Necrotizing Pancreatitis: Best Approaches

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Introduction

In the United States approximately 290,000 patients develop acute pancreatitis annually. More than 80% of cases of acute pancreatitis are due to either alcohol consumption or gallstone disease. More uncommon causes include metabolic disorders, trauma, tumors, and iatrogenic injuries (ERCP, surgery). The severity of acute pancreatitis ranges from edema to necrosis of the gland. The edematous form of the disease (mild acute pancreatitis) occurs in about 80–85% of patients and is self-limited, with recovery in a few days. In the 15–20% of patients with the most severe form of pancreatitis, hospitalization is prolonged, and commonly associated with the systemic inflammatory response syndrome (SIRS), multi-organ failure, and infection of the pancreatic necrosis. In these patients, mortality can be as high as 20% [1, 2].

Case Presentation

A 68-year-old-male with a history of coronary artery disease, atrial fibrillation, and diabetes mellitus, is transferred to a tertiary referral center 2 weeks into a course of severe necrotizing pancreatitis due to hypertriglyceridemia. On presentation, the

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T.M. Pawlik et al. (eds.), *Case-Based Lessons in the Management of Complex Hepato-Pancreato-Biliary Surgery*, DOI 10.1007/978-3-319-50868-9_33

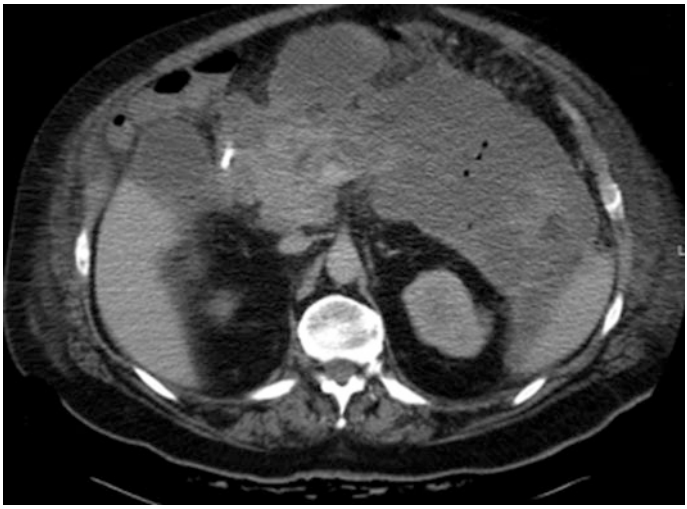

Fig. 33.1 IV contrast-enhanced CT scan showing pancreatic and peripancreatic necrosis and small gas bubbles, suggesting infected necrosis

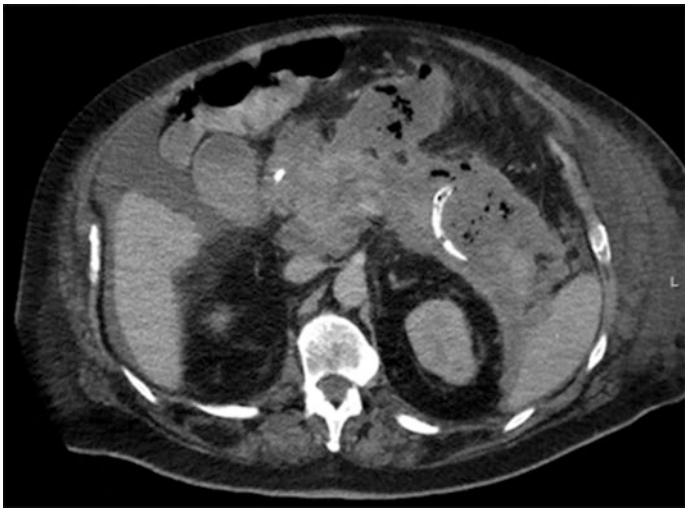

Fig. 33.2 CT scan showing percutaneous drain placed in a necrotic collection

patient was febrile to 39.5 °C and required norepinephrine to maintain a systolic blood pressure > 90 mmHg. He required mechanical ventilation for hypoxemia and required continuous veno-venous hemodialysis for an acute kidney injury. Contrast-enhanced computed tomography (CT) scan (Fig. 33.1) revealed necrotizing pancreatitis with evidence of infected pancreatic and peripancreatic necrosis

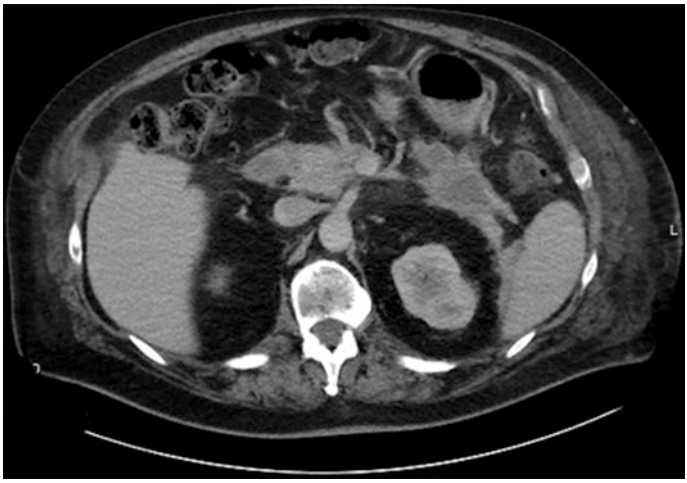

Fig. 33.3 Four-week post video-assisted retroperitoneal debridement CT scan demonstrating near complete resolution of the peripancreatic necrosis

in the lesser sac and tracking into the left pericolic gutter. He was started on broad-spectrum antibiotics and taken to interventional radiology for placement of a percutaneous drain into the infected fluid collection (Fig. 33.2). After percutaneous drainage his clinic course stabilized, and at four weeks he was taken to the operating room for a video-assisted retroperitoneal pancreatic debridement (VARD). Postoperatively he was able to be extubated, and his renal function recovered. Follow-up CT 1-month post debridement showed near complete resolution of his peripancreatic and pancreatic necrosis (Fig. 33.3).

Pathophysiology and Determination of Severity

Acute pancreatitis is a consequence of the intra-acinar cell cleavage of trypsinogen to trypsin, with subsequent activation of other enzymes. The local inflammatory response in the pancreas is associated with the liberation of oxygen-derived free radicals and cytokines, including interleukin (IL)-1, IL-6, IL-8, tumor necrosis factor alpha (TNFα), and platelet-activating factor (PAF) [3]; these mediators play an important role in the transformation of a local inflammatory response to systemic illness. The revised Atlanta classification of acute pancreatitis [4] stratifies patients with acute pancreatitis into mild, moderately severe, or severe categories based on the presence of organ failure and the presence of local or systemic complications. Organ failure is assessed by the modified Marshall scoring system (Table 33.1). Organ failure is defined by a score of two or more for the respiratory, cardiovascular, or renal systems. Local complications include acute peripancreatic fluid collections, pancreatic pseudocysts, acute necrotic collections (sterile or infected),

Table 33.1 Modified Marshall scoring system for organ dysfunction

Organ system	Score[a]				
	0	1	2	3	4
Respiratory ($Pa0_2/FiO_2$)	>400	301–400	201–300	101–200	<101
Renal (serum Cr, mg/dl)	<1.4	1.4–1.8	1.9–3.6	3.6–4.9	>4.9
Cardiovascular (systolic BP, mm Hg)	>90	<90 fluid responsive	<90 not fluid responsive	<90 pH < 7.3	<90 pH < 7.2

[a]A score of 2 or greater defines organ failure

and walled of pancreatic necrosis (sterile or infected). Patients with mild pancreatitis have no evidence of organ failure or local or systemic complications. Moderately severe acute pancreatitis is defined by transient organ failure (resolves within 48 h) and/or local or systemic complications without persistent organ failure. Severe acute pancreatitis is characterized by persistent organ failure of one or multiple systems.

Medical Therapy

Initial therapy for patients with pancreatitis is mostly supportive. Severe acute pancreatitis is divided into two clinical phases; an early vasoactive and a late septic phase. The vasoactive phase typically occurs during the first 2 weeks and is dominated by the consequences of SIRS. Severe pancreatitis is associated with a marked increase in microvascular permeability, leading to large volume losses of intravascular fluid into the tissues, thereby decreasing perfusion of the lungs, kidneys, and other organs. The single most important element in preventing multiple organ failure is vigorous fluid resuscitation with electrolyte solutions in order to optimize cardiac output and to maintain hemodynamic stability. The management of the first phase of severe pancreatitis is summarized here:

Management of the First Phase of Severe Pancreatitis

- Fluid resuscitation
- Respiratory support
- Cardiovascular support
- Relief of pain
- Limitation of systemic complications
- Treatment of metabolic complications
- Nutritional support
- Prevention of infection

Table 33.2 Randomized controlled trials of enteral versus parenteral nutrition in severe pancreatitis

Author	Year	Country	Enteral	TPN	Rate of pancreatic infection
Kalfarentzos	1997	Greece	18	20	Decreased with enteral
Gupta	2003	UK	8	9	Decreased with enteral
Louie	2005	Canada	10	18	Decreased with enteral
Eckerwall	2006	Sweeden	23	25	Equal TPN and enteral
Petrov	2006	Russia	35	34	Decreased with enteral
Casas	2007	Spain	11	11	Decreased with enteral
Doley	2009	India	25	25	Equal TPN and enteral
Wu	2010	China	53	54	Decreased with enteral

The second phase of the disease is characterized by infection of pancreatic necrosis and subsequent sepsis. Both phases can result in multi-organ failure and death. Patients with mild pancreatitis usually experience resolution of their pain within 24–48 h after a regimen of no oral intake, narcotics for pain relief, and intravenous fluids.

Nutrition

Increasing evidence has suggested that enteral nutrition may be feasible, safe, and even desirable in severe pancreatitis (Table 33.2). Several randomized trials have documented that enteral nutrition, when tolerated, has the advantage of avoiding the high cost of total parenteral nutrition (TPN), as well as catheter-related complications, particularly line sepsis. Furthermore, the use of enteral nutrition, usually through a nasojejunal tube, may support intestinal mucosal integrity and avoid the alterations to intestinal barrier function and altered intestinal permeability associated with TPN. Enteral nutrition should be used if tolerated.

Prophylactic Antibiotics

Pancreatic infection is common with pancreatic necrosis, and the incidence of this infection increases with time, although it rarely occurs before the second week. Aerobic and anaerobic gastrointestinal flora are the primary organisms involved, and infections may be monomicrobial or polymicrobial. An association between pancreatic infection and mortality has been the rationale behind the widespread use of prophylactic systemic antibiotics in patients with pancreatic necrosis. Multiple prospective, randomized trials have compared prophylactic antibiotic treatment versus no treatment to prevent infection in patients with pancreatic necrosis (Table 33.3). Each trial has limitations; however, none have conclusively proved prophylactic antibiotic treatment decreases infectious complications, the rate of

Table 33.3 Randomized controlled trials of IV antibiotics for prophylaxsis for acute pancreatitis

Author	Year	Country	Abx	Treat group	Control group	Result
Howes	1975	US	Amp	48	47	No difference
Craig	1975	US	Amp	23	23	No difference
Finch	1976	US	Amp	31	27	No difference
Pederzoli	1993	Italy	Imipen	41	33	Decreased pancreatic infection
Sainio	1995	Finland	Cefurox	30	30	Decreased mortality
Delcenserie	1996	France	Ceftaz + Amik + Met	11	12	No difference
Schwarz	1997	Germany	Oflox + Met	13	13	No difference
Spicak	2003	Czech	Meropen	20	21	No difference
Isenmann	2004	Germany	Cipro + Met	58	56	No difference
Dellinger	2007	NA + Europe	Meropen	50	50	No difference
Rokke	2007	Norway	Imipen	36	37	Decreased pancreatic infection
Xue	2009	China	Imipen	29	27	No difference
Garcia-Barrasa	2009	Spain	Cipro	22	19	No difference

Amp Ampicillin, *Cefurox* Cefuroxime, *Ceftaz* Ceftazadine, *Amik* Amikacin, *Met* Metronidazole, *Cipro* Ciprofloxacin

need for surgical intervention, or mortality. The use of broad-spectrum antibiotics for this purpose is known to change the bacterial flora of pancreatic infection, and has been demonstrated to encourage the development of antibiotic-resistant bacterial and fungal infections [5]. The risk of superinfection is thought to be related to the length of treatment with prophylactic antibiotics. Currently, most authorities advocate against prophylactic antibiotic administration in necrotizing pancreatitis.

Management of Pancreatic Necrosis

Between 5 and 10% of patients with acute pancreatitis will develop necrosis of the pancreas and/or peripancreatic tissue. Intravenous contrast-enhanced CT scanning is the preferred imaging test for identifying pancreatic necrosis, as seen in Fig. 33.1. The impairment of pancreatic perfusion and subsequent pancreatic necrosis usually evolves over several days from the acute injury, and therefore early CT scanning may underestimate the degree of pancreatic necrosis. The sensitivity for identifying pancreatic necrosis using contrast-enhanced CT scan approaches 100% after four

days from presentation. It is therefore reasonable to recommend an abdominal CT scan with intravenous contrast in patients with clinical and biochemical features of acute pancreatitis who do not improve after several days of conservative management. The extent of pancreatic and peripancreatic necrosis estimated on early contrast-enhanced helical CT is a specific predictor of morbidity and mortality.

Pancreatic and peripancreatic necrosis may be sterile or infected. There appears to be no correlation between the extent of necrosis and the development of infection. Infected pancreatic and peripancreatic necrosis is usually diagnosed by the demonstration of extra luminal gas on a contrast-enhanced CT scan, shown in Fig. 33.2, or by a positive gram stain or culture on image-guided fine-needle aspiration.

Sterile necrosis is best managed medically during the first 3–4 weeks. After this interval, if abdominal pain persists and prevents oral intake, debridement should be considered. This may be accomplished surgically, but percutaneous or endoscopic debridement is a reasonable choice in selected circumstances, if appropriate expertise is available. Delaying operative intervention for 4 weeks allows for consolidation of the peripancreatic necrosis, and allows for a safer debridement.

In the setting of infected pancreatic and peripancreatic necrosis the goal of intervention is to debride all necrotic infected tissue, drain infected fluid collections, minimize the risk of technical complications (including bleeding and enteric fistula), and ensure abdominal wall integrity. These goals can be accomplished either operatively, endoscopically, percutaneously, or by a combination of all approaches. Operative intervention should be delayed for at least 4 weeks after the original presentation due to the excessive mortality and morbidity from early operative debridement. Percutaneous drainage may be employed earlier if clinically indicated for control of sepsis.

Operative approaches can be categorized as either open (performed through a laparotomy incision) or minimally invasive, in which the retroperitoneum is reached endoscopically, laparoscopically, or through a small flank incision. The choice of approach depends on the specific anatomic locations of the areas to be drained or debrided and the severity of critical illness, which determines the rate at which source control needs to be achieved. Our approach is summarized in Fig. 33.4. In patients with necrosis limited to the lesser sac, we would recommend either an endoscopic or a laparoscopic transgastric necrosectomy. If the necrosis is limited to the lesser sac and tracks down the right or left pericolic gutter, we would favor a video-assisted retroperitoneal debridement (VARD) after establishing percutaneous drainage of the necrosis cavity. An open pancreatic debridement is preferred for patients with extensive necrosis that tracks into both pericolic gutters, or centrally down the root of the small bowel mesentery.

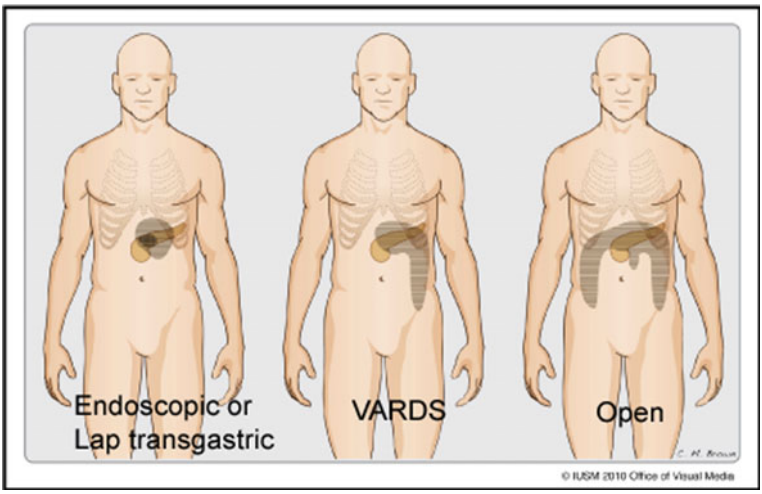

Fig. 33.4 Patterns of pancreatic necrosis and preferred debridement technique. Reprinted from Journal of Gastrointestinal Surgery. 2016; 20(2):445–9. Transgastric pancreatic necrosectomy: how I do it. Zyromski NJ, Nakeeb A, House MG, Jester AL. With permission of Springer

Management of Pancreatic Necrosis

- Supportive care during early phase of severe acute pancreatitis
- Avoid prophylactic antibiotic therapy
- Early enteral nutrition
- Percutaneous drainage of infected necrosis
- Delay pancreatic debridement for minimum of 4 weeks
- Individualize debridement technique to pattern of necrosis.

Endoscopic Necrosectomy

Endoscopic necrosectomy can be accomplished from either the stomach or the duodenum. Puncture of the fluid collection can be made either directly by visualizing a bulge or with endoscopic ultrasound (EUS) guidance. The collection is punctured with a 19-gauze needle and a guide-wire is advanced under fluoroscopic guidance. The tract is balloon dilated up to 8 mm and either two double pigtail plastic stents or a lumen-opposing metal stent is placed. The cavity is irrigated with 1 L of normal saline per 24 h via a nasogastric tube placed into the collection. Necrotic tissue is evacuated with a basket, a net, or a polypectomy snare [6, 7]. The endoscopic approach often requires multiple procedures to adequately remove all

necrotic material. Lumen-opposing stents should be removed once the necrosectomy is completed. Several authors recommend leaving double pigtail stents in permanently, though long-term follow-up of this strategy is lacking.

Laparoscopic Transgastric Necrosectomy

An alternative approach to an endoscopic necrosectomy is the laparoscopic transgastric necrosectomy [8]. Advantages of this approach are the ability to accomplish debridement in a single procedure, the creation of a large cystogastrostomy to drain residual collections, and the ability to perform cholecystectomy for patients with gallstone pancreatitis. This approach can also be used in patients with gastric varices from sinistral portal hypertension, making endoscopic transgastric drainage too dangerous. The procedure is shown in Fig. 33.5. A gastrotomy is created in the anterior wall of the stomach between stay sutures, and a laparoscopic aspirating needle is placed thru the posterior wall of the stomach into the necrosis cavity to localize the collection. Alternatively, intraoperative ultrasound can be used to identify the point of contact between the posterior stomach and the necrosis cavity. A posterior stay suture is placed into the posterior gastric wall and used as a traction suture to facilitate a posterior gastrotomy made with an ultrasonic scalpel. The posterior gastrotomy is extended, and a running 2–0 monofilament suture or endovascular stapler can be used to secure the stomach to the cyst cavity wall. Laparoscopic instruments and suction irrigation are then used to debride all loose necrosis from the retroperitoneum and placed in an endocatch bag for extraction. The anterior gastrotomy is then closed with a linear stapler or suture. This transgastric approach is also feasible through a short (open) upper midline incision.

Video-Assisted Retroperitoneal Debridement (VARD)

The initial step to performing VARD procedure is to have the interventional radiologists place a 14 French percutaneous drain into the peripancreatic collection through a retroperitoneal flank approach. This drain may be serially upsized, and may provide definitive treatment for the necrosis in up to one-third of patients [9]. If percutaneous drainage does not lead to clinical improvement, VARD may be undertaken (Fig. 33.6). The patient is placed in supine position with the left side elevated 30°–40° A 5-cm incision is made close to the exit point of the percutaneous drain. The drain is then used as a guide to carefully dissect into the retroperitoneum, and the cavity entered. Irrigation and debridement of the superficial necrosis are carried out under direct vision. A 0° laparoscope or a videoendoscope can be placed into the cavity and further debridement can be accomplished using ring forceps or laparoscopic graspers and suction irrigators [10]. The debridement should be performed cautiously, removing only loose nonadherent necrosis, to avoid injury to any underlying blood vessels. Bleeding can be controlled with electrocautery or laparoscopic clips. In the rare case of extensive

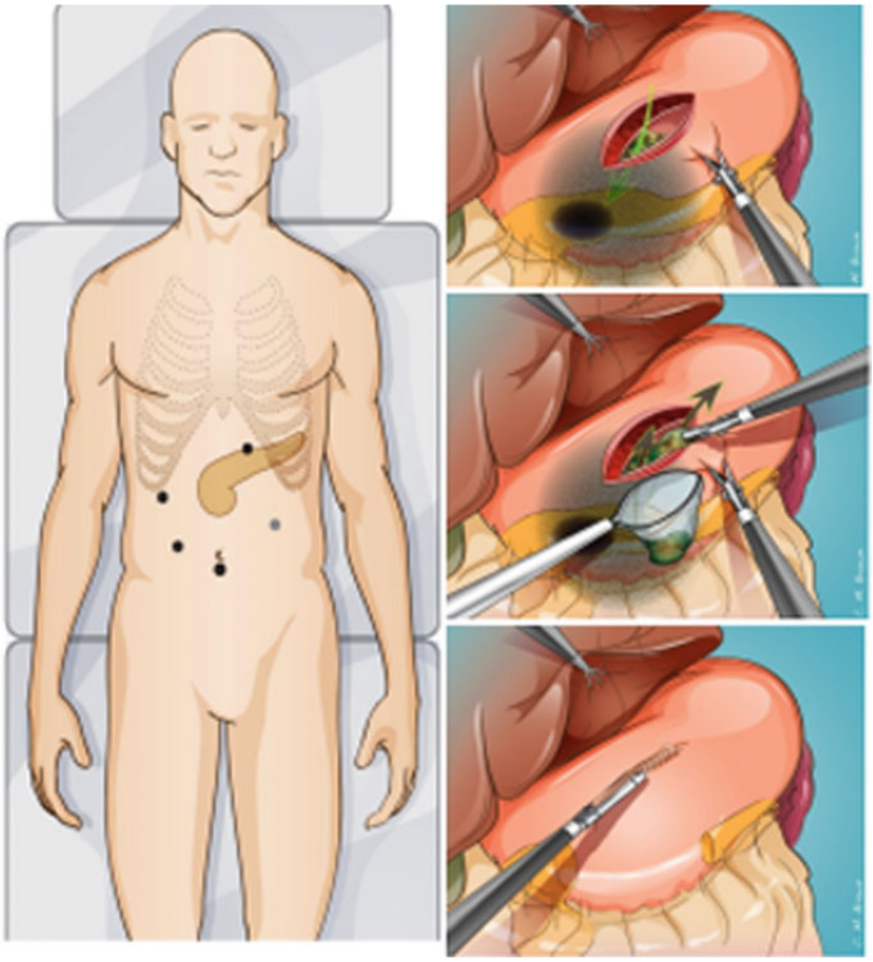

Fig. 33.5 Technique of laparoscopic transgastric debridement. Reprinted from Journal of Gastrointestinal Surgery. 2016; 20(2):445–9. Transgastric pancreatic necrosectomy: how I do it. Zyromski NJ, Nakeeb A, House MG, Jester AL; with permission of Springer

hemorrhage, packing of the retroperitoneal cavity can be performed and the procedure converted to a laparotomy, or the patient can be taken to radiology for angiographic embolization. After the debridement is completed, the percutaneous drain is exchanged for two drains that are brought out through the incision, and the fascia is closed. Continuous lavage is performed through the drains, with either normal saline or dialysis fluid, until the effluent is clear.

This step-up approach to the management of pancreatic necrosis was compared to traditional open debridement in a multicenter, randomized, prospective trial completed in the Netherlands [9]. The authors found that of the patients assigned to the step-up approach, 35% were treated with percutaneous drainage only.

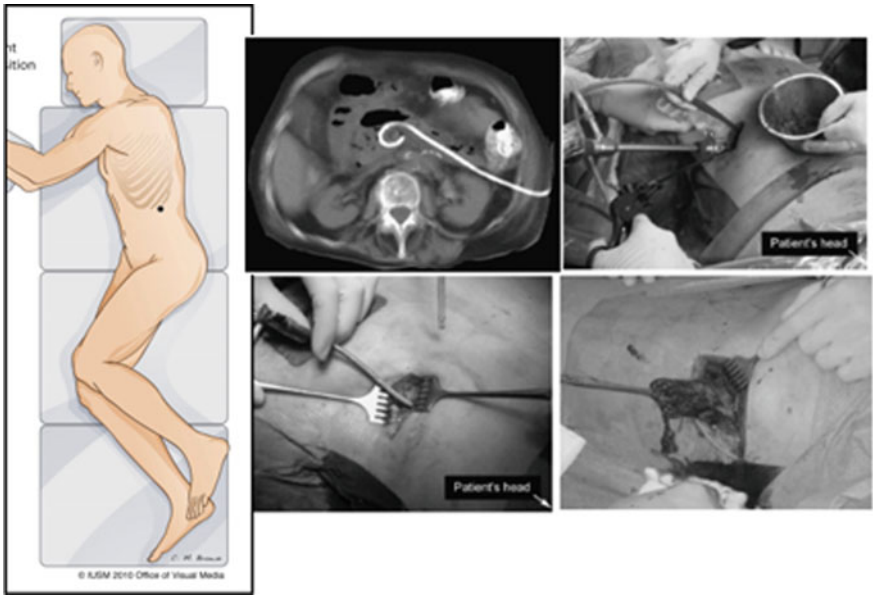

Fig. 33.6 Technique of video-assisted retroperitoneal debridement. Reprinted from HPB (Oxford) 2007;9:156–59. van Santvoort HC, Besselink MG, Horvath KD, Sinanan MN, Bollen TL, van Ramshorst B, et al. Videoscopic assisted retroperitoneal debridement in infected necrotizing pancreatitis; with permission from Elsevier

New-onset multiple organ failure occurred less often in patients assigned to the step-up approach than in those assigned to open necrosectomy (12% vs. 40%, $P = 0.002$). Mortality did not differ significantly between groups (19% vs. 16%). Additionally, patients assigned to the step-up approach had a statistically significant lower rate of incisional hernias (7% vs. 24%) and new-onset diabetes (16% vs. 38%) than patients treated with open debridement.

Open Pancreatic Debridement

Open pancreatic debridement remains a viable option for patients with infected pancreatic and peripancreatic necrosis that are not amenable to, or have failed, minimally invasive techniques. The goals of open pancreatic debridement are to control infection, to evacuate all peripancreatic fluid and necrotic debris, to externally drain any pancreatic fistulae, and to establish enteral access for postoperative nutrition. Surgical intervention should be delayed for a minimum of 4 weeks if possible. The extent of debridement should be based on careful interpretation of preoperative CT imaging to ensure all necrotic collections are addressed.

Open pancreatic debridement can be accomplished through a midline or bilateral subcostal incision. The pancreatic and peripancreatic necrosis can be accessed

either through the transverse mesocolon or directly through the gastrocolic ligament. If a transverse mesocolon approach is chosen, the transverse colon and omentum are elevated anteriorly and an opening is made in the avascular plane to the left of the middle colic vessels to enter the lesser sac. Care must be taken to dissect any adherent small bowel away from the mesocolon. If a gastrocolic approach is chosen, the gastrocolic omentum should be divided inferior to the gastroepiploic vessels to enter the lesser sac. Oftentimes this plane is difficult to enter, due to significant inflammation in the lesser sac, and care should be taken not to injure the colon or its mesentery. Once the lesser sac is entered, all the peripancreatic fluid and necrosis can be gently debrided using a combination of ring forceps and suction/irrigation. Again, only nonadherent necrosis and devitalized tissue are removed, and care should be taken not to avulse any blood vessels. If the necrosis tracks down the pericolic gutters, the colon should be mobilized medially to facilitate debridement. Large-caliber drains should be placed into the necrosis cavity to control any potential pancreatic fistulae and to facilitate postoperative lavage. Our preference is to place a gastrojejunostomy tube for postoperative gastric decompression and enteral feeding.

Approaches to Pancreatic Debridement

- Percutaneous drainage
- Endoscopic transgastric
- Laparoscopic transgastric
- Video-assisted retroperitoneal debridement (VARDS)
- Open debridement (laparotomy).

Complications

The main complications associated with pancreatic necrosectomy include perioperative hemorrhage; pancreatic fistula and disconnected left pancreatic remnant; enteric fistulas (colon, duodenum, stomach); intestinal/gallbladder ischemia; and pancreatic endocrine and exocrine insufficiency.

Hemorrhage in acute pancreatitis may be venous or arterial, and may occur prior to or following intervention. Pre-intervention hemorrhage is reliably diagnosed by the presence of high attenuation (30 Hounsfield Units) material in peripancreatic collections visualized by contrast-enhanced computed tomography (CT). After intervention (either operation or percutaneous drainage of pancreatic necrosis), the presence of blood in surgical or radiologically placed drains is the most common manifestation of this complication. Though relatively minor venous bleeding (perhaps from irritation by the drains) is fairly common, potentially life-threatening bleeding from visceral arterial pseudoaneurysm (PSA) must be considered and

ruled out. Currently, dedicated CT angiogram is the exam of choice to diagnose PSA; in addition to offering a high-contrast evaluation of the entire visceral arterial tree with a single contrast bolus, this test also provides cross-sectional abdominal images of residual peripancreatic collections. Angiographic embolization provides definitive therapy for PSA in nearly all cases [11].

Pancreatic fistula by definition involves disruption of the pancreatic ductal system, and may manifest as an external fistula (following intervention), or as pancreatic ascites or pleural effusion with amylase rich fluid in patients who have not been instrumented. Defining the pancreatic ductal anatomy is central to planning treatment; this work-up generally requires endoscopic retrograde cholangiopancreatography (ERCP)—which may be therapeutic as well as diagnostic. It is worthy of note that magnetic resonance cholangiopancreatography (MRCP) is less helpful in the setting of ascites or peripancreatic fluid collections, which obscure ductal anatomic features. Fistulae from smaller side branches are typically lower volume, on the order of 50 mL daily. These side branch fistulae generally "dry up" spontaneously, and may be managed by sequential "cracking" and withdrawal of drains. Fluoroscopic sinogram in these situations often provides valuable information. Major pancreatic fistulae result from "disconnection" of the main pancreatic duct, where a viable body/tail loses ductal continuity with the pancreatic head and duodenum. The viable, disconnected left pancreatic remnant generally requires operative intervention, with the patient's anatomy dictating ideal operation—pancreaticojejunostomy versus left pancreatectomy/splenectomy [12]. In the setting of disconnected left pancreatic remnant, transgastric debridement with "cyst-gastrostomy" draining the pancreatic tail at the time of initial debridement is an attractive solution for select patients.

Intestinal or colonic ischemia probably occurs with much greater frequency than is commonly recognized in patients with necrotizing pancreatitis; clinicians caring for these patients must keep a high degree of suspicion for this problem, especially in patients who suddenly turn for the worse after a period of relative stability. The only way to assuredly rule out (or rule in) ischemic bowel is by laparotomy and direct inspection of the abdominal contents. The price of a "nontherapeutic" laparotomy is small compared to that of missing the diagnosis and potential to treat ischemic bowel before perforation occurs.

Awareness of the abdominal compartment syndrome is important in patients with severe acute pancreatitis. Patients with findings of intra-abdominal compartment syndrome require decompressive laparotomy if they fail to respond to nonoperative measures.

Conclusion

Management of patients with severe pancreatitis and pancreatic necrosis require a multi-disciplinary team. Surgeons, critical care physicians, gastroenterologists, and interventional radiologists must be involved in caring for these complex patients.

Over the past decade, better critical care, the introduction of early enteral nutrition, the appropriate use of antibiotics, delaying intervention for a minimum of 4 weeks, and the application of minimally invasive techniques have all led to lower morbidity and mortality in patients with pancreatic necrosis.

References

1. Fagenholz PJ, Fernandez-del Castillo C, Harris NS, Pelletier AJ, Camargo Jr CA. Direct medical costs of acute pancreatitis hospitalizations in the United States. Pancreas. 2007;35 (4):302–7.
2. Freeman ML, Werner J, van Santvoort HC, Baron TH, Besselink MG, Windsor JA, et al. Interventions for necrotizing pancreatitis: summary of a multidisciplinary consensus conference. Pancreas. 2012;41(8):1176–94.
3. Frossard JL, Steer ML, Pastor CM. Acute pancreatitis. Lancet. 2008;371(9607):143–52.
4. Banks PA, Bollen TL, Dervenis C, Gooszen HG, Johnson CD, Sarr MG, et al. Classification of acute pancreatitis–2012: revision of the Atlanta classification and definitions by international consensus. Gut. 2013;62(1):102–11.
5. Howard TJ, Tempel MB. Prophylactic antibiotics alter the bacteriology of pancreatic necrosis in severe acute pancreatitis. J Am Coll Surg. 2002;195(6):759–76.
6. Bakker OJ, van Santvoort HC, van Brunschot S, Geskus RB, Besselink MG, Bollen TL, et al. Endoscopic transgastric vs surgical necrosectomy for infected necrotizing pancreatitis: a randomized trial. JAMA. 2012;307(10):1053–61.
7. Haghshenasskashani A, Laurence JM, Kwan V, Johnston E, Hollands MJ, Richardson AJ, et al. Endoscopic necrosectomy of pancreatic necrosis: a systematic review. Surg Endosc. 2011;25(12):3724–30.
8. Zyromski NJ, Nakeeb A, House MG, Jester AL. Transgastric pancreatic necrosectomy: how I do it. J Gastrointest Surg. 2016;20(2):445–9.
9. van Santvoort HC, Besselink MG, Bakker OJ, Hofker HS, Boermeester MA, Dejong CH, et al. A step-up approach or open necrosectomy for necrotizing pancreatitis. N Engl J Med. 2010;362(16):1491–502.
10. van Santvoort HC, Besselink MG, Horvath KD, Sinanan MN, Bollen TL, van Ramshorst B, et al. Videoscopic assisted retroperitoneal debridement in infected necrotizing pancreatitis. HPB (Oxford). 2007;9(2):156–9.
11. Zyromski NJ, Vieira C, Stecker M, Nakeeb A, Pitt HA, Lillemoe KD, et al. Improved outcomes in postoperative and pancreatitis-related visceral pseudoaneurysms. J Gastrointest Surg. 2007;11(1):50–5.
12. Murage KP, Ball CG, Zyromski NJ, Nakeeb A, Ocampo C, Sandrasegaran K, et al. Clinical framework to guide operative decision making in disconnected left pancreatic remnant (DLPR) following acute or chronic pancreatitis. Surgery. 2010;148(4):847–56; discussion 856–7.