sterile, and resolve spontaneously without intervention.

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pancreatic pseudocyst is extremely rare in acute pancreatitis and is often confused with ANC. However, it may form many weeks after operative necrosectomy due to localized leakage of a disconnected duct in the necrosectomy cavity.

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Infected necrosis is a significant source of sepsis, and removal of devitalized tissue is believed to be necessary for control of sepsis. However, infection usually continues after necrosectomy, especially if necrotic tissue is left in place. Before demarcation of necrosis develops, usually after 4 weeks from disease onset, it is impossible to remove all necrotic tissue without causing bleeding, and too early surgical debridement is associated with high risk of hemorrhage leading to increased organ dysfunction and death. Because high mortality is associated with early surgery and multiple organ dysfunction, surgery for infected necrosis should be postponed as late as possible, preferable later than 4 weeks from the onset of the disease.

Percutaneous drainage of the liquid component of the infected acute necrotic collection may serve as a bridge to surgery and sometimes suffices alone. Sterile collections do not need drainage, because placement of a drain into a sterile necrotic collection can result in secondary infection, especially after prolonged drainage. There are no randomized studies comparing operative treatment and catheter drainage in patients with worsening multiple organ failure within the first few weeks from disease onset. The only randomized trial comparing open necrosectomy and minimally invasive step-up approach included only 28 (32%) patients with multiple organ failure, and the median time of interventions was 30 days from disease onset. In

this study, the mortality rate was the same between the groups; no data of subgroup analysis of patients with multiple organ failure was shown.

Although the use of mini-invasive techniques are increasingly used for infected pancreatic necrosis, the lowest published mortality rate in patients operated on for infected necrosis is with open debridement and closed packing with 15 % mortality. In patients without preoperative organ failure, minimally invasive necrosectomy is associated with fewer new-onset organ failure than open surgery. However, a considerable number of patients are not suitable for mini-invasive surgery because of the localization of the necrotic collection.

According to the IAP/APA evidence-based guidelines for the management of acute pancreatitis, the indications for intervention (surgical, radiological, or endoscopic) in necrotizing pancreatitis are listed in Table 11.2. The timing of intervention is usually postponed until at least 4 weeks after the initial presentation to allow the WON to be formed, and for some of the other indications, it is more than 8 weeks.

The preferred technique for open necrosectomy used at our institution is as follows: transverse bilateral subcostal incision (often extending more to the left), dividing the gastrocolic ligament (we prefer not to go through the transverse mesocolon), opening the right tissue planes with blunt dissection, and utilizing harmonic scalpel or old-fashioned ligatures for good exposure. Usually the necrosis is mostly found around the pancreas, while the pancreas itself is firm and protrudes like a transverse ridge. In these cases it should be left alone. If on the other hand (and as might be suggested in a preoperative CT) the necrotizing process has destroyed the middle part of the pancreas, the distal part can usually be removed easily by squeezing it out distally with gentle finger dissection being careful not to damage the splenic vessels. The spleen is left intact if possible. Once the dead distal pancreas has been removed (sometimes only a small proximal remnant is left), one can try to find the

Table 11.2 Indications for surgical radiological or endoscopic indications in severe acute pancreatitis

Clinically suspected or documented infected necrosis with clinical deterioration or ongoing organ failure for several weeks
Ongoing gastric outlet, intestinal, or biliary obstruction due to mass effect of WON
Patient not getting better with WON but no infection (after 8 weeks)
Disconnected duct syndrome (full transection of the pancreatic duct) with persisting symptomatic collection with necrosis without signs of infection (>8 weeks)

divided pancreatic duct and ligate it. Usually it cannot be seen and a pancreatic fistula may occur, but that can be managed with an endoscopically placed stent later on. After removing the necrotic tissue, the area is packed for a few minutes and the hemostasis is secured with amply placed sutures. Minor oozing usually stops by itself. Draining the peripancreatic area with a couple of well-placed (one coming behind the left hemicolon into the pancreatic area if the necrosis is mainly on the left side) completes the procedure. Unless there is a risk of increased IAP, the wound is usually closed.

Endoscopic variations for the management of peripancreatic necrotic collections have been introduced and include endoscopic transgastric or retroperitoneal drainage or necrosectomy. The value of these techniques is still under assessment, and only small randomized series with well-selected patients have been published.

If the disease process has eroded the pancreas leaving a considerable portion of the distal pancreas intact (disconnected duct syndrome) and the patients develop symptomatic collections, the distal pancreatic remnant can be resected or connected to a Roux-en-Y loop with pancreaticojejunostomy. Although saving viable pancreatic tissue might be beneficial, the long-term benefits of internal drainage over resection have not been established.

11.3.5 Surgery for Extrapancreatic Complications

Bleeding is a rare complication in severe acute pancreatitis, but when occurring requires prompt management either by surgical intervention or angiographic embolization. Sometimes the bleeding has to be packed in a reoperation leaving the abdomen open and doing a reoperation two days later removing the packs.

Necrosis of a part of the colon in acute pancreatitis is associated with high mortality and is difficult to diagnose until perforation occurs. Gas bubbles in the colonic wall can be a useful hint. Colon necrosis is probably caused by retroperitoneal spread of the necrotizing process to colon with fat necrosis and pericolitis. Usually, the inner layers of colon remain viable longer. The most common places of colon necrosis are in the cecum where it is aggravated by dilatation or in the transverse colon where it can be related to the thrombosis of the middle colic artery branches associated with the peripancreatic necrosis. There should be a low threshold for colonic resection due to unreliable detection of ischemia or imminent perforation just by seeing the outside of the colon during surgical exploration. Obviously, in patients with clear perforation, removal of the affected segment is mandatory. Primary colonic anastomosis under these circumstances is risky, and a temporary colostomy is a safer option.

11.3.6 Biliary Surgery

The 2002 evidence-based guidelines of the International Association of Pancreatology recommended early cholecystectomy in mild gallstone-associated acute pancreatitis and delayed cholecystectomy in severe pancreatitis. Cholecystectomy should be delayed in patients with moderate to severe pancreatitis and

demonstrated peripancreatic fluid collections or pseudocysts until the pseudocysts either resolve or beyond 6 weeks, at which time the pseudocyst drainage can safely be combined with cholecystectomy. Therefore, in patients with severe gallstone-induced acute pancreatitis, cholecystectomy should be delayed until the inflammatory response resolves and clinical recovery occurs.

In patients with mild gallstone pancreatitis, laparoscopic cholecystectomy performed within 48 h of admission, regardless of the resolution of abdominal pain or laboratory abnormalities, is safe and results in a shorter hospital length of stay with no apparent impact on the technical difficulty of the procedure or perioperative complication rate. It has become more common for patients with mild biliary pancreatitis to undergo laparoscopic cholecystectomy at the same hospitalization period once the clinical signs of pancreatitis have resolved.

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