Operative Management of Acute Pancreatitis

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KEYWORDS

- Acute pancreatitis
 Management
 Necrosis
 Pseudocysts
 Pancreatic resection
- Pseudocyst drainage

KEY POINTS

- The operative management of acute pancreatitis is focused on managing the acute complications, the long-term sequelae, or the prevention of recurrent pancreatitis.
- Using the least amount of intervention to achieve the stated goals has always been the
 case; however, the evolution of videoscopic and endoscopic techniques have greatly
 expanded the tools available.
- Patience, vigilance, expertise, judgment, and an ability to be humbled are necessary for the successful practitioner who manages patients with severe pancreatitis.

Acute pancreatitis is more of a range of diseases than it is a single pathologic entity. Its clinical manifestations range from mild, perhaps even subclinical, symptoms to a life-threatening or life-ending process. The classification of acute pancreatitis and its forms are discussed in fuller detail by Sarr and colleagues elsewhere in this issue. For the purposes of this discussion, the focus is on the operative interventions for acute pancreatitis and its attendant disorders.

The most important thing to consider when contemplating operative management for acute pancreatitis is that we do not operate as much for the acute inflammatory process as for the complications that may arise from inflammation of the pancreas. In brief, the complications are related to: necrosis of the parenchyma, infection of the pancreas or surrounding tissue, failure of pancreatic juice to safely find its way to the lumen of the alimentary tract, erosion into vascular or other structures, and a persistent systemic inflammatory state. The operations may be divided into three major categories: those designed to ameliorate the emergent problems associated with the ongoing inflammatory state, those designed to ameliorate chronic sequelae

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of an inflammatory event, and those designed to prevent a subsequent episode of acute pancreatitis. This article provides a review of the above.

ACUTE PANCREATITIS

Acute pancreatitis may be histologically classified as either interstitial or necrotizing edematous, each of which have its unique complications. Acute pancreatic inflammation will progress to pancreatic necrosis in approximately 20% to 30% of patients with severe acute pancreatitis. The terminology used to describe acute pancreatitis and its consequences has a long history of being less than well standardized. Over the past 20 years or so, attempts at using standardized descriptions have been markedly improved. In another article by Sarr and colleagues in this issue, the most recent consensus agreements are reviewed in detail. Pancreatic necrosis is defined by the International Symposium on Acute Pancreatitis as the presence of one or more diffuse or focal areas of nonviable pancreatic parenchyma, usually associated with peripancreatic fat necrosis. The Acute Pancreatitis Classification Working Group has further elaborated on this in defining three distinct subgroups: pancreatic parenchymal and associated peripancreatic necrosis, pancreatic parenchymal necrosis alone, or peripancreatic necrosis alone. All of these can be either sterile or infected.

Walled-off necrosis, formerly referred to as organized pancreatic necrosis, is a well-circumscribed collection of purulent material in close proximity to the pancreas that develops greater than 4 weeks after an episode of necrotizing pancreatitis. It is due to secondary infection of liquefied necrosis that then becomes walled off.^{3–8} It is generally a highly viscous collection and contains liquid and solid or semisolid debris.

Pancreatic ductal disruptions may develop in up to 50% of patients who have acute necrotizing pancreatitis and may lead to peripancreatic fluid collections. Loss of ductal integrity may lead to further necrosis, infection, or fistulization. ^{9–11} The persistent leakage of pancreatic fluid may result in pseudocyst formation (a bounded collection) or diffuse leakage into the retroperitoneum yielding worsening inflammation, leakage into the peritoneal cavity resulting in pancreatic ascites, or leakage into the thorax resulting in pancreatic pleural effusion. The latter two phenomena are essentially pathognomonic for main pancreatic ductal disruption.

Disconnected tail syndrome is defined by a complete disruption of the main pancreatic duct (demonstrated by loss of opacification of the duct or inability to place a guidewire into the distal duct) and CT imaging demonstrating contrast-enhancing viable pancreatic tissue upstream from the disruption. A nonhealing pancreatic fistula, pseudocyst, or fluid collection despite a course of conservative medical management is added to the definition by some investigators. The downstream pancreas can drain via the papilla or retrograde into a fluid collection or fistula, whereas the upstream pancreas will drain aberrantly until the drainage is redirected or that section of pancreas is removed or atrophies. Of note: the nomenclature that is conventionally used for the pancreas and its ducts is potentially confusing. The generally accepted anatomic principal of referring to proximal and distal based on direction of flow is completely ignored when referring to the pancreas. In the opinion of the corresponding author, correcting this and renaming some operations accordingly would be optimal. However, this is most unlikely to occur.

The cause of acute pancreatitis is most often related to gallstones or ethanol use, though a myriad of other causes have been identified. In the United States, gallstone pancreatitis is the most common form of acute pancreatitis due to transient obstruction at the ampulla or from increased pancreatic ductal pressure secondary to persistent stone impaction or ampullary scarring secondary to stone passage. 13,14

Operations and Indications

As mentioned above, the operations used for acute pancreatitis are designed to manage the complications in the acute phase, the long-term sequelae, or prevention of subsequent episodes of acute pancreatitis. In this section, procedures used in the acute phase of inflammation are discussed. These operations are mostly aimed at removing dead or devitalized tissue, removing or draining infected solid or semisolid tissue, draining pus, and/or providing a safer avenue for egress of pancreatic secretions. Although much is written about these procedures, and there always seems to be more confusion than necessary about these operations, they are really quite straightforward in nature. Some simple principles pertain: all infected material must go; the degree of containment, or lack thereof, of the suspect material must be considered; the viscosity of the problematic tissue or fluid collection influences choices; and consideration of draining or resecting potentially sterile fluid or tissue invariably is a clinical judgment based on the overall status of the patient.

PANCREATIC NECROSIS

Pancreatic necrosis results from insufficient perfusion of pancreatic parenchyma to support metabolic requirements. There are many models of how this actually happens and they are discussed by Sarr and colleagues elsewhere in this issue in greater detail. Necrosis of the pancreas may be relatively minor and self-limiting or it may progress to a more substantial and potentially life-threatening process. ¹⁵ Although pancreatic necrosis remains sterile, the main indications for intervention are an uncontrolled systemic inflammatory response syndrome (SIRS) or significant question over whether the presumption of sterility is secure. It is generally accepted that infected pancreatic necrosis is an indication for intervention. ¹⁵ Over the past several years, the enthusiasm for early operative intervention in suspected sterile necrosis has waned because, even in the setting of multiorgan failure, many studies have shown that operative management does not confer significant mortality benefit and may actually increase morbidity. The rare indications for intervention in sterile necrosis include worsening organ failure despite maximal support, inability to tolerate enteral nutrition, weight loss, worsening jaundice, fevers, or failure to improve after 4 to 6 weeks of nonoperative management. ¹⁶

In the setting of infected, possibly infected, or worsening sterile necrosis, debridement is preferred to resection in an attempt to preserve as much functional organ as possible. Also, attempts at an anatomic resection in the setting of severe acute pancreatitis are frequently not technically possible and are likely to yield more complications than they resolve. The survival rate is generally improved the longer operative management can be delayed—unless, of course, clear evidence of significant infection is present. This is likely due to better demarcation resulting in removal of less vital tissue and less bleeding. Some investigators have suggested that optimal outcomes have been realized when surgery can be delayed for at least 1 month. Althory The only randomized controlled trial comparing debridement within the first 72 hours with debridement after at least 12 days was terminated early; however, preliminary data demonstrated a mortality of 56% for the early group and 27% for the late group. In the authors' experience, the main benefit from waiting, when possible, to operate is reducing the number of operations required to achieve the main clinical objectives.

Significant complication rates in cases associated with pancreatic debridement include: pancreatic fistula in 41% to 50%; exocrine insufficiency in 20%; endocrine insufficiency in 16%; enteric fistulas in greater than 10%; postoperative hemorrhage in 3% to 20%; prolonged postoperative hospitalization, typically greater than 1 month; and greater than 4 months before return to regular activities.^{2,18,19} Surgical debridement

is associated with an overall morbidity rate of 19% to 62% and a mortality rate of 6% to 28% .^{2,19}

Technical Considerations in Managing Pancreatic Necrosis

The technical choices made in the management of pancreatic necrosis must always be made with some basic principles in mind. First, be mindful of the desired end state—removal of all devitalized and infected tissue in the setting of an alive patient. Second, achieve the first goal in the least invasive and least traumatic way possible. Einstein is quoted as saying, "Everything should be made as simple as possible but not simpler." The same philosophy applies to pancreatic debridement; it should be as minimally invasive as possible but not more so. If achieving the clinical objectives in a timely and cost-effective manner requires a more invasive procedural choice, then do so. Using minimally invasive techniques for the sake of doing so misses the point altogether.

Open Necrosectomy

Open approach to necrosectomy can be done in many ways. One approach includes a necrosectomy and closure with standard surgical drains that are left in place for an average of 7 days. Reoperation is performed on an as-needed basis. Reports of this technique show a mortality rate of 4% to 19%. 14 Some investigators suggest abandoning this approach because of inadequate debridement and a 40% incidence of reinfection.¹⁴ However, other contributors feel this technique may be sufficient for very small, well-delineated processes.¹⁵ The open or semiopen technique includes necrosectomy combined with open packing and scheduled repeat laparotomies, usually every 48 hours, until all necrotic tissue has been removed. Followed by closure, or not, of the abdomen depending on the clinical circumstances. This technique has a high reported rate of postoperative complications, including pancreatic fistulas, bowel compromise, and bleeding, as well as mortality rate of 4% to 18%. 14 The closed technique entails necrosectomy with extensive intraoperative lavage of the pancreatic bed followed by closure over large-bore drains for continued postoperative high-volume lavage of the lesser sac. Reports of this technique suggest lower mortality rates of 7% to 9% compared with other open procedures. 14

Although all the techniques listed above can and do work, the literature reporting their relative effectiveness may be difficult to compare. Even attempts at carefully controlled studies are challenging in patients with these maladies. Also, in the corresponding author's opinion, it may be impossible to control for all the operative and surgical variation that inherently exists in the management of these patients.

Minimally Invasive Surgery Approach for Necrosis

As in all other areas of surgery, minimally invasive approaches are gaining popularity in the management of pancreatic necrosis. Potential benefits claimed are: minimizing operative trauma; decreased incidence of incisional hernias; avoidance of bacterial contamination and translocation, thereby improving the postoperative septic response; and decreasing the need for ICU care. Some of these claims may be aspirational over actual. To date, no survival benefit over open procedures has been clearly demonstrated. Potential limitations include poor surgical exposure, difficulty removing solid or highly viscous necrotic tissue through small ports, loss of tactile guidance, need for multiple procedures, longer overall hospital stays, and need for reliance on interventional radiology.

The semiopen technique uses a retroperitoneal approach via a small 5 cm incision. The Dutch Acute Pancreatitis Study Group has compared this technique to open necrosectomy with continuous postoperative lavage. They found favorable outcomes

in the semiopen group, which developed less postoperative multiorgan failure and demonstrated a trend toward decreased mortality. In addition, there was no difference in the number of procedures required between the two groups.²¹

Videoscopic options include transperitoneal or retroperitoneal approaches. ¹⁴ The retroperitoneal approach potentially avoids peritoneal contamination and is typically done after a period of CT-guided drainage. One major disadvantage of this approach is the inability to perform other intraabdominal procedures such as cholecystectomy or jejunostomy tube placement. ¹⁴ With the transperitoneal approach, the lesser sac is explored via a transmesocolic route. ¹⁴ At the conclusion of either of these procedures, drains are left in place for postoperative drainage and/or lavage. ¹⁴ There are no randomized controlled trials showing that the laparoscopic approach is superior to open surgery; however, the theoretical advantages include less postoperative pain, shorter length of stay, and earlier return to normal activities. ¹⁴

Transgastric Resection or Drainage

When necrosis and/or fluid collections are contained and limited to the lesser sac they may be approached by a transgastric technique. Transgastric drainage or debridement of the lesser sac can be achieved by open, videoscopic, or endoscopic means. As with other procedures, the choice will not be just based on the tools available and the technical expertise of the operator but also, and more importantly, on the nature and viscosity of the material to be removed.

A step-up approach for controlling the liquid component (pus) of infection, instead of treating the definitive source, is sometimes used as a temporary measure en route to removing the infected necrotic tissue. The first step is to drain the collection of infected fluid using either percutaneous or endoscopic means to mitigate sepsis. If this does not lead to clinical improvement, the next step is minimally invasive retroperitoneal necrosectomy or open necrosectomy. A multicenter trial conducted in 2010 randomized 88 subjects with suspected or confirmed infected necrosis to undergo primary open necrosectomy or a step-up approach. The primary endpoints were major complication or death, which occurred in 69% of subjects assigned to open necrosectomy compared with 40% of those in the step-up group. Of subjects in the step-up approach group, 35% required only percutaneous drainage. New-onset multiorgan failure occurred in 12% of the step-up approach group compared with 40% in the open group. The step-up approach was associated with a lower incidence of incisional hernias (7% vs 24%) and new-onset diabetes (16% vs 38%) compared with the open group; however, the mortality rates did not differ between the two groups.

Percutaneous

The use of percutaneous large-bore catheters for drainage is somewhat controversial. Some early studies have shown promising results, but patient selection and characterization is key. Freeny and colleagues²³ published their results of 34 subjects with necrotizing pancreatitis and medically uncontrolled sepsis treated with drainage and irrigation through large-bore catheters started a mean of 9 days after symptom onset. Of these subjects, 47% avoided an operation, 26% required immediate surgery, and 26% ultimately required delayed repair of a pancreaticocutaneous fistula. The overall mortality rate was 12%, which is comparable to the previously described open procedures. Echenique and colleagues²⁴ published their results of 20 subjects with necrotizing pancreatitis who were treated in a similar fashion, finding a 100% success rate with none requiring further operative interventions. These subjects differed in that they were all hemodynamically stable at time of selection, which may have contributed to their overwhelmingly positive results. Some investigators have advocated this

technique only be used as a bridging procedure in unstable patients, whereas others feel that the percutaneous approach is a good tool for draining abscesses but less useful for performing extended necrosectomy. ¹⁴ As with other procedures, the risk for developing pancreaticocutaneous fistulae is significant, as high as 45% in those with disconnected duct syndrome. ²⁵ Therefore, it may be helpful to evaluate the ductal system before implementing this technique.

Endoscopic techniques may combine transpapillary drainage with transluminal drainage. Placement of transpapillary drains, such as nasopancreatic tubes, can be used for drainage or for continuous lavage and endoscopic ultrasound (EUS)-guided transgastric or transduodenal catheter or stent placement for internal drainage of fluid or low-viscosity debris. This is followed by debridement via a gastroscope that is repeated as needed until all necrotic debris is removed, at which time the nasopancreatic tube may be removed. The stents, however, are left in place until CT evidence of resolution. ^{19,26,27}

An early study, published in 1997, by Baron and colleagues, 28 reported the results of 31 subjects treated in this manner, 81% of which had complete resolution and avoided an open operation. Multiple subsequent studies have demonstrated similar results, with success rates varying between 69% and 100%. 19,26 Two studies reported specifically on bleeding complications, citing an incidence of 17% to 31%. 26 The reported overall complication rates are between 7% and 25%. The largest study to date, by Seifert and colleagues, 29 included 93 subjects and reported a mortality rate of 7.5%. A retrospective study by Connor and colleagues, 30 compared the outcomes in 88 subjects treated with either endoscopic or open necrosectomy. They found that, despite a similar preoperative Acute Physiology and Chronic Health Evaluation (APACHE) II score, the open group had higher scores postoperatively. In addition, they found a trend toward increased survival in the minimally invasive surgery group (P=.06) along with a shorter postoperative ICU stay but longer overall hospital stay. 30

A similar approach using a combination of transgastric or transduodenal endoscopic stent placement and percutaneous drainage is reported. The percutaneous drains are used for lavage and the transluminal stents are used for drainage. Results from a study of 15 subjects receiving this therapy demonstrated a 100% success rate with 0% mortality and no pancreaticocutaneous fistulas.²⁵

Some investigators believe that, for minimally invasive techniques, endoscopic therapy may be associated with less postprocedural SIRS than is associated with open necrosectomy. Direct comparisons are hard to find to validate these assertions. There are no randomized controlled trials comparing the morbidity and mortality of endoscopic techniques and open necrosectomy. Although percutaneous, endoscopic, and advanced videoscopic techniques may have use in very specific situations, there are several caveats that must be understood. All of these techniques are only as good as the people who perform them. The requisite technical expertise and judgment for many of these procedures is not widely available in all centers, let alone all geographic regions. Even when the personnel, resources, and equipment are available, biology still wins. The nature of the material that needs to be drained or be removed will be the absolute determinant of what approaches can be effectively used. Furthermore, the solution must be not only clinically effective but also cost-effective.

PERIPANCREATIC FLUID DRAINAGE

Fluid collections associated with acute pancreatitis fall into two main categories: those acute pancreatic peripancreatic fluid collections that may or may not be

infected and long-standing fluid collections, such as walled-off necrosis or abscess, or pancreatic pseudocysts. A significant fluid collection is seen in 40% of hospitalized patients with acute pancreatitis who undergo CT imaging.³¹ Although half of these collections spontaneously resolve, the other half may require intervention.³¹ Potential complications of chronic fluid collections include rupture, infection, hemorrhage, and biliary or enteric obstruction.³² The specific treatment depends on the cause, which may include ductal disruptions, edema, or liquefied necrosis.

The most recent update on the Atlanta Classification of peripancreatic fluid collections includes acute peripancreatic fluid collection, pseudocyst, acute necrotic collection, walled-off necrosis, and postnecrosectomy pseudocyst. ^{2,7} Acute peripancreatic fluid collections typically do not have connection with the ductal system and more than 50% to 70% resolve spontaneously within a few weeks. ¹⁵ The other 30% to 50% may develop into pseudocysts. Because they frequently resolve spontaneously and usually remain sterile, observation is the treatment of choice. ^{5,15}

An acute necrotic collection contains fluid and necrotic tissue and is found less than 4 weeks following an episode of necrotizing pancreatitis. ^{2,7} If this entity is mistaken for a pseudocyst there is near universal failure of any drainage procedure. ³ Instead, treatment should consist of observation and medical management unless the patient develops symptoms or the fluid collection becomes infected, in which case intervention is required. ³ In cases that require intervention, open surgical drainage is probably the gold standard, ³ although many of the same arguments that are applied to the treatment of pancreatic necrosis apply.

PSEUDOCYSTS

Pseudocysts are defined by the International Symposium on Acute Pancreatitis as "a collection of pancreatic juice enclosed by a wall of fibrous or granulation tissue which arises as a consequence of acute pancreatitis, trauma, or chronic pancreatitis." The incidence is 5% to 16% of all new cases of acute pancreatitis. They present greater than 4 weeks after the onset of symptoms and occur most commonly with interstitial edematous pancreatitis. Pseudocysts are the result of direct leakage of pancreatic juice. The communication with the secretory space may spontaneously seal or remain patent, with the latter having a much lower incidence of spontaneous resolution. The diagnosis of pseudocyst is confirmed with contrast-enhanced CT.

Although it is not essential to delineate communication to the ductal system to secure a diagnosis, it may be helpful in predicting spontaneous resolution and in helping to direct therapy. ^{2,33} However, if contrast via endoscopic retrograde cholangiopancreatography (ERCP) demonstrates a communication, some investigators suggest drainage should be performed within 24 hours to prevent bacterial contamination of the pseudocyst. ^{17,34}

Earlier work by Bradley and colleagues, ³⁵ published in 1979, showed that 46% of all pseudocysts will develop major complications and only 20% will spontaneously resolve. Therefore, the advice was to intervene on pseudocysts that were larger than 6 cm or those that persisted for more than 6 weeks. Mortality rates of 7% and morbidity rates of greater than 40% were reported with these operations. ^{33,35} It now seems that half of patients who have asymptomatic pseudocysts, regardless of size, will probably resolve spontaneously. Acute intervention is generally reserved for those patients who are symptomatic, have signs of infection, develop complications, show increasing size of the pseudocyst, or in whom it is not possible to differentiate a pseudocyst from a cystic neoplasm. In the asymptomatic patient, regardless of pseudocyst size, it is

recommended to postpone intervention for at least 6 weeks to monitor for signs of resolution and to allow the wall to mature. 3,36

Internal Drainage Procedures

The goal of drainage procedures, simply put, is to allow for controlled drainage of pancreatic juice into the lumen of the alimentary tract (preferably proximal) or to an external source (controlled pancreatic fistula).

Open internal drainage procedures were once first-line treatment of pancreatic pseudocysts.² They still work quite well in many situations in which other techniques fall short. Usually, they are now reserved for cases that fail less invasive options or in cases of complete obstruction of the main pancreatic duct, ductal changes associated with chronic pancreatitis, strictures, stones, or giant or multiple pseudocysts.³³ Options include Roux-en-Y pseudocyst jejunostomy, pseudocystgastrostomy, or pseudocyst duodenostomy.² Overall, mortality rates are reported to be 7%, whereas recurrence rates range from 5% to 20%³ and complication rates are 12% to 24%.^{2,4} However, the corresponding author thinks many practitioners would consider those rates to be on the high end for well-selected patients.

Percutaneous internal drainage can be done with the assistance of either ultrasound or CT guidance. Most commonly, the anterior abdominal wall, the anterior gastric wall, and the posterior gastric wall are punctured with a needle to gain entry into the pseudocyst. This is then exchanged over a guidewire for a double-J, 5F to10F catheter that drains the pseudocyst into the stomach.⁴ Because treatment failures are higher, percutaneous internal drainage is reserved for patients who are poor candidates for operative or other more definitive treatment or as a temporary measure in patients with infected pseudocysts.⁴ The risk of developing pancreaticocutaneous fistulae and drain tract or pseudocyst infections is increased with percutaneous techniques compared with open internal drainage.⁴

Endoscopic internal drainage has excellent outcomes that, in many cases, are similar to operative management but with perhaps less procedural morbidity. However, there are no randomized controlled trials directly comparing the two. Despite the lack of comparative trials, seven published guidelines recommend endoscopic therapy as the initial treatment of uncomplicated pseudocysts. The endoscopic approach may also require multiple endoscopic attempts and repeated imaging studies, which will influence comparative cost and risk.

Endoscopic drainage seems to be associated with a learning curve as evidenced by increasing rates of pseudocyst resolution from 45% to 93% with endoscopists who have performed more than 20 procedures. ^{2,34} Transpapillary access is used when there is demonstrable ductal communication with the pseudocyst.⁴ A 5F or 7F stent is then placed into the pancreatic duct and either directly into the cyst or used to bridge the leak.² This is left in place until there is CT evidence of resolution.³ Biliary and pancreatic sphincterotomies are also performed.⁴ If no ductal communication can be demonstrated, transluminal access may be used via a transgastric or medial duodenal wall approach.32 EUS may be helpful when there is no visible bulge into the lumen. EUS may also identify the distance between the viscera and the pseudocyst, intervening vessels, the thickness of the cyst wall, or the presence of necrosis, which may alter management in up to 20% of patients. Once the pseudocyst is identified, contrast injection is performed to confirm placement of the needle and enterotomy and pseudocystotomy are made to allow passage of a guidewire followed by placement of one or two 7F to 10F stents. 4 Stents are left in place for several months or until radiographic resolution.^{3,4} Reported success rates vary from 82% to 100%² with decreasing success rates as the pseudocyst location moves from the head to the body to the tail of

the pancreas.⁴ Recurrence rates range from 5% to 20%.⁴ Overall complication rates are as high as 34% and consist of hemorrhage, perforation, infection, stent migration or occlusion, or recurrent pancreatitis.^{2,4}

Disadvantages of this approach are that the stents are fairly small, thus the cyst must be minimally loculated, nonviscous, and contain little or no debris. Also, in cases of diagnostic insecurity, there is limited ability to obtain a cyst wall biopsy. ^{4,17} Necrosis is associated with a 50% failure rate and increased rates of recurrence and complications. Treatment in patients with necrosis should probably be surgical. ⁴

Laparoscopic approaches seem, thus far, to have similar results to open procedures. However, there are no prospective controlled trials comparing the two. In reviewing all large series (n \geq 10) of laparoscopic internal drainage (total of 89 subjects), 10.1% required conversion to an open procedure, 6.7% had bleeding complications, 5.6% developed sepsis, 3.4% had a recurrence, 4.5% required an additional procedure, and the mortality rate was 1.1%.

Pseudocystgastrostomy may be created via either an anterior or a posterior approach.⁴ For either open or videoscopic approach, the techniques for pseudocystgastrostomy are similar. The anterior approach involves making an anterior gastrotomy, then identifying the point of maximal bulge on the posterior wall signifying the pseudocyst location. A needle is then used to confirm this. A biopsy of the pseudocyst wall may be obtained to rule out malignancy when clinical suspicion is present. Pseudocystgastrostomy is made with either a stapler or sutures followed by closure of the anterior gastrotomy. A similar anterior approach has been described using two to three balloon-tipped trocars placed into the peritoneum and then directly through the anterior stomach via separate 10 mm gastrotomies. The balloons are then inflated and the anterior stomach wall is compressed to the abdominal wall such that the remainder of the procedure can be performed without traversing the peritoneum. A minilaparoscopic technique uses 2 mm instruments placed directly into the gastric lumen using endoscopic guidance, thus avoiding the need for pneumoperitoneum. However, 10% require conversion due to inability to localize the pseudocyst.⁴

The posterior or lesser sac approach was developed to avoid the technical challenges of the anterior approach and to avoid an additional gastrotomy. The lesser sac is accessed by dividing the greater omentum along the greater curve of the stomach. The proponents of this technique suggest there is better visualization and improved ability to locate the pseudocyst without the need for a visible bulge in the gastric wall. In addition, there is less bleeding and a larger anastomosis resulting in improved patency rates. The corresponding author does not recommend this technique because the inflammatory process frequently obliterates the lesser sac and this approach may convert a contained situation into a diffuse intraabdominal process.

Drainage via pseudocyst jejunostomy may be preferable if the pseudocyst is not in close proximity to the stomach or duodenum. The pseudocyst may be anastomosed to a Roux-en-Y jejunal limb. The pseudocyst is entered via the transverse mesocolon or through the gastrocolic ligament. One small study evaluating eight subjects using this approach demonstrated no conversions, complications, or recurrences at 2 years.⁴

External Drainage Procedures

External drainage may be necessary when there is evidence of gross infection of the pseudocyst or in situations in which the pseudocyst wall is too thin to use for a secure anastomosis.¹⁷ These procedures generally result in a pancreatic fistula, which is fine as long as it is controlled.

Open external drainage has become extremely uncommon due to high morbidity but may be considered in complex situations. Percutaneous drainage can be performed

via a retroperitoneal or transperitoneal approach.³¹ A transgastric approach may also be used; however, it is associated with an increased risk for infecting a previously sterile fluid collection; therefore, it should only be considered when other options are less available. Aspiration alone has recurrence rates of greater than 70% and should not be used.³¹ Drains are typically left in place for 3 weeks but can be required for as long as several months or longer if ductal communication is present.¹⁷ Once drain output has decreased to a low volume per day and imaging shows no persistent fluid collection, sinography may be performed to evaluate for resolution of the pseudocyst, persistence of ductal communication, retained debris, or presence of a pseudocyst enteric fistula.³¹ Potential complications of percutaneous drainage include bleeding (1%–2%), transversal of the pleural space or other viscera (1%–2%), and chronic pancreaticocutaneous fistula (5%).³¹ The risk of developing a pancreaticocutaneous fistula increases when persistent ductal communication is present.³ Reported success rates are variable, ranging from 60% to 100%.^{4,31,34}

D'Egidio and Schein³⁹ evaluated the success of percutaneous drainage based on a proposed ductal classification system. Type 1 ducts have normal anatomy and rare communication with the pseudocyst, type 2 ducts have a diseased pancreatic duct without stricture but often with duct-pseudocyst communication, and type 3 ducts have stricture and pseudocyst communication. In their study, subjects with type 1 ducts showed resolution with drainage in all 13 cases, those with type 2 ducts achieved resolution in 9 of 10 cases, and those with type 3 ducts were excluded. Zhang and colleagues⁴⁰ also reported outcomes based on D'Egidio classifications and found a success rate of 82% for type 1 ducts, 60% for type 2, and again excluded type 3 ducts.

Most published literature comparing percutaneous external drainage to open external drainage report a higher mortality, longer length of stay, and higher incidence of complications with percutaneous drainage. The only prospective study comparing percutaneous with open drainage found resolution rates of 93% with open drainage and 75% with percutaneous drainage. The data seem to suggest that percutaneous drainage should be reserved for patients with normal ducts who are not candidates for operative management or as a temporizing measure until more definitive measures can be used. It is also a valid option for infected pseudocysts if there is no associated necrosis. 2,15,33,37

Resection

Resection of the pseudocyst with associated segmental pancreatectomy should be performed in cases in which it is impossible to differentiate a pseudocyst from a malignant cystic neoplasm³³ or for patients in whom the acute inflammatory process has long since resolved and the morbidity of the pancreatic resection is less than that of a drainage procedure. Most frequently, this is in the case of pseudocysts located in the very distal portion of the tail or in patients who develop a disconnected pancreatic tail syndrome.^{17,33} If the pseudocyst is near the spleen or involves the splenic vessels, it may be necessary to perform concomitant splenectomy.³³

WALLED-OFF NECROSIS

Walled-off necrosis differs from pancreatic necrosis because it is typically associated with a limited degree of necrosis, a more clinically benign course, and mortality rates of 5%. It is also rare, comprising less than 5% of all peripancreatic fluid collections.^{2,17} Walled-off necrosis represents the mature encapsulated form of an acute necrotic collection that usually develops more than 4 weeks after the initial episode of acute necrotizing pancreatitis. Walled-off necrosis now includes the former entities described as organized pancreatic necrosis, pancreatic pseudocyst with necrosis, pancreatic

sequestrum, necroma, and subacute pancreatic necrosis.⁶ The diagnosis is made by CT demonstrating an encapsulated peripancreatic collection of solid and fluid debris in the correct clinical context and infection is confirmed with fine-needle aspiration demonstrating organisms.⁸ Walled-off necrosis can be sterile or infected and may or may not be in communication with the ductal system.² The treatment includes drainage and antibiotics until radiographic evidence of resolution.^{2,3,8} Open drainage or debridement has been the most common form of treatment. The addition of continuous post-operative lavage and open packing with debridement repeated every 2 to 4 days has decreased mortality in some cases.¹⁵ If the necrosis is well delineated, it may be possible to perform only one operation for debridement with drains for postoperative irrigation.¹⁵ The optimal timing for open drainage is 3 to 4 weeks after the onset to allow for demarcation and additional improvement in mortality,² as well as to decrease the need for subsequent operation.

Percutaneous drainage may be acceptable when there is no surrounding necrosis, the collection is well localized and there is no communication with the ductal system. The success rate with this approach approximates 90% with appropriate patient selection. It can also be used as a bridge to open drainage if the fluid collection has been present fewer than 4 weeks since the onset of symptoms.^{2,17} Laparoscopic and endoscopic techniques may be equally as effective as surgery.²

DUCTAL DISRUPTIONS

In patients in whom main pancreatic ductal disruption is suspected, it is imperative to clearly define the ductal anatomy to the best degree possible. ⁴¹ Imaging with CT may suggest the site of the leak based on fluid location and may also yield information about ductal dilatation that may be important in determining optimal management.²

Significant ductal disruptions will result in internal fistula, external fistula, pancreatic pleural effusion, pancreatic ascites, or disconnected pancreatic tail syndrome. Although these sound like a collection of entities, they are more likely just a continuum of the same pathophysiologic process with different resolutions.

Internal fistulae are uncommon and are the result of ductal disruptions that are not contained by the inflammatory response. The location of the ductal disruption corresponds to the location of the fluid collection with anterior ductal disruptions resulting in pancreatic ascites and posterior disruptions resulting in pancreatic pleural effusions.⁴¹ Once it is confirmed that a large fluid collection in either the chest or peritoneal cavity is pancreatic enzyme rich, the diagnosis is secure. Management begins with percutaneous external drainage to covert the clinical picture to that of a controlled fistula. This alone may result in spontaneous closure in 70% to 82% of cases. 41 It can also serve as a temporary measure until later definitive management can be done. 12,41 After percutaneous drainage, endoscopic retrograde pancreatography (ERP) with papillary decompression via sphincterotomy or transpapillary stenting should be performed to decrease the resistance to flow of pancreatic juice into the duodenum.⁴¹ More definitive operative intervention will most likely be required in situations such as the inability to cannulate the duct, multiple ductal strictures are present, the duct has a large defect, or there is a disconnected duct. 41 Dilated ducts (main pancreatic duct measuring greater than 7 mm) may be treated with lateral pancreaticojejunostomy with relatively low morbidity and mortality rates.⁴¹

If the ducts are small, the specific site of duct disruption becomes more important in directing management.⁴¹ If the disruption is in the tail, a caudal pancreatectomy may be a better solution.⁴¹ If the disruption is in the body, a distal pancreatectomy plus or minus concomitant splenectomy may be performed.⁴¹

If the ductal disruption is in the head of the pancreas, matters become more challenging. The solution may require a pancreatic head resection or operative conversion to internal drainage. The reported success rates range from 77% to 100% and long-term failure rates are due to obliteration of the fistula tract.⁴¹ All of these management options can be very challenging and correct choice of procedure, as well as correct choice of timing of intervention, are mandatory for success. Very experienced teams should do all of these operations.

External fistulae are typically the result of a percutaneous drain placed for pseudocyst treatment. The chance of developing this complication is greater if there is also a stricture or obstruction of the main pancreatic duct resulting in ductal hypertension. Other causes include previous operative therapy such as necrosectomy, pancreatic resection, or pancreatic injury during splenectomy or nephrectomy. Treatment involves ERP with sphincterotomy and stenting, which results in fistula closure in 40% to 90%. The sphince of the results in fistula closure in 40% to 90%.

The location of the leak may give insight into the chance of closure without operative intervention. Howard and colleagues⁴⁴ found spontaneous closure with conservative therapy in 87% of postoperative side fistulas compared with 53% of inflammatory side fistulas and 0% of end fistulas. Better results are seen in side fistulas that can be bridged with a stent, with 92% to 100% resolving without an operation.⁴² At least 6 weeks should be allowed for ductal disruptions to heal before any further intervention is undertaken unless the main pancreatic duct is dilated, in which case the fistula is unlikely to heal and operative intervention is the treatment of choice.⁴¹ The operation is determined by the location of the ductal disruption, as stated above. Despite operative therapy, recurrence of the pancreaticocutaneous fistula occurs in 23% of cases.⁹ A more detailed description of the management of pancreatic fistulae is found by Hardacre and colleagues elsewhere in this issue.

Disconnected pancreatic tail syndrome is perhaps the most common variant of an inflammatory fistula and develops in 16% of patients with a pancreatic fluid collection and/or fistula. ¹² A high index of suspicion should be present in cases of recurrent pseudocysts or persistent fistulae. ¹² Initial work-up should include exclusion of vascular abnormalities such as pseudoaneurysm or splenic vein thrombosis. ⁹ Management options are similar to those mentioned above, including long-term stenting, endoscopic transluminal drainage, transpapillary drainage, surgical drainage via pseudocystgastrostomy, pseudocyst enterostomy, or side-to-side pancreaticojejunostomy of the upstream pancreas to a Roux-en-y limb, or resection, including left-sided pancreatectomy with or without splenectomy and the Whipple procedure. ^{9,10,12} Endoscopic drainage is preferred initially because recurrence rates are high but equal to operative drainage results. ⁹

ASSOCIATED OPERATIONS AND PROCEDURES Biliary Procedures

Early studies demonstrated that ERCP with endoscopic sphincterotomy (ES) may reduce morbidity in acute pancreatitis but has no effect on mortality. This reduction in morbidity is due to decreasing the impact of biliary sepsis and not because removing the stone decreases the evolution of pancreatitis. ¹⁸ In 2002, the International Association of Pancreatology (IAP) released evidence-based guidelines regarding ERCP and ES in the management of gallstone pancreatitis, stating that these interventions are indicated in cases of obstructive jaundice and cholangitis. ¹⁶ The 2007 American Gastroenterology Association guidelines state that ERCP and ES should only be used for treatment of cholangitis. ¹⁶

The role of prophylactic biliary procedures is less clear. In patients who develop pancreatitis as the result of gallstones, the source of the gallstones must be addressed given an overall recurrence rate of biliary pancreatitis of 29% to 63%. 16 However, the debate continues in regard to the optimal timing at which cholecystectomy is performed. An early study by Kelly and Wagner, in 1988, assigned 165 subjects to cholecystectomy before or after 48 hours. They found a much higher morbidity and mortality in the early cholecystectomy group compared with the late group (83% vs 48% and 18% vs 12%, respectively). 45 In 2012, van Baal and colleagues 46 conducted a systematic review that included 998 subjects, 48% of whom had a cholecystectomy during their index admission and 52% had an interval cholecystectomy at a median of 40 days. They found a statistically significant increase in complications in the interval cholecystectomy group, with 18% requiring readmission, 8% developing recurrent biliary pancreatitis, 3% developing acute cholecystitis, and 7% requiring readmission for biliary colic. This is in contrast to those who had a cholecystectomy during their index hospitalization, in which none suffered any recurrent biliary events. They also found the overall conversion from laparoscopic to open to be 7%, with no significant difference between the groups. Sinha and colleagues⁴⁷ concluded that interval cholecystectomy resulted in more frequent difficult dissection of the Calot triangle compared with index cholecystectomy in 42% versus 12% cases, respectively. The main problem with the published reports on this topic is the variation in practice and the extreme range of timing options evaluated.

The IAP recommends that, in mild gallstone pancreatitis, cholecystectomy should be performed as soon as the patient recovers and, ideally, this should be done in the same hospitalization. In severe cases, they recommend delaying surgery until there is sufficient resolution of the inflammation and clinical recovery. In addition, they state that to reduce the risk of recurrence endoscopic sphincterotomy is an alternative to cholecystectomy in patients who are not fit for surgery. ¹⁶

Vascular Complications

Vascular complications affect approximately 2.4% to 10% of patients with pancreatitis and include pseudoaneurysms and bleeding from erosion into an artery. ⁴⁸ The most frequently involved vessels are the splenic, gastroduodenal, pancreaticoduodenal, and the left gastric arteries. ⁴⁸ These may rupture into the peritoneal cavity or into the gastrointestinal tract, which is accompanied by mortality rates of approximately 50%. ⁴⁸ In the past, operative management was the mainstay of therapy though fraught with great difficulty. Advances in interventional radiographic techniques have made embolization the preferred management. ⁴⁸ Radiological management has been proven to be effective and reliable in both elective and emergency treatment of arterial complications of pancreatitis. ⁴⁸ In patients with hemorrhage into pseudocysts, a combined approach should be used that begins with hemorrhage control by angiographic means followed by treatment of the pseudocyst as required. ⁴⁸

Colonic Complications

Colonic complications of acute pancreatitis are uncommon, occurring in approximately 1% of cases, but can be associated with a very poor prognosis despite surgical intervention. ⁴⁹ The most common manifestation is colonic ileus, which is not life threatening. Sustained ileus may, however, necessitate the need for prolonged parenteral nutritional support, which is attended by potential complication. The more grave forms of colonic complications are obstruction, necrosis, perforation, and fistulae. Obstruction may occur from either extrinsic compression by a pancreatic inflammatory mass or fluid collection or from pericolonic fibrosis. The most common site of stenosis is

the splenic flexure, which may be due relatively poor arterial supply and close proximity to the tail of the pancreas. Colonic necrosis and perforation are potentially lethal complications, with an overall mortality of 58%. ⁴⁹ The most common locations are the transverse colon and splenic flexure with pancreatic necrosis and abscess with vascular compromise being the most common inciting events. The surgical management remains difficult and includes resection of the involved colon and exteriorization with either a proximal colostomy or ileostomy and a distal mucous fistula. The decision to resect is based on surgical experience and adherence to fundamental surgical principles because there are few guidelines to assist with management decisions. ⁴⁹

SUMMARY

The operative management of acute pancreatitis is focused on managing the acute complications, the long-term sequelae, or the prevention of recurrent pancreatitis. Using the least amount of intervention to achieve the stated goals has always been the case. However, the evolution of videoscopic and endoscopic techniques have greatly expanded the tools available. Patience, vigilance, expertise, and judgment, and an ability to be humbled are necessary for the successful practitioner who manages patients with severe pancreatitis.

REFERENCES

- 1. Baron TH, Morgan DE. Acute necrotizing pancreatitis. N Engl J Med 1999; 340(18):1412-7.
- 2. Brun A, Agarwal N, Pitchumoni CS. Fluid collections in and around the pancreas in acute pancreatitis. J Clin Gastroenterol 2011;45(7):614–23.
- 3. Baron TH, Morgan DE. The diagnosis and management of fluid collections associated with the pancreas. Am J Med 1997;102:555–63.
- 4. Berman S, Melvin S. Operative and nonoperative management of pancreatic pseudocysts. Surg Clin North Am 2007;87:1447–60.
- 5. Bollen TL. Imaging of acute pancreatitis: update of the revised Atlanta classification. Radiol Clin North Am 2012;50:429–45.
- 6. Bollen TL, Besselink MG, van Santvoort HC, et al. Toward an update of the Atlanta classification on acute pancreatitis. Pancreas 2007;35(2):107–11.
- 7. Bollen TL, van Santvoort HC, Besselink MG, et al. Update on acute pancreatitis: ultrasound, computed tomography, and magnetic resonance imaging features. Semin Ultrasound CT MR 2007;28:371–83.
- 8. Stamatakos M, Stefanaki C, Kontzoglou K, et al. Walled-off pancreatic necrosis. World J Gastroenterol 2010;16(14):1707–12.
- 9. Lawrence C, Howell DA, Stefan AM, et al. Disconnected pancreatic tail syndrome: potential for endoscopic therapy and results of long-term follow-up. Gastrointest Endosc 2008;67(4):673–9.
- Traverso WL, Kozarek RA. Interventional management of peripancreatic fluid collections. Surg Clin North Am 1999;79(4):745–57.
- 11. Traverso LW, Kozarek RA. Pancreatic necrosectomy: definitions and technique. J Gastrointest Surg 2005;9(3):436–9.
- Solanki R, Koganti SB, Bheerappa N, et al. Disconnected duct syndrome: refractory inflammatory external fistula following percutaneous drainage of an infected peripancreatic fluid collection. A case report and review of the literature. JOP 2011;12(2):177–80.
- 13. Cappell MS. Acute pancreatitis: etiology, clinical presentation, diagnosis, and therapy. Med Clin North Am 2008;92:889–923.

- 14. Schneider L, Buchler MW, Werner J. Acute pancreatitis with an emphasis on infection. Infect Dis Clin North Am 2010;24:921–41.
- 15. Farthmann EH, Lausen M, Schoffel U. Indications for surgical treatment of acute pancreatitis. Hepatogastroenterology 1993;40:556–62.
- 16. Uhl W, Warshaw A, Imrie C, et al. IAP guidelines for the surgical management of acute pancreatitis. Pancreatology 2002;2:565–73.
- 17. Tsiotos GG, Sarr MG. Management of fluid collections and necrosis in acute pancreatitis. Curr Gastroenterol Rep 1999;1:139–44.
- 18. Clancy T, Ashley S. Current management of necrotizing pancreatitis. Adv Surg 2002;36:103–21.
- 19. Voermans RP, Veldkamp MC, Rauws EA, et al. Endoscopic transmural debridement of symptomatic organized pancreatic necrosis. Gastrointest Endosc 2007;66(5):909–16.
- 20. Warner EA, Ben-David K, Cendan JC. Laparoscopic pancreatic surgery: what now and what next? Curr Gastroenterol Rep 2009;11:128–33.
- van Santvoort HC, Besselink MG, Bollen TL, et al. Casematched comparison of the retroperitoneal approach with laparotomy for necrotizing pancreatitis. World J Surg 2007;31:1635–42.
- 22. Van Santvoort HC, Besselink MG, Bakker OJ, et al. A step-up approach or open necrosectomy for necrotizing pancreatitis. N Engl J Med 2010;362(16):1491–502.
- 23. Freeny PC, Hauptmann E, Althaus SJ, et al. Percutaneous CT-guided catheter drainage of infected acute necrotizing pancreatitis: techniques and results. Am J Roentgenol 1998;170:969.
- 24. Echenique AM, Sleeman D, Yrizarry J, et al. Percutaneous catheter-directed debridement of infected pancreatic necrosis: results in 20 patients. J Vasc Interv Radiol 1998;9:565.
- 25. Ross A, Gluck M, Irani S, et al. Combined endoscopic and percutaneous drainage of organized pancreatic necrosis. Gastrointest Endosc 2010;71(1):79–84.
- 26. Friedland S, Kaltenbach T, Sugimoto M, et al. Endoscopic necrosectomy of organized pancreatic necrosis: a currently practiced NOTES procedure. J Hepatobiliary Pancreat Surg 2009;16:266–9.
- 27. Ho HS, Frey CF. Gastrointestinal and pancreatic complications associated with severe pancreatitis. Arch Surg 1995;130:817–22.
- 28. Baron TH, Morgan DE. Organized pancreatic necrosis: definition, diagnosis, and management. Gastroenterol Int 1997;10:167–78.
- 29. Seifert H, Biermer M, Schmitt W, et al. Transluminal endoscopic necrosectomy after acute pancreatitis: a multicenter study with long-term follow-up (The GEPARD Study). Gut 2009;58:1260.
- 30. Connor S, Alexakis N, Raraty MG, et al. Early and late complications after pancreatic necrosectomy. Surgery 2005;137:499–505.
- 31. Neff R. Pancreatic pseudocysts and fluid collections—percutaneous approaches. Surg Clin North Am 2001;81(2):399–403.
- 32. Kozarek RA, Ball TJ, Patterson DJ, et al. Endoscopic transpapillary therapy for disrupted pancreatic duct and peripancreatic fluid collections. Gastroenterology 1991;100:1362–70.
- 33. Behrns KE, Ben-David K. Surgical therapy of pancreatic pseudocysts. J Gastrointest Surg 2008;12:2231–9.
- 34. Cannon JW, Callery MP, Vollmer CM. Diagnosis and management of pancreatic pseudocysts: what is the evidence? J Am Coll Surg 2009;209(3):385–93.
- 35. Bradley EL, Clements JL, Gonzalez AC. The natural history of pancreatic pseudocysts: a unified concept of management. Am J Surg 1979;137(1):135–41.

- 36. Nealon WH, Bawduniak J, Walser EM. Appropriate timing of cholecystectomy in patients who present with moderate to severe gallstone-associated acute pancreatitis with peripancreatic fluid collections. Ann Surg 2004:239(6):741–51.
- 37. Loveday BP, Mittal A, Phillips A, et al. Minimally invasive management of pancreatic abscess, pseudocyst, and necrosis: a systematic review of current guidelines. World J Surg 2008;32:2382–94.
- 38. Martin RF, Marion MD. Resectional therapy for chronic pancreatitis. Surg Clin North Am 2007;87:1461–75.
- 39. D'Egidio A, Schein M. Percutaneous drainage of pancreatic pseudocysts: A prospective study. World J Surg 1991;16:141.
- 40. Zhang AB, Zheng SS. Treatment of pancreatic pseudocysts in line with D'Egidio's classification. World J Gastroenterol 2005;11(5):729–32.
- 41. Morgan KA, Adams DB. Management of internal and external pancreatic fistulas. Surg Clin North Am 2007;87:1503–13.
- 42. Rana SS, Bhasin DK, Nanda M, et al. Endoscopic transpapillary drainage for external fistulas developing after surgical or radiological pancreatic interventions. J Gastroenterol Hepatol 2010;25:1087–92.
- 43. Ranson JH. The role of surgery in the management of acute pancreatitis. Ann Surg 1990;211(4):382–93.
- 44. Howard TJ, Stonerock CE, Sarkar J, et al. Contemporary treatment strategies for external pancreatic fistulas. Surgery 1998;124:627–32.
- 45. Kelly TR, Wagner DS. Gallstone pancreatitis: A prospective randomized trial of the timing of surgery. Surgery 1988;104:600–5.
- 46. van Baal M, Besselink MG, Bakker OJ, et al. Timing of cholecystectomy after mild biliary pancreatitis. Ann Surg 2012;255(5):860–6.
- 47. Sinha R. Early laparoscopic cholecystectomy in acute biliary pancreatitis: the optimal choice? HPB (Oxford) 2008;10:332–5.
- 48. Sawlani V, Phadke RV, Baijal SS, et al. Arterial complications of pancreatitis and their radiological management. Australas Radiol 1996;40:381–6.
- 49. Aldridge MC, Francis ND, Glazer G, et al. Colonic complications of severe acute pancreatitis. Br J Surg 1989;76:362–7.