Management of Acute Pancreatitis in Elderly

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27.1 Aging Pancreas

In the US, pancreatitis is a major cause of morbidity and mortality based on the severity of the the diease [1]. Age-related changes in the pancreas include fatty infiltration, parenchymal atrophy, fibrosis, and ductal ectasia. These changes have a varying degree of impact on the functional ability of the pancreas resulting in a very limited or marginal exocrine and endocrine capacity of the pancreas in elderly patients [2]. Poor structural integrity and a limited functional reserve of the pancreas in addition to comorbidities contribute to the severity of pancreatitis. A complicated disease course leads to higher complication rates including multi-organ failure, diabetes, and exocrine insufficiency, and significantly higher mortality rates. Hastier et al. have reported that only one-third of the patients with an age >70 without a history of pancreatic pathology have duct diameters within normal defined limits [3]. These structural changes can pose a challenge in the diagnosis and management of acute pancreatitis in the elderly. These changes should be taken into account and are of prime importance while making the diagnosis or managing patients with pancreatitis.

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27.2 Etiology and Pathophysiology

27.2.1 Gallstones

Gallstone is the leading cause of acute pancreatitis in the US at any age. Approximately 35% of acute pancreatitis cases have an underlying biliary etiology. In the elderly, gallstones account for up to 75% of cases of acute pancreatitis. Old age is associated with lithogenicity of the bile hence a higher incidence of gallstones and resulting gallstone pancreatitis. It is proposed that blockade of the ampulla by a gallstone or edema induced by a passing gallstone is the inciting factor in the pathogenesis of acute pancreatitis [4].

27.2.2 Alcohol

Only 5% of the episodes of acute pancreatitis are related to alcohol use, which is significantly lower as compared to younger adults. However, alcohol is still the most common cause of chronic pancreatitis in the elderly. A potential mechanism by which alcohol induces pancreatitis include, spasm of the sphincter of Oddi, obstruction of small pancreatic ductules by proteinaceous material, metabolic abnormalities, and direct impact of alcohol and its metabolites [5].

27.2.3 Other Etiologies

Acute pancreatitis associated with metabolic and medication-induced etiologies is much higher in comparison to younger patients. This is related to a higher comorbidity burden, polypharmacy, and hence extensive drug interactions that are very difficult to manage. Common drug classes that are known to cause pancreatitis include ACE inhibitors, statins, diuretics, antiretroviral, anti-seizure, and hypoglycemic agents. Unfortunately, with advancing age, the probability of being on one or more of these above inciting medications increases exponentially. New onset of pancreatitis in the elderly with no obvious cause should also raise the suspicion of underlying carcinoma and a clinician should have a low threshold to rule out malignancy in elderly patients [6].

27.3 Clinical Presentation

The most prevalent features in patients presenting with acute pancreatitis are abdominal pain, nausea, and vomiting. Pain is usually localized to the epigastrium; it classically radiates towards the back and patients report relief with sitting up and leaning forward. The most common sign is epigastric tenderness. Depending on the severity of pancreatitis, patients may present hemodynamic lability, which can be drastic in the elderly due to limited reserve. Although it is not very common, patients

with necrotizing pancreatitis and retroperitoneal hemorrhage can present with Grey Turner sign (bruising of the flanks), Cullen sign (bruising in the periumbilical region), or Fox sign (bruising in the inguinal region resulting from blood dissection into subcutaneous tissues of the respected areas).

27.4 Diagnosis

27.4.1 Lab Workup

Pancreatic injury results in the release of a variety of digestive enzymes from acinar cells that escape into the systemic circulation. Amylase is one of the most commonly assayed enzymes to confirm the diagnosis of acute pancreatitis. Amylase levels rise within several hours after the onset of symptoms and typically remain elevated for 3–5 days during uncomplicated episodes of mild acute pancreatitis. Amylase has a short half-life of about 10 h; the downside to this is that levels can normalize as soon as 24 h after onset of the disease. The sensitivity of this test depends on what threshold value is used to define a positive result (90% sensitivity with a threshold value just above the normal range vs. 60% sensitivity with a threshold value at three times the upper limit of normal). Specificity is limited because a wide range of disorders can cause elevations in serum amylase concentration.

Serum lipase concentrations have similar kinetics as those of amylase. As compared to amylase, serum lipase has a longer serum half-life; however, it may be useful for diagnosing acute pancreatitis late in the course of an episode. Generally, lipase is more specific than amylase in the diagnosis of acute pancreatitis.

27.5 Imaging

27.5.1 Ultrasonography

Ultrasonography can be useful in visualizing the pancreas in thin and lean patients, but this is highly dependent on expertise. Ultrasonographic images can reveal a diffusely enlarged, hypoechoic pancreas. However, overlying bowel gas (Ileus) severely limits the visualization of the pancreas in a large percentage of cases. Although Ultrasound has poor sensitivity and specificity for detecting pancreatic pathology, ultrasonography plays a vital role in the identification of the etiology of pancreatitis, i.e., the detection of gallstones.

27.5.2 Computed Tomographic Scan

The most important imaging test in the evaluation of acute pancreatitis is CT scanning. CT scan images obtained with intravenous contrast provide vital information based on which pancreatitis can be classified as either interstitial edematous or

necrotizing pancreatitis. Findings of mild acute pancreatitis include pancreatic enlargement and edema, effacement of the normal lobulated contour of the pancreas, and stranding of peripancreatic fat. Whereas necrotizing pancreatitis is characterized by hypo-enhancing areas within pancreatic parenchyma or surrounding tissue. Furthermore, CT scans later in the course of the disease help in making the diagnosis of local complications including infected necrosis or pseudocyst and walled-off necrosis [7].

27.6 Assessment of Severity

Approximately, 70–80% of acute pancreatitis are mild and generally resolve within 5–7 days with minimal therapy. Overall mortality with the mild disease is less than 1%. About 20% of the patients present with a severe disease, either a severe local disease or a severe systemic disease leading to multi-organ failure. In severe disease, the mortality rate goes as high as 20% or even higher in elderly patients due to limited organ reserve and poor overall resilience. Age is one of the most important factors that is associated with adverse outcomes in patients with acute pancreatitis. Friability of the pancreas and limited organ reserve put the elderly patient at a higher risk of both severe local disease as well as multi-organ failure. Moreover, failure to rescue once the elderly patient suffers from severe disease is much higher as compared to their younger counterparts. Multiple scores and criteria exist for early prediction of the severity of disease and hence appropriate triaging and resource allocation. Two of the most commonly used criteria for severity assessment, i.e., Ranson and APACHE use age as a major factor to stratify patients who are at higher risk of developing serve disease and resulting complications [8, 9].

27.6.1 Revised Atlanta Criteria

The Atlanta Classification system was developed at a consensus conference in 1992 to establish standard definitions for the classification of acute pancreatitis. Revision of the Atlanta Classification provides a detailed system that emphasizes disease severity and includes comprehensive definitions of pancreatic and peripancreatic collections. According to revised Atlanta Criteria, the diagnosis of AP requires two of the following three features: (1) abdominal pain consistent with acute pancreatitis; (2) serum lipase activity (or amylase activity) at least three times greater than the upper limit of normal; or (3) characteristic findings of acute pancreatitis on contrast-enhanced computed tomography (CECT). In addition, based on the CECT criteria, acute pancreatitis is divided into two distinct types: acute interstitial edematous pancreatitis and acute necrotizing pancreatitis (ANP) (Figs. 27.1 and 27.2). ANP is further subdivided into pancreatic parenchymal necrosis, or peripancreatic necrosis or both combined. Local complications refer to the presence of peripancreatic fluid collections which are classified as acute peripancreatic fluid collection (APFC), pancreatic pseudocyst (Fig. 27.3), acute necrotic collection (ANC), and walled-off necrosis (WON) (Fig. 27.4). Characteristic and CT appearance of these findings are

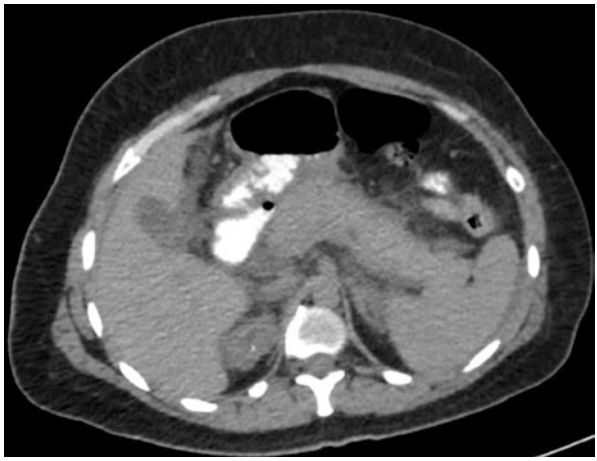

Fig. 27.1 Acute interstitial edematous pancreatitis

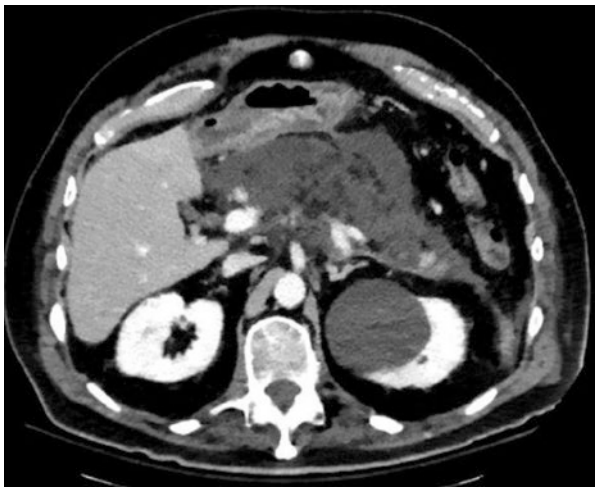

Fig. 27.2 Necrotizing pancreatitis with ccute necrotic collection

summarized in Table 27.1. Systemic complications are defined as exacerbation of preexisting comorbidities, i.e., chronic obstructive pulmonary disease (COPD), chronic kidney disease (CKD), congestive heart failure (CHF), and chronic liver disease (CLD). Organ failure is defined based on the Modified Marshall Scoring system, which evaluates the dysfunction of three major organ systems, respiratory, renal, and cardiovascular based on PaO2 to FiO2 ratio, serum creatinine, and systolic blood pressure, respectively. Each organ system is scored from 0 to 4 based on the degree of dysfunction. According to Revised Atlanta Criteria, mild acute pancreatitis is defined as the absence of organ failure and local or systemic complications. Moderately severe pancreatitis is characterized by transient organ failure (lasting less than 48 h) and/or local or systemic complications. Severe acute pancreatitis is associated with persistent organ failure (>48 h) single or multiple [7].

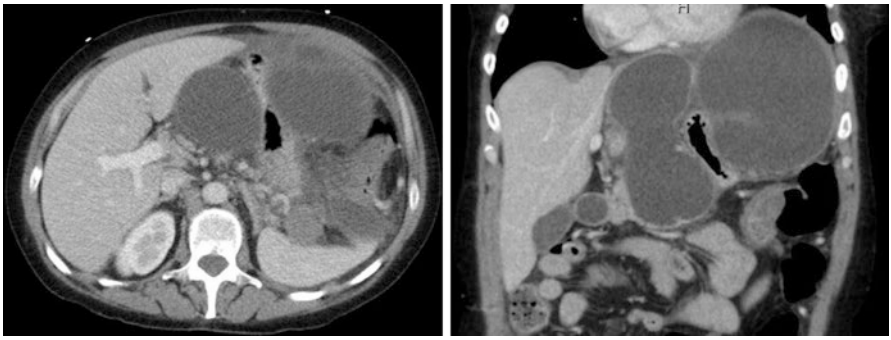

Fig. 27.3 Pancreatic pseudocyst

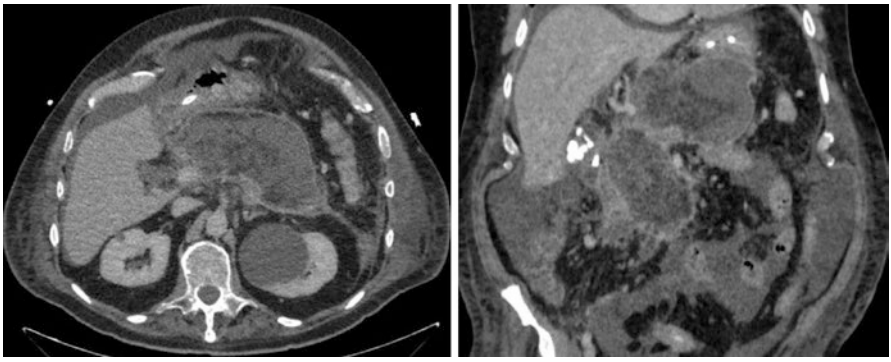

Fig. 27.4 Walled-off necrosis

Table 27.1 Types of fluid collections

Type of collection	Type of pancreatitis	Description	CT scan characteristics
Acute peripancreatic fluid collection (APFC)	Acute interstitial edematous pancreatitis	Within 4 weeks after onset.	Homogeneous fluid density collection surrounded by normal peripancreatic fascial planes. No definable wall or encapsulation
Pancreatic pseudocyst	Acute interstitial edematous pancreatitis	>4 weeks after onset	Homogeneous fluid collection, well-circumscribed. Encapsulated with no solid component or septa
Acute necrotic collection (ANC)	Acute necrotizing pancreatitis	Within in 4 weeks after onset	Heterogeneous collection with solid (necrotic component) and liquid components. No definable wall encapsulation
Walled-off necrosis (WON)	Acute necrotizing pancreatitis	>4 weeks after onset	Heterogeneous, with varying degree of solid and liquid components. Encapsulated with well-defined wall

Modified Atlanta Criteria

27.6.2 Ranson Criteria

Ranson Criteria is based on age, white blood cell count, glucose, serum lactate dehydrogenase (LDH), aspartate aminotransferase (AST) determined on admission, and drop in hematocrit, blood urea nitrogen (BUN), serum calcium, PaO2, base deficit, and fluid requirements measured 48 h post-admission shown in Table 27.2. Based on the abovementioned variables, the score is calculated correlating with morbidity and mortality related to acute pancreatitis. Limitations to Ranson criteria are that the score cannot be calculated until after 48 h and it can only be used once [8].

27.7 Treatment Strategies

27.7.1 Initial Resuscitation

The most important component of initial management is the assessment of fluid deficit and resuscitation. Due to widespread systemic inflammatory response, third space fluid losses can be immense and often underestimated. In elderly patients special care should be taken as over resuscitation can exacerbate CHF, which can further complicate the problem and sometimes can be fatal. Frequent reassessment of intravascular volume, aggressive fluid administration, and electrolyte replacement should be ensured. Patients with severe acute pancreatitis or those who failed to respond to initial fluid resuscitation are best managed in an intensive care unit setting with close cardiopulmonary monitoring. The degree and intensity of monitoring are tailored to disease severity and a comorbidity burden especially in the elderly. Urinary catheter should be inserted to monitor adequacy of urine output and watch for impending renal failure. Nasogastric tubes have been previously advocated to avoid pancreatic stimulation. There is no clinical data that supports this practice and hence should only be limited to patients with altered mental status, increased risk of aspiration, or present with paralytic ileus and intractable vomiting.

Table 27.2 Ranson's criteria

Ranson criteria and prognosis	
At admission	At 48 h
• Age >55 years • Leukocyte count >16 × 10^3/mcL • Blood glucose >200 mg/dL • Serum LDH >350 IU/L • Serum AST >250 IU/L	• Decrease in hematocrit >10% • Increase in BUN of >8 mg/dL • Serum calcium less than 8 mg/dL • PaO_2 < 60 mmHg • Base deficit >4 mEq/L • Estimated fluid sequestration >6000 mL
Score < 3 = mortality 0–3%, score < 3 = mortality 0–3%, score ≥ 6 = mortality 40%	

27.7.2 Nutritional Support

Traditionally, in patients presenting with acute pancreatitis, enteral feeding was limited to provide "pancreatic rest" as it was believed that enteral feeding stimulated the secretion of proteolytic pancreatic enzyme thus exacerbating the inflammatory process. However, on the other hand, limiting nutritional intake can have grave consequences. Inflammatory stress is a state of increased nutritional demand leading to catabolism and negative nitrogen balance.

Patients with mild acute pancreatitis generally need no or minimal nutritional support, as their disease typically resolves within 1 week. In contrast, patients with severe pancreatitis usually have a more prolonged disease course and should begin to receive nutritional support as early as feasible. Although these patients traditionally have been administered total parenteral nutrition (TPN), recent evidence suggests that enteral nutrition is safe, is less costly, and is associated with a lower complication rate as compared to parental nutrition. Administration of enteral nutrition supports the integrity of the intestinal mucosal barrier thus limiting or preventing bacterial translocation. A meta-analysis of eight randomized trials comparing enteral vs parenteral nutrition for acute pancreatitis revealed that enteral nutrition significantly reduced mortality, multiple organ failure, systemic infections, and the need for operative interventions compared to those who received TPN [10]. Traditionally, feeds have been delivered to the jejunum through nasojejunal tubes to avoid stimulating pancreatic exocrine secretion; however, recent studies have shown that continuous feedings through nasogastric tubes are equally safe and effective.

27.7.3 Endoscopic Retrograde Cholangiography (ERCP)

The benefit of early ERCP in acute pancreatitis has been studied extensively. Studies have shown that early ERCP with stone extraction and sphincterotomy clearly benefits the subset of patients with gallstone pancreatitis who present with cholangitis and biliary obstruction. Current recommendations for patients, with cholangitis, consist of performing ERCP urgently (within 24 h). The timing of ERCP in patients with biliary obstruction is not clear (24–72 h). It is reasonable to wait up to 48 h for biliary obstruction to resolve [11]. Magnetic Resonance Cholangiopancreaticography (MRCP) and endoscopic ultrasound (EUS) can be helpful to look for persistent choledocholithiasis in equivocal cases to prevent unnecessary intervention. In patients with no signs and symptoms of cholangitis or biliary obstruction, ERCP is associated with high complication rates and no apparent benefits; therefore, it is not recommended.

27.7.4 Cholecystectomy

The guidelines recommend that cholecystectomy should ideally be performed at the index admission, and should not be delayed by >2 weeks for patients with mild

acute gallstone pancreatitis who are good surgical candidates for the procedure. Laparoscopic Cholecystectomy should be the procedure of choice considering less postoperative pain and shorter hospital length of stay with comparable procedure-related morbidity and mortality compared to open procedure. The incidence of recurrent pancreatitis or associated gallstone complications during the 6-week period after an episode of gallstone pancreatitis is 18% in patients who do not undergo cholecystectomy [12]. Even the risk of recurrent pancreatitis is substantially high within 2 weeks after the initial episode of biliary pancreatitis who are discharged without cholecystectomy [13].

Therefore, current recommendations are to perform cholecystectomy in the same hospital admission once the acute phase of the episode has resolved which can be followed by analyzing the overall clinical condition, physical exam, and down-trending laboratory markers. This strategy does not increase operative complications, conversion to open procedures, or mortality [14].

27.7.5 Approach to Infected Necrosis

Surgery used to be the mainstay treatment for acute pancreatitis, this is no longer the case and surgical management has largely been replaced by more conservative and supportive care. However, surgery is still an integral component of treatment in patients with acute pancreatitis with gallstone pancreatitis and local complications.

27.7.6 Percutaneous Drainage

Invasive management is usually indicated in the presence of infection. Infected pancreatic necrosis/collection is suggested by clinical signs such as persistent fevers, leukocytosis, and radiological evidence of gas in peripancreatic collection. Drainage alone is the initially recommended intervention for infected pancreatic necrosis [15]. This is most often accomplished through a percutaneous image-guided approach, which is technically feasible in the vast majority of cases and is also the first step of step-up approach [16, 17]. The minimally invasive nature of this technique allows intervention even in the early phase of severe necrosis, as compared to an open approach which is associated with significantly higher mortality. It can be used as the primary treatment, as an adjunct to other techniques, or to reduce postoperative persistent fluid collections. The preferred approach for percutaneous drains is retroperitoneal so that the drain tract can later be used to perform video-assisted retroperitoneal debridement.

27.7.7 Video-Assisted Retroperitoneal Debridement (VARD)

VARD procedure is part of a “step-up” approach, it is the second-line therapy and usually follows percutaneous drainage when it fails to show an impact on clinical

condition of the patient. A small incision is made in the left flank in proximity to the previously placed percutaneous drain, which is used as a guide to accessing the retroperitoneum. The cavity is cleaned for better visualization using standard suction and irrigation. All necrotic tissue that is easily visualized is carefully removed until the deeper cavity is reached and further dissection and debridement cannot be performed under direct visualization. At this point, a long laparoscope trocar is placed from the incision followed by a zero-degree video scope. The cavity is then insufflated via the percutaneous drain. Under video scope guidance further debridement of retained necrotic tissue is performed. Complete necrosectomy is not the ultimate goal of this procedure, so only loose necrotic tissue is debrided thus keeping the risk of tearing of underlying blood vessels to a minimum. Once the debridement is completed, the percutaneous drain is removed and replaced with two large-bore drains. Irrigation is usually continued postoperatively through surgically placed drains. In a multicenter randomized controlled trial, patients with infected pancreatic necrosis were randomized to undergo primary open necrosectomy or a step-up approach consisting of percutaneous drainage followed by VARD. Open necrosectomy was rarely used only in cases where VARD could not be accomplished. Although there was no difference in mortality between the two groups, primary open necrosectomy was associated with a higher rate of major complications and increased cost [18].

27.7.8 Direct Endoscopic Nacrosectomy (DEN)

DEN is performed via transmural puncture into a necrotic collection with the help of an endoscope. The prerequisite to this technique is that the collection must be in close proximity or abutting either stomach or duodenum. With the help of transluminal endoscopic ultrasound, the collection is visualized and accessed using the FNA needle. The track into the collection is then dilated and large-bore stents are placed. Mechanical debridement can be performed by passing the endoscope via stent into the cavity and by using a snare, net, or a basket. Typically, multiple sessions are necessary to completely debride the cavity. The use of hydrogen peroxide has also been described in the literature and has been shown to decrease the number of sessions required to clean up the necrosis. This endoscopic approach can be used as a step-up approach replacing VARDS or as a primary treatment for walled-off necrosis [19].

27.7.9 Minimally Invasive Nacrosectomy

Minimally invasive necrosectomy was first described in 1996 by Ganger. Three minimally invasive approaches were initially described, retrogastric debridement, full retroperitoneal approach, and transgastric drainage with a success rate of about 75% [20]. Retrogastric technique is a preferred approach for acute necrotizing collection. Retrogastric debridement can be performed by either a transgastrocolic or

transmesocolic/infracolic approach. In a transgastrocolic approach, the gastrocolic ligament was opened to access the necrosed tissue. This approach is preferable for necrosis involving the head and body of the pancreas. In the transmesocolic or infracolic approach, the mesocolon was opened near the ligament of Treitz, between the middle colic artery and left colic artery. It is the preferred approach in necrosis involving the tail region of the pancreas. Necrotic tissue is dissected and removed using blunt dissection. The cavity is copiously irrigated with normal saline followed by placement of two large-bore surgical drains in the cavity. The transgastric approach is mostly used for walled-off pancreatic necrosis [21].

The minimally invasive approach is associated with less surgical trauma in these severely ill patients and there is substantial data suggesting a significant reduction in the incidence of new-onset organ failure compared to open approach.

27.7.10 Open Necrosectomy

Open necrosectomy used to be the standard approach for the treatment of pancreatic necrosis. Historically, early necrosectomy was recommended for all patients with necrotizing pancreatitis. However, its role has undergone resolution and is now only recommended for patients with infected necrosis not amenable to endoscopic and/or minimally invasive approaches or in centers with a lack of expertise in these techniques (Fig. 27.5).

Midline or bilateral subcostal (Chevron or Rooftop) incisions are used to access the abdomen. The lesser sac is approached by dividing the gastrocolic ligament. If the inflammatory reaction is intense and dissection planes are obliterated alternate route is opted by dividing the avascular portion of the transverse mesocolon. Loose nonviable necrotic tissue is removed by blunt dissection without performing anatomic resections. Special care should be taken to avoid injury to underlying vessels that are at high risk of rupture secondary to an intense inflammatory reaction. Any

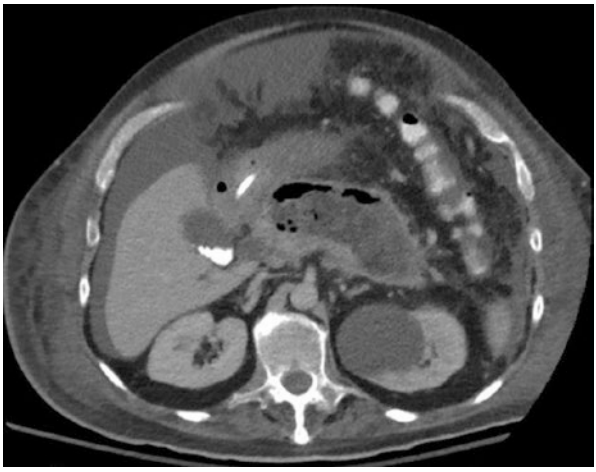

Fig. 27.5 Infected pancreatic necrosis (Courtesy of Dr. Latifi)

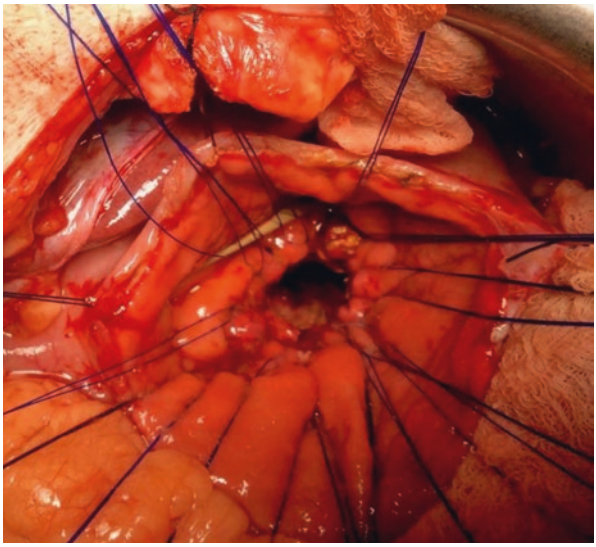

Fig. 27.6 Transgastric cystogastrosotomy (Courtesy of Dr. Latifi)

injury to surrounding major vessels including splenic, superior mesenteric vessels, or portal veins can result in massive hemorrhage that is difficult to control and can be fatal. Once the necrosectomy is performed, the cavity is lavaged with normal saline and surgical drains are left in place with the intent to continue irrigation via drains during the postoperative period [22].

Often times, the lesser sac cannot be entered at all, due to vascularization and inflammation. In this situation, the surgeon should select arriving at the necrotic tissue through anterior and posterior stomach. Care should be taken to stay in the midbody of the stomach. Anteriorly, the incision should be made longitudinally. The posterior opening of the stomach should be large as well, at least 5–6 cm, enough to allow gentle but complete evacuation of a dark clay-like necrotic material. One has to be careful and gentle during this process. The posterior stomach wall and pseudocyst wall are sutured with interrupted, slow absorbing sutures (Fig. 27.6).

The procedure has been modified in an attempt to achieve optimal results. Sometimes the abdomen is left open with packing and planned staged re-laparotomies for subsequent debridements and packing changes to ensure the adequacy of debridement. In other modifications, the drains are used for continuous lavage of the cavity to minimize stasis and risk of infection. Open necrosectomies for acute infected pancreatic necrosis are considered as a last tier therapy due to high morbidity, mortality, and debilitating complications including enteric-cutaneous and pancreatic-cutaneous fistulas [17].

27.7.11 Pseudocyst and Walled-off Necrosis

About 30–50% of patients developed acute collections with acute pancreatitis. Without evidence of any infection, most of these collections remain asymptomatic

and spontaneously resolve without any intervention. However, based on size and location they can cause upper abdominal pain, gastric outlet obstruction, or obstructive jaundice. Pseudocysts are typically round, unilocular, and have a dense wall. In contrast, walled-off necrosis is typically heterogeneous with liquid and non-liquid density, with varying degrees of loculations, and is encapsulated by a well-defined wall. Previously, it was believed that walled off collections that existed beyond 6 weeks rarely resolve and are associated with high complication rates; hence, it was recommended that all walled off collections (pseudocyst/walled-off necrosis) that persist beyond a period of 6 weeks should undergo drainage. This concept has now evolved and the natural history of asymptomatic walled off collections follows a benign course, especially those with less than 6 cm diameter that have a high tendency for spontaneous resolution. Currently, indications of surgical drainage in the absence of any infection are persistent symptoms, i.e., intractable pain, gastric outlet obstruction, and obstructive jaundice. However, any intervention of a symptomatic patient without evidence of infection should be delayed 4–6 weeks to provide adequate time for the maturation of the cyst wall.

Multiple treatment options are available including percutaneous drainage and internal drainage either trans-abdominally or endoscopically. Percutaneous drainage that is obsolete is avoided due to high failure rates especially in patients with ductal abnormalities [23]. The endoscopic approach is feasible in patients when the collection is in close proximity to the stomach or duodenum. (Refer to DEN for description) [19]. A small single-center study has shown equal efficacy of open vs endoscopic cystogastrostomy for pseudocyst with advantages of no general anesthesia, shorter hospital length of stay, and decreased cost for patients undergoing an endoscopic approach [24]. However, for walled-off necrosis due to the presence of a solid component endoscopic approach, a multistage procedure in order to achieve complete evacuation of debris is required. No high-quality data exists to show any advantages if any of the endoscopic approaches compared to the transabdominal approach.

Transabdominal procedures for pseudocyst or walled-off necrosis include cystogastrostomy, cystoduodensotomy, and Roux-en-Y cystojujenostomy. These can be performed open, laparoscopically, or robotically depending upon the feasibility, complexity of the collection, and expertise of the surgeon. The internal drainage of the cyst is achieved by creating communication between the cyst wall and the gastrointestinal tract. For transgastric cystogastrostomy, an anterior longitudinal gastrostomy is made to enter and palpate the lumen posterior gastric wall. The location of the cyst wall can be confirmed using an intraoperative ultrasound or by needle aspiration which also determines the distance of the cyst wall to the posterior gastric wall. The cyst is entered by incising the posterior gastric wall. Intraoperative ultrasounds can be used to evaluate the complete extent of the cavity. Necrotic tissue within the cyst cavity is evacuated. Cyst cavity s explored for any loculations and are lysed. A generous cystogastrostomy (Fig. 27.6) is done 5 cm or more ensuring a wide patent connection between the cyst and stomach wall, and finally, the anterior gastrostomy site is closed. Extra gastric cystogastrostomy can also be performed by approaching the lesser sac and identifying the spot where the posterior gastric wall

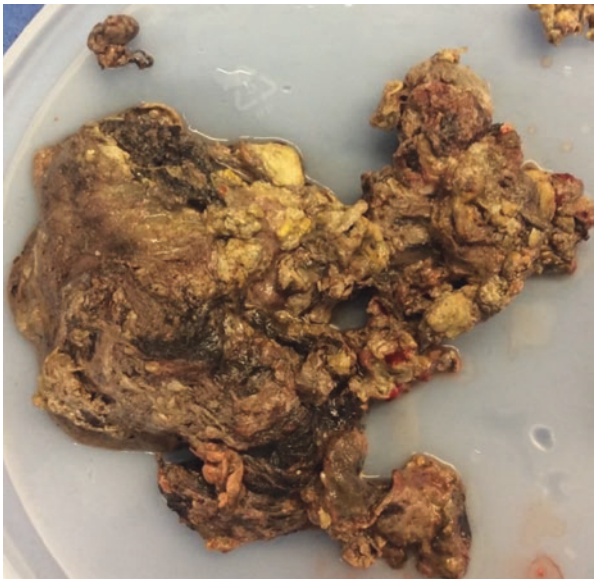

Fig. 27.7 Pancreatic necrosum (Courtesy of Dr. Latifi)

and pseudocyst wall lie in close proximity. Cystogastrostomy can be performed between the posterior gastric wall and anterior cyst wall either by stapler or in a hand-sewn fashion (Fig. 27.7).

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