Updates in the Management of Acute Pancreatitis

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Key Points

- Definition of acute pancreatitis (AP) is based on the fulfillment of "two out of three" of the following criteria: clinical (upper abdominal pain), laboratory (serum amylase or lipase >3 upper limit of normal), and/or imaging.
- There are three grades of severity of acute pancreatitis, namely, the mild, the moderately severe, and the severe.
- Systemic inflammatory response syndrome (SIRS) is advised to predict severe acute pancreatitis at admission, while systemic organ dysfunction is a main determinant of clinical outcome.
- Local complications of acute pancreatitis include acute peripancreatic fluid collections, pancreatic pseudocysts, acute necrotic collections, and walled-off necrosis. Each collection may be either sterile or infected.
- Initial management of acute pancreatitis consists of supportive care with fluid resuscitation, pain control, and nutritional support.
- Invasive intervention should be delayed whenever possible until at least 4 weeks after initial presentation to allow the collection to become "walled-off."
- Primarily, image-guided percutaneous catheter drainage or endoscopic transluminal drainage, followed, if necessary, by endoscopic or surgical necrosectomy represent the optimal interventional strategy for patients with suspected or confirmed infected necrosis.
- Late, well-organized, and predominantly fluid collections are usually managed by endoscopic or laparoscopic transgastric drainage.
- Early ERCP is no longer recommended in AP, regardless of disease severity, in the absence of coexisting biliary sepsis.

30.1 General Description

Acute pancreatitis (AP) is an acute inflammatory process of the pancreas. Although its pathogenesis is still not completely understood, several conditions are known to induce AP, with gallstones and chronic alcohol abuse accounting for two-thirds or more cases in the United States [1], where AP is the leading gastrointestinal cause of hospitalization [2].

For most of the cases, AP is a self-limiting illness. In uncomplicated attacks, management involves appropriate supportive care until resolution, followed by treatment of the precipitating cause, reducing the chance of a future attack. For a minority of patients, AP is a life-threatening condition that results in prolonged hospital admission and significant mortality, requiring early recognition of the high-risk group and multidisciplinary management of complications in centers of high expertise.

Mortality ranges from 3% in patients with interstitial edematous pancreatitis to 17% in patients who develop pancreatic necrosis [3, 4]. Mortality within the first 2 weeks (early death) is usually due to systemic inflammatory response syndrome (SIRS) and organ failure, while after this time frame, it is usually due to local pancreatic complications related to pancreatic necrosis and sepsis [5, 6].

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Early triage, improvements in intensive care management, nutritional support, and timely and appropriate intervention for local complications with less invasive approaches at high-volume centers have led to the drop of the overall mortality during the last decades.

30.2 Clinical Presentation, Diagnosis, and Severity Assessment

Patients with AP present with persistent, severe, epigastric abdominal pain. The pain is often radiating to the back, associated with nausea and vomiting, and may persist for several hours.

In patients with hereditary, metabolic, or alcoholic AP, the onset of pain may be less abrupt, and the pain itself may be poorly localized than in patients with gallstone pancreatitis.

The degree of systemic disturbance is variable, as well as the physical findings of severe cases, such as abdominal tenderness, obstructive jaundice (due to choledocholithiasis or edema of the pancreatic head), and ecchymotic discoloration due to retroperitoneal bleeding.

Per the 2012 revision of the Atlanta classification, the definition of acute pancreatitis (AP) is based on the fulfillment of "two out of three" of the following criteria (regardless of etiology): clinical (upper abdominal pain), laboratory (serum amylase or lipase >3 upper limit of normal), and/or imaging (computed tomography (CT), magnetic resonance (MR), ultrasonography (US)) criteria [7].

Cross-sectional imaging may be useful but not strictly necessary to diagnose acute pancreatitis. Exceptions include a prolonged period between onset of symptoms and presentation (lipase and amylase may have normalized), unconscious patients, or suspicion of duodenal perforation.

In general, AP can be subdivided into two types (Table 30.1):

This represents a radiologic and pathologic classification, to be distinguished from a clinical assessment of severity. Defining the severity of AP is crucial to stratify patients into subgroups, based on the presence of transient or persistent organ failure and local or systemic complications, allowing for appropriate early triage to intensive care units (ICUs) and selection of patients for specific interventions.

The revised Atlanta classification [7] defines three grades of severity of pancreatitis (Table 30.2):

Local complications of acute pancreatitis include peripancreatic fluid collections, pancreatic pseudocysts, acute necrotic collections, and walled-off necrosis [7]. Organ failure (OF) is defined as a score of two or more for any one of three organ systems (respiratory, cardiovascular, or renal) using the modified Marshall scoring system [8].

Table 30.1 Radiologic/pathologic classification of AP

Interstitial edematous pancreatitis	Necrotizing pancreatitis
Acute inflammation of the pancreatic parenchyma and peripancreatic tissues, without recognizable tissue necrosis	Inflammation associated with pancreatic or peripancreatic necrosis

Table 30.2 Grades of severity of AP according to the revised Atlanta classification [7]

Mild AP	Moderately severe AP	Severe AP
Absence of organ failure or local complications	Presence of transient organ failure (resolving within 48 h) or local complications developing in the absence of organ failure	Presence of persistent organ failure (>48 h) with or without local complications

Table 30.3 Definition of SIRS [12]; two or more of the criteria should be present

SIRS
• Temperature <36 °C or >38 °C
• Heart rate >90/min
• Respiratory rate >20/min
• White blood cells (<4 × 10^9/L, >12 × 10^9/L) or 10% bands

Many predictive models (e.g., APACHEII, Ranson, and modified Glasgow score) have been developed to predict the clinical outcome of AP during the early hours after hospital admission, based on clinical, laboratory, and radiologic risk factors [9].

Per the IAP/APA guidelines [10], systemic inflammatory response syndrome (SIRS) is advised to predict severe AP at admission and persistent SIRS at 48 h; identifying patients at risk of developing systemic organ dysfunction represents a main determinant of clinical outcome [11].

SIRS is defined by the presence of two or more of the following four criteria (Table 30.3):

Other scores can be used only 24–48 h after the disease onset and have not been shown to be consistently superior to the assessment of SIRS or the APACHE II score.

30.3 Management of Acute Pancreatitis

30.3.1 Management of the Early Phase

The initial evaluation of AP should comprehend a clinical examination to assess for early fluid losses, organ failure (cardiovascular, respiratory, or renal compromise), and severity, with the measurement of the SIRS score and APACHE II score. Patients with severe AP should be admitted to an intensive care unit (ICU) for optimal support.

Amylase and lipase value is useful for the diagnosis, but serial measurements in patients with acknowledged AP are not useful to predict severity, prognosis, or following management.

Since the extent of pancreatic and peripancreatic necrosis may only become apparent 72 h after the onset of AP,

cross-sectional imaging (e.g., abdominal computed tomography (CT) scan) is not recommended at initial presentation if the diagnosis is already established.

Initial management of AP consists of supportive care with fluid resuscitation, pain control, and nutritional support.

30.3.1.1 Fluid Resuscitation

In the initial stages (within the first 12–24 h) of acute pancreatitis, fluid replacement has been associated with a reduction in morbidity and mortality [13–15] with decreased rates of persistent SIRS and organ failure.

Current guidelines [10] recommend Ringer's lactate for initial fluid resuscitation in acute pancreatitis; a multicenter RCT demonstrated that resuscitation with Ringer's lactate decreased the incidence of SIRS when compared to resuscitation with normal saline in 40 patients with acute pancreatitis [16]. A fluid infusion rate of 5–10 mL/kg/h is recommended, until resuscitation goals are reached. A total infusion of 2500–4000 mL in the first 24 h will likely suit, as an overly aggressive fluid therapy was demonstrated to increase morbidity and mortality [17, 18]. Resuscitation goals should be guided by restoration of physiologic homeostasis using markers such as pressure and flow parameters, urine output, lactate, mixed venous oxygen saturation, and base deficit.

30.3.1.2 Pain Control

Pain is a predominant symptom in AP and should be treated with analgesics according to its duration and severity. A review by the Cochrane has examined five randomized trials comparing different analgesics in AP, founding no evidence of increased complications related to opioid use [19], scaling down the historical concern about their potential (for morphine in particular) to induce sphincter of Oddi spasm and exacerbate the severity of AP. Opioids were instead associated with a reduction in the need for supplementary analgesia, and therefore remains the treatment of choice for the vast majority of patients.

Protracted, severe pain may require administration of opioids through a specific patient-controlled analgesia (PCA) pump.

30.3.1.3 Nutritional Support

Oral feeding in predicted mild pancreatitis can be restarted once abdominal pain is decreasing and inflammatory markers are significantly improving. Those patients can often be managed with intravenous hydration alone since recovery occurs rapidly (usually within 1 week).

Enteral tube feeding, either via naso-jejunal or nasogastric route [20, 21], should be the primary therapy in patients with predicted severe acute pancreatitis requiring nutritional support; two meta-analyses demonstrated enteral nutrition's superiority in decreasing systemic infections, multi-organ failure, need for surgical intervention, and mortality when compared with parenteral nutrition [22, 23]. The reasons for such superiority might be explained by a reduction of bacterial translocation and consequent infection of pancreatic necrosis, as enteral feeding contributes to gut barrier function and the higher rate of complications associated with the parenteral route, including line sepsis. Moreover, enteral feeding is also less expensive than parenteral nutrition. Parenteral nutrition can be administered in acute pancreatitis as a second-line therapy if naso-jejunal tube feeding is not tolerated and nutritional support is further required.

30.3.2 Management of Systemic Complications

30.3.2.1 Antibiotics

A secondary infection of pancreatic necrosis appears in about the 40% of patients with necrotizing AP, leading to a second peak in mortality between 2 and 4 weeks after the disease onset. However, international guidelines do not recommend intravenous antibiotic prophylaxis for the prevention of infectious complications in acute pancreatitis [10]. A Cochrane review and a meta-analysis of 14 RCTs showed no benefits from antibiotic prophilaxis [24, 25]. Probiotic prophylaxis and selective gut decontamination are not currently recommended for the prevention of infectious complications.

Intravenous antibiotics should be given in case of suspected infection of necrotizing pancreatitis or extrapancreatic infections; when an infection is suspected, antibiotics should be started while the source of the infection is being determined.

30.3.2.2 Organ Failure

The most common systemic complication in AP is single or multiple organ failure (OF). Regardless of its etiology, OF often requires transfer to the ICU and could be represented by respiratory, cardiovascular, renal, and intestinal severe dysfunction. Noninvasive respiratory support or mechanical ventilation, volume resuscitation, and vasoactive agents with invasive monitoring and dialysis could be required.

Gastrointestinal failure manifests as nausea, vomiting, and abdominal distension and occurs because of reduced perfusion, with a failure to tolerate enteral nutrition and the breakdown of the gut barrier function, with bacterial translocation leading eventually to infected pancreatic necrosis.

30.3.2.3 Abdominal Compartment Syndrome

Abdominal compartment syndrome (ACS) is defined as a sustained intraabdominal pressure greater than 20 mmHg (with or without abdominal arterial perfusion pressure <60 mmHg) that is associated with new-onset OF [26].

According to the available international guidelines, measurement of intra-abdominal pressure via the bladder should be considered in patients with severe acute pancreatitis associated with mechanical ventilation, especially in case of clinical deterioration, as intra-abdominal hypertension (IAH) contributes to the development of OF.

The first approach for the treatment should be medical, targeting the main contributors to IAH like hollow viscera volume (with nasogastric drainage, prokinetics, rectal tubes, and endoscopic decompression, if necessary), intravascular and extravascular volume status, and abdominal wall compliance (analgesia, sedation, and neuromuscular blockade if necessary).

Due to the risk of infecting a previously sterile pancreatic necrosis and the significant fluid losses that may occur in the open abdomen, invasive decompression should only be used after a multidisciplinary discussion in patients with a sustained intra-abdominal pressure >25 mmHg with a new onset of organ failure refractory to medical therapy. Invasive treatment options include percutaneous catheter drainage of ascites, midline laparostomy, bilateral subcostal laparostomy, or subcutaneous midline fasciotomy.

30.3.3 Management of Local Complications

The revised Atlanta classification [7] divides AP into three discrete grades of severity (mild, moderately severe, and severe) based on the absence or presence of systemic and/or local complications.

Furthermore, local complications are categorized based on the time from presentation (< or >4 weeks) and on the presence of necrosis (Table 30.4). Each collection may be in addiction, sterile, or infected.

Acute, non-necrotic fluid collections develop during the first days of AP, usually without a defined wall, and remain asymptomatic. They resolve spontaneously without the need for drainage, as only less than 10% of acute fluid collections persist beyond 4 weeks as pancreatic pseudocysts [27]. Failure of resolution could be related to the presence of necrosis or main pancreatic duct (MPD) interruption.

Intervention for an acute necrotic collection in the first weeks has a high risk of mortality and morbidity [28]. Invasive intervention (i.e., percutaneous catheter drainage, endoscopic transluminal drainage/necrosectomy, minimally invasive or open necrosectomy) should be delayed whenever possible until at least 4 weeks after initial presentation to allow the collection to become "walled-off," following a philosophy expressed by the "3D" concept: delay, drain, and debride.

Common indications for intervention in necrotizing AP, according to the IAP guidelines [10], are indicated in Table 30.5:

Pancreatic infection is a leading cause of morbidity and mortality in acute necrotizing pancreatitis, affecting one-third of patients with pancreatic necrosis [29], often later in the clinical course (10 days) [30, 31].

The presence of gas in peripancreatic collections at CT is considered as evidence of infected necrosis, irrespective of the source of the gas [32]. Fine needle aspiration of collections to detect bacteria is not routinely indicated, because of the high risk of false-negative results [33] and lack of evidence assessing the possible benefits in shortening the period to diagnosis of infection and tailoring the antibiotic treatment.

Traditionally, primary open necrosectomy has been the treatment of choice in infected necrotizing AP. Nowadays, there is consensus in the current literature toward a principle of early organ and nutritional support, followed ideally by

Table 30.4 Local complications in AP according to 2012 revised Atlanta classification [7]

Time	Necrosis −	Necrosis +
<4 weeks	*Acute peripancreatic fluid collection* (Peripancreatic fluid associated with interstitial edematous pancreatitis with no associated peripancreatic necrosis)	*Acute necrotic collection* (A collection containing variable amounts of both fluid and necrosis; the necrosis can involve the pancreatic parenchyma and/or the extrapancreatic tissues)
>4 weeks	*Pancreatic pseudocyst* (An encapsulated collection of fluid with a well-defined inflammatory wall usually outside the pancreas with minimal or no necrosis)	*Walled-off necrosis* (A mature, encapsulated collection of pancreatic or extrapancreatic necrosis that has developed a well-defined inflammatory wall)
Infection	Each collection type may be sterile or infected	

Table 30.5 Indications for intervention in necrotizing AP according to the IAP/APA guidelines [10]

Common indications	Uncommon indications
• Clinically suspected/documented infected necrosis with clinical deterioration (preferably when walled-off/>4 weeks) • In the absence of documented infected necrosis, ongoing organ failure for several weeks after the onset of AP (preferably when walled-off/>4 weeks)	• Abdominal compartment syndrome • Ongoing acute bleeding • Bowel ischemia • Ongoing gastric outlet, intestinal, or biliary obstruction due to mass effect from large walled-off necrosis (>4–8 weeks) • Persistent symptoms (pain, "unwellness") in patient with walled-off necrosis without signs of infection (>8 weeks) • Disconnected duct syndrome (>8 weeks)

delayed minimally invasive intervention within a step-up approach, whenever possible.

A fluid collection with minimal or no necrotic component that became persistent ("true" pancreatic pseudocysts) is very uncommon. The great majority of persistent pancreatic collections, especially when requiring intervention, has at least a small amount of necrosis with varying degrees of fluid content.

Indication for sterile, necrotizing AP is gastric, intestinal, or biliary obstruction due to mass effect or persistent symptoms (e.g., pain, "persistent unwellness") in patients without signs of infection, arbitrarily >4–8 weeks after onset of acute pancreatitis.

Furthermore, a disconnected duct syndrome (i.e., full transection of the pancreatic duct in the presence of pancreatic necrosis) seems to be very common in patients during the follow-up after necrotizing pancreatitis, most of them requiring an intervention later than 8 weeks after the acute attack [34].

Conventional management of late pancreatic collections was by open pancreatic cystogastrostomy, but with the development in interventional radiology, therapeutic endoscopy, and minimal access surgery, new minimally invasive techniques have been employed as alternatives to the traditional approach. Late, well-organized, and predominantly fluid collections are usually managed by endoscopic or laparoscopic transgastric drainage. There is a consistent degree of overlap between the early and late patient populations, most studies in the literature including heterogeneous groups.

30.3.3.1 Percutaneous, Endoscopic, and Surgical Step-Up Approach

Primarily, image-guided percutaneous (retroperitoneal) catheter drainage (Fig. 30.1) or endoscopic transluminal drainage (Fig. 30.2), followed, if necessary, by endoscopic or surgical necrosectomy, represents the optimal interventional strategy for patients with suspected or confirmed infected necrosis.

The PANTER (PAncreatitis, Necrosectomy versus sTEp-up appRoach) trial from the Dutch Pancreatitis Study Group, a multicenter RCT accounting 88 patients with (suspected) infected necrotizing pancreatitis, demonstrated how this approach decreased death or major complications, as well as costs, when compared to primary open necrosectomy [35].

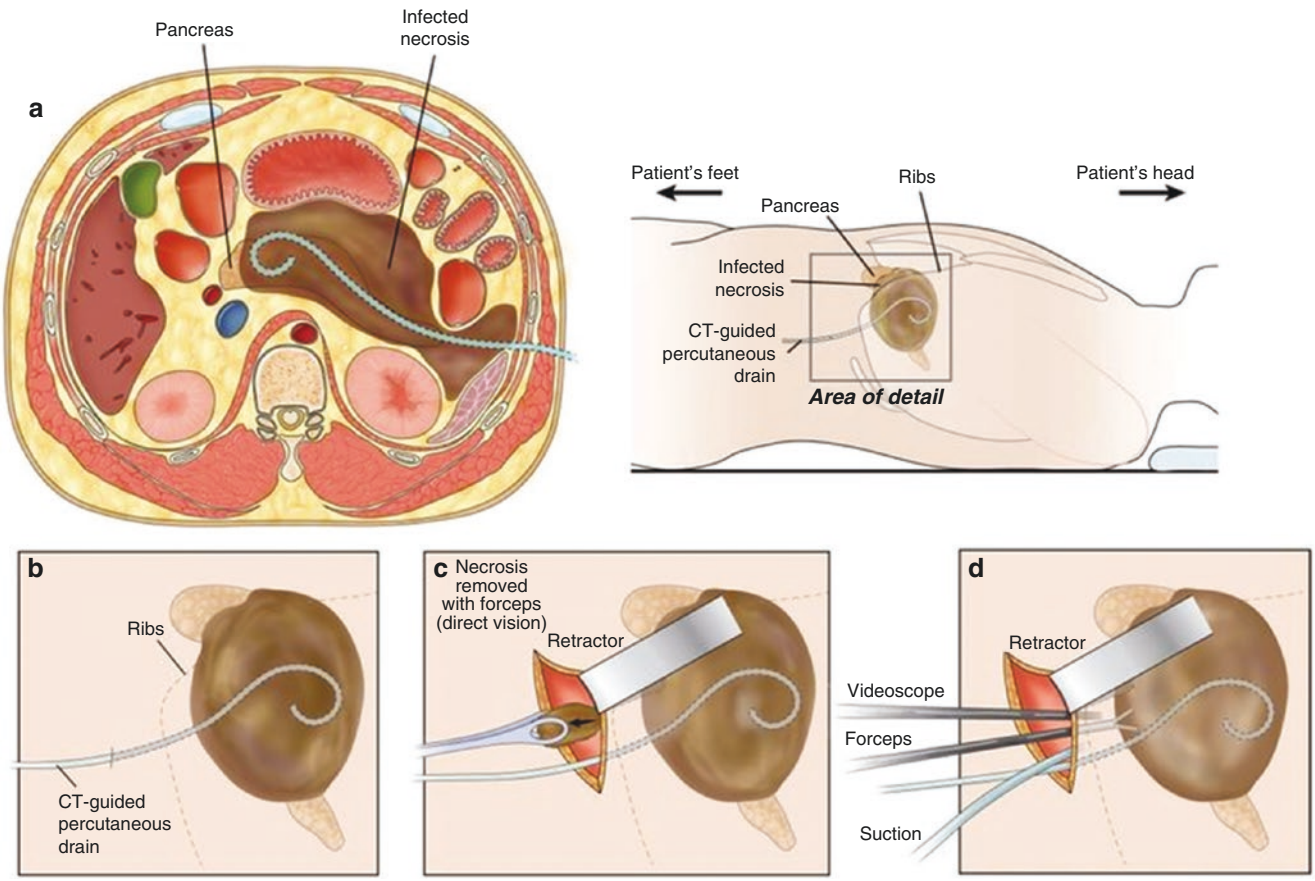

Fig. 30.1 Surgical step-up approach. (**a**) Peripancreatic collection drained by a percutaneous catheter inserted through the left retroperitoneal space. (**b**) Detail of CT-guided percutaneous drain. (**c**) First necrosis removal under direct vision, following the percutaneous drain. (**d**) Video-assisted retroperitoneal debridement (VARD). *Courtesy of S van Brunschot* et al., *Dutch Pancreatitis Study Group* [37]

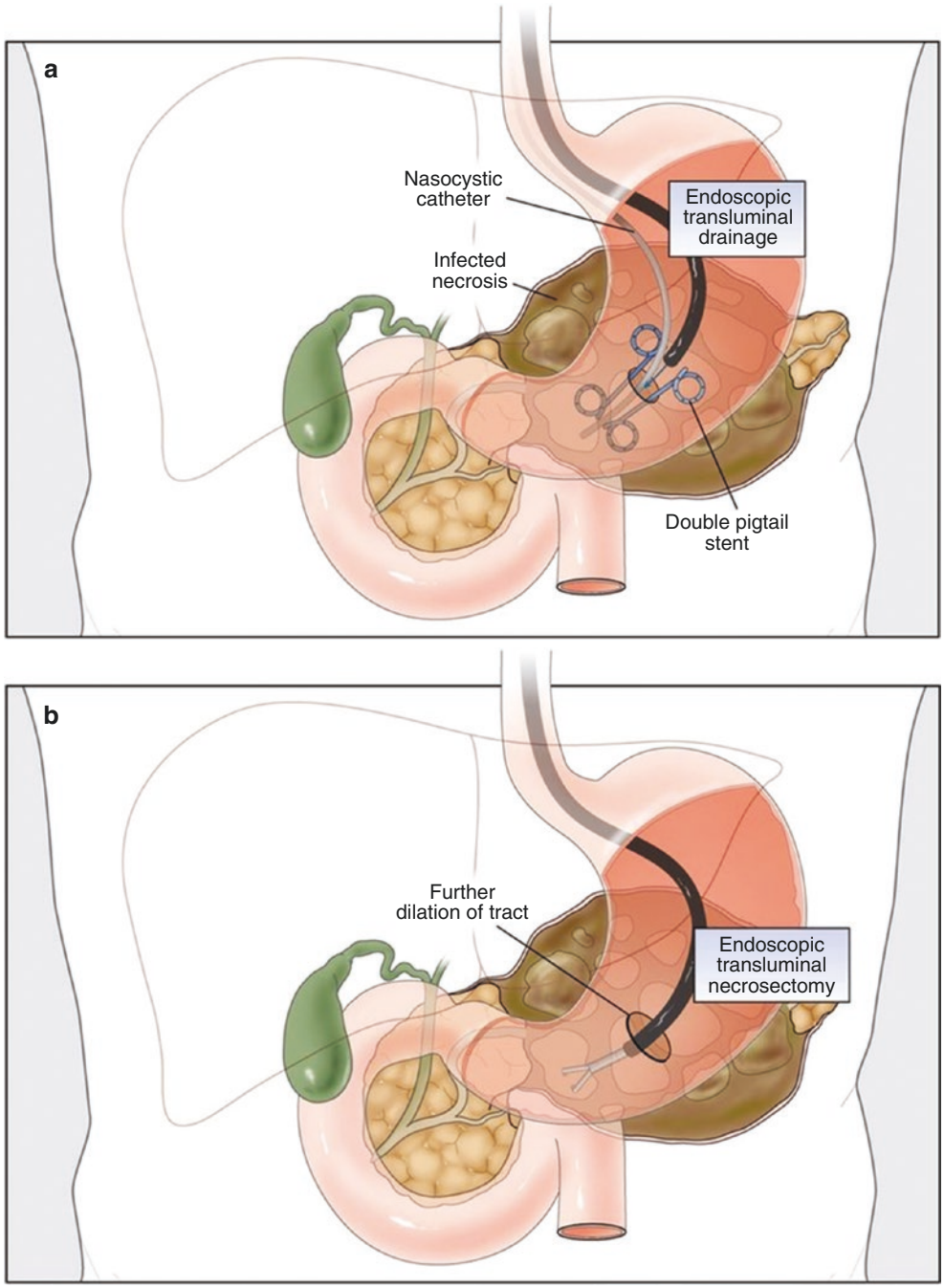

Fig. 30.2 Endoscopic step-up approach. (**a**) Endoscopic transluminal drainage (ETD) followed by (**b**) endoscopic transluminal necrosectomy (ETN) *Courtesy of S van Brunschot* et al., *Dutch Pancreatitis Study Group* [37]

Indeed, 35% of patients in the trial were successfully managed with percutaneous drainage alone and did not require subsequent debridement. Left retroperitoneal catheter drainage can facilitate minimally invasive retroperitoneal necrosectomy. After placement, the catheters undergo vigorous manual irrigation with isotonic saline every several days, creating a "closed circuit" of lavages, combined with contrast CT scans to follow the necrotic debridement and, if necessary, the placement of progressively larger-bore catheters (up to 24–28 French).

Even if percutaneous catheter drainage seems to be technically feasible in >95% of patients with infected necrosis [35], a promising alternative gaining worldwide popularity is natural orifice transluminal endoscopic surgery (NOTES) for drainage and/or necrosectomy purpose. Endoscopic cystogastrostomy was initially reported for the management of a mature pancreatic abscess with minimal necrosis, but the technique has evolved in the past 10 years, extending the indication also for debridement of necrosis.

First, the collection is punctured through the gastric wall, followed by balloon dilatation of the tract. Two double-pigtail stents and a nasocystic catheter for continuous postoperative irrigation are usually placed (endoscopic transluminal

drainage). Later, the cystostomy tract is dilated, the collection is entered with an endoscope, and necrosectomy is performed (endoscopic transluminal necrosectomy).

Several trials are testing NOTES efficacy compared to minimally invasive intervention (the early pilot PENGUIN [36] trial and the ongoing TENSION [37] trial from the Dutch Study Group) showing at least an equivalence between the two procedures.

The choice of initial percutaneous or endoscopic drainage is largely based on the position of the collection, with some authors suggesting to approach lateral collections and those extending behind the colon from the left or right flank and to prefer endoscopic drainage for those medial collections where a percutaneous route is compromised by overlying the bowel, spleen, or liver.

As discussed before, transgastric EUS-guided drainage is also the treatment of choice for late, persistent pancreatic collections, especially when mainly fluid. If catheter or endoscopic drainage fails, the optimal method of necrosectomy is still debated [38]. Minimally invasive necrosectomy seems to be associated with a decreased risk of complications and death as compared to open necrosectomy [39]. Several minimally invasive approaches have been described for surgical necrosectomy, including percutaneous minimally invasive retroperitoneal (MIRP) [40] necrosectomy, video-assisted retroperitoneal debridement (VARD) [41], and laparoscopic cystogastrostomy [42].

30.3.3.2 Video-Assisted Retroperitoneal Debridement (VARD)

VARD [41] is a drain-guided, minimally invasive retroperitoneal procedure requiring a 5 cm flank incision (Fig. 30.1). Using the retroperitoneal drain for guidance, the collection is entered, and loosely adherent necrosis is removed under video assistance (using a 0° laparoscope). The cavity is cleared of the infected/purulent material using a standard suction device. At the end of the procedure, two large-bore surgical drains are inserted, and a continuous postoperative lavage system is installed.

30.3.3.3 Laparoscopic Cystogastrostomy

In large, organized, solid, predominant post-acute collections, EUS-guided transgastric approach is at risk of inadequate drainage, with the necessity of repeated interventions. The improvements of laparoscopic operative technique and equipment have refocused interest on the potential of a single laparoscopic intervention. The current technique has evolved from the intragastric approach to a true laparoscopic transgastric approach [42], which requires an anterior gastrotomy for access and cystogastrostomy creation through the posterior gastric wall.

30.3.3.4 Laparoscopic Open Necrosectomy

Open necrosectomy is currently practiced and remains popular even with the increasing experience in minimally invasive approaches, especially in case of failure of those first-line treatments, extended infected necrosis (>50% of the pancreas), or severe acute complications (intestinal perforation, ACS, intracavitary bleeding).

The abdomen is entered though a midline or a bilateral subcostal incision preferred for minimizing contamination of the lower abdomen and allowing lateral access. The pancreas is exposed by dividing the gastrocolic ligament or gastrohepatic omentum by accessing into the lesser sac, allowing for manual necrosectomy.

In terms of necrosectomy, all described procedures are generally similar, being the main differences in terms of how they prevent recurrence of an infected collection within the debridement cavity.

In classical open necrosectomy approach with open packing, as described in 1987 by Bradley [43], the abdomen remains open following the debridement, and the cavity is packed with a laparostomy, with drains in place, allowing for resolution with healing by secondary intention. This open packing technique has been reported to have higher incidence of incisional hernias, bleeding, fistulae, and mortality rates [44].

For these reasons, the concept has been re-elaborated to mimic the step-up approach, with a first intervention aimed to a more conservative debridement, followed by planned reinterventions with sequential pack changes, in response to the bleeding and fistulation that can arise following aggressive necrosectomy. The pancreatic bed is drained or packed, and the abdomen is closed by suturing mesh or a zipper to the fascial edges of the wound [45].

Open necrosectomy could also be followed by closed packing, where a thorough debridement and infected tissue removal is followed by a primary closure of the abdomen, with the goal to achieve sepsis control and minimize the need for reoperation or subsequent drainage [46, 47]. Silicone drains (Jackson-Pratt) may be placed and removed sequentially. After debridement, reconstruction of a closed peripancreatic compartment is also possible, by suturing the gastrocolic and duodenocolic ligaments over large-bore drains, allowing side to side continuous lavage [48].

30.3.4 Management of Underlying Conditions Predisposing to Acute Pancreatitis

30.3.4.1 ERCP and Cholecystectomy

Controversy still exists regarding the role and timing of endoscopic retrograde cholangiopancreatography (ERCP) in gallstone pancreatitis. A recent meta-analysis of 7 RCTs including 757 patients found no evidence of benefits of early

routine ERCP in the attempt of avoiding mortality or local/systemic complications, regardless of the predicted severity of biliary AP [49]. Therefore, early ERCP is no longer recommended in acute pancreatitis, regardless of disease severity, in the absence of coexisting biliary sepsis/cholangitis.

For patients with evidence of biliary obstruction and AP, without signs of cholangitis, evidence is still lacking. Early ERCP remains recommended in the presence of jaundice and SIRS, while patients with jaundice without SIRS can be managed with observation for 24–48 h. ERCP is therefore reserved for those cases in which bilirubin levels arise after the acute attack or persist as elevated, as in most of the cases, a spontaneous passage of stones causes only a temporary jaundice. This last group of patients could benefit of MRCP or EUS to assess the presence or absence of persistent ductal stones.

Cholecystectomy should be delayed in patients with peripancreatic collections until the collections either resolve or if they persist beyond 6 weeks, a timeline after which cholecystectomy can be safely performed [50].

On the other hand, cholecystectomy during index admission for mild biliary pancreatitis appears safe and is recommended over interval cholecystectomy, as demonstrated by a recent RCT [51], reducing the rate of recurrent gallstone-related complications, with a very low risk of cholecystectomy-related complications.

Case Scenario

A 54-year-old male patient admitted to the ICU for severe biliary AP with no clinical signs of sepsis was treated with initial fluid resuscitation and enteral nutrition.

After initial stabilization, the patient was then transferred to the gastroenterology unit and underwent ERCP with sphincterotomy. A CT scan performed 3 weeks after the presentation showed extended (>50% of the pancreatic gland) walled-off sterile necrosis (Fig. 30.3). The patient was at that time asymptomatic, and oral food intake was administered.

1. What would be the preferred treatment?
   A. Follow-up with CT scan at 7–10 days
   B. Percutaneous drainage
   C. Endoscopic cystogastrostomy
   D. Open necrosectomy

Four days later, the patient presented clinical and laboratory signs of sepsis. An urgent CT scan was repeated (Fig. 30.4).

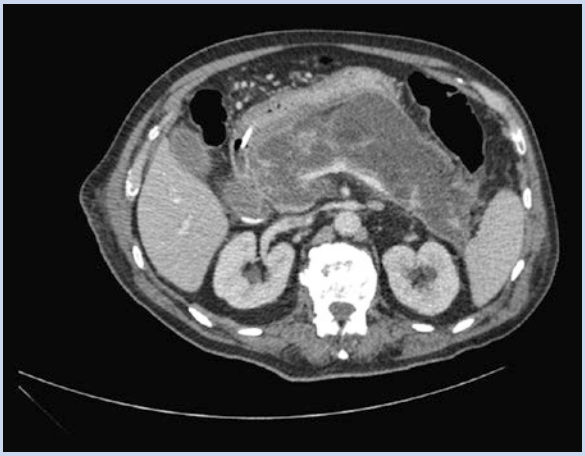

Fig. 30.3 Third week CT scan

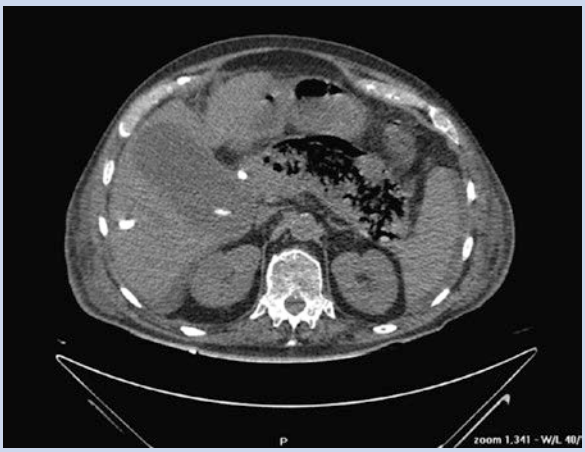

Fig. 30.4 CT scan performed while the patient presented with sepsis showed gas in peripancreatic collections

2. What would be the preferred treatment?
   A. Conservative treatment with parenteral antibiotics
   B. Percutaneous drainage
   C. Laparoscopic cystogastrostomy
   D. Open necrosectomy

The infected necrotic collection was drained via a percutaneous drainage (20F) (Fig. 30.5).

Cultural examination of the necrotic output permitted a targeted antibiotic treatment.

The patients developed persistent sepsis associated with hemodynamic instability despite the previously described treatment.

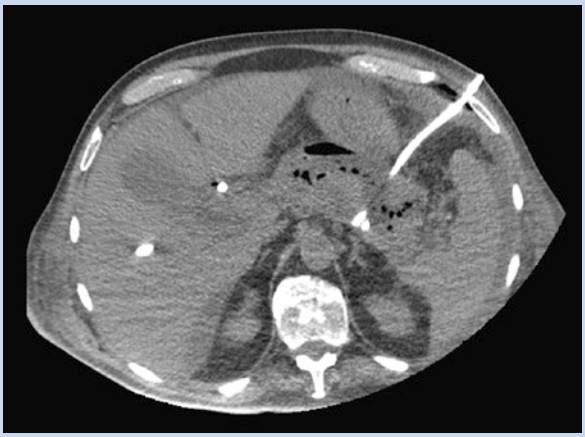

Fig. 30.5 Percutaneous 20F drainage with a left-side access

3. What would be the next step of intervention (extended pancreatic infected necrosis, sepsis)?
   A. Change of antibiotic therapy
   B. Percutaneous drainage with larger-bore drain
   C. Endoscopic transluminal drainage and necrosectomy
   D. Open necrosectomy

Please see Chap. 58 for the correct answer.

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