Severe acute pancreatitis for the acute care surgeon

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Acute pancreatitis (AP) remains an extremely common yet underappreciated disease. Approximately 240,000 new cases per year are diagnosed within the United States alone.[1] AP represents the third most common gastrointestinal disease overall and requires more than 2 billion per year in diagnostic and treatment costs.[1] Among all cases of AP, 15% to 20% manifest the severe necrotizing variant with a classically reported mortality rate of 20%. For comparison, these demographics are within the realm of both breast (178,480 cases per year; mortality, 22%) and prostate (218,890 cases per year; mortality, 12%) cancer.[2] As traditionally noted, the most common causes of AP include chronic alcohol ingestion, biliary sources, and a multitude of less frequent etiologies (calcium disturbances, autoimmune genetics, drug associations, lipid alterations). It should be noted that even in patients labeled with a diagnosis of "idiopathic" pancreatitis, the most common cause (44%) remains a biliary source.[3] This is true even in the presence of a "negative" ultrasound evaluation result denying the presence of either cholelithiasis or biliary sludge. As a result, a scheduled cholecystectomy is recommended for each patient who carries a diagnosis of idiopathic pancreatitis to reduce the risk of recurrent attacks.

It is clear that the most severe variants of AP challenge our clinical acumen, stress our therapeutic talents, and strain our ability to work together as an acute care surgery team. More specifically, advanced organ support strategies (pulmonary, renal, cardiovascular), sepsis control techniques, and nutritional optimization algorithms are required to salvage patients with the most severe cases. The purpose of this review was therefore to deliver a high-level, acute care surgical commentary focused on the current optimal management strategies surrounding severe acute pancreatitis (Table 1). This objective has been achieved by discussing the high-volume experiences of the coauthors supplemented by both recent and historically relevant peer-reviewed literature.

PATIENTS AND METHODS

The content and conclusions described within this article are composed from a systematic literature review on severe acute pancreatitis. The sources of this material include a long history of peer-reviewed publications (PubMed and EMBASE), textbooks outlining technique, and many of the authors' own extensive experiences at high-volume pancreatitis institutions.

TABLE 1. Topics Addressed in This Systematic Review of Severe AP

1. Population based impact
2. Modern terminology and lexicon
3. Classification and scoring systems
4. Prognosis
5. Critical care
   a. Nutrition
   b. Organ support
   c. Sepsis and antimicrobial therapy
6. Optimal timing of intervention
7. Technical pearls for therapeutic procedures

TABLE 2. Modern Lexicon of Terminology for Severe AP

<4 wk after onset of pancreatitis:
- Acute peripancreatic fluid collection
  - Sterile
  - Infected
- Postnecrotic pancreatic/peripancreatic fluid collection
  - Sterile
  - Infected

>4 wk after onset of pancreatitis
- Pancreatic pseudocyst (high amylase/lipase)
  - Sterile
  - Infected
- WOPN (may or may not have high amylase/lipase)
  - Sterile
  - Infected

Given the tremendous shift in both the timing and format of operative intervention for necrotizing pancreatitis during the past decade, the specific aim of this review was to update the acute care surgeon with the latest thoughts regarding the diagnosis, treatment, and outcome of patients with severe acute pancreatitis. Considering the broad knowledge required of the acute care surgery teams managing AP and the high incidence of this disease on a population basis, it is clear that a thorough and modern understanding of severe acute pancreatitis that combines best evidence and hard won experience is crucial to all acute care surgery programs.

Terminology and Lexicon

The terminology surrounding AP and its associated complications has been extremely varied over the years.[4] In 1993, the Atlanta Symposium redefined a common lexicon while jettisoning multiple terms now considered obsolete[5] including phlegmon, infected pseudocyst, hemorrhagic pancreatitis and "persistent" AP to name a few (Table 2). In 2007, an updated working group further clarified this terminology with a specific reference to the pathophysiologic processes that begin to gain prominence and influence the trajectory of AP beyond the 4 weeks of disease onset.[6] As a result, it has become clear that entities such as walled-off pancreatic necrosis (WOPN) are much more common than true pancreatic pseudocysts. The 2012 publication from this working group further delineates local complications (sterile vs. infected [peri]pancreatitis) from systemic ones (transient vs. persistent organ failure) and compartmentalizes the spectrum of AP into mild, moderate, severe, and critical (Table 3).[6] Close attention to these definitions can enable clinical teams to better understand complex disease processes, prioritize diagnostic approaches, select appropriate therapeutic algorithms, and

TABLE 3. Determinant-Based Classification of AP Severity

	Mild AP	Moderate AP	Severe AP	Critical AP
(Peri)pancreatic necrosis	No	Sterile	Infected	Infected
	AND	AND/OR	OR	AND
Organ failure	No	Transient	Persistent	Persistent

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identify opportunities for subspecialty consultation. A notable example of this concept is the strong utility of endoscopic drainage for a rare true pseudocyst (i.e., fluid only) compared with predictable failure when using this technique to attempt to drain WOPN (i.e., thick solid material).

Critical Care

The necessary critical care for patients with severe or critical AP encompasses all systems. Pulmonary support, shock resuscitation, prevention of sepsis, optimization of nutritional support, and close attention to pain, agitation, and delirium are all key priorities. As with many intra-abdominal surgical emergencies for the acute care surgeon, timely and appropriate resuscitation remains a mainstay of therapeutic excellence. Underresuscitation can lead to mesenteric ischemia, acute kidney injury, and worsening multiorgan failure. Excessive crystalloid fluid administration leads to increased reperfusion injury, leukocyte adhesion, and inflammation and as a result worsens acute lung injury, systemic inflammatory responses, coagulopathies, acid-base disturbances and finally multiorgan failure.[7,8] Overresuscitation may also lead to both difficulty obtaining definitive abdominal wall fascial closure because of visceral and abdominal wall edema as well as secondary abdominal compartment syndrome (ACS).[9] While the specific incidence of secondary ACS in the context of severe and/or critical AP is debated,[10] it is clear that monitoring intra-abdominal pressures (e.g., three-way Foley catheter) is a helpful practice within the critical care suite.[11] Surgical teams must balance the competing risks of underresuscitation and overresuscitation: precise resuscitation requires close attention to physiologic data and indicators of organ hypoperfusion (lactate levels, central venous oxygenation) as well as close observation of intravascular volume and cardiac function through tools such as the point-of-care echocardiography.

Although the thoughtful use of decompressive laparotomy for the management of refractory ACS and damage-control laparotomy in the instance of extreme intraoperative physiologic instability are powerful adjuncts in the management of AP, it must be noted that the use of open abdomens for persistent/recurrent pancreatic debridements (i.e., pancreatic stomas) has become a relic from the past, supplanted by the removal of pancreatic necrosum in one stage at the optimal moment. The disappearance of the pancreatic stoma is a reflection of an improved understanding of the cadence of pancreatic necrosis, which favors delaying surgical intervention beyond the 28-day period from the onset of the disease.

Numerous pancreatitis-specific trials have confirmed the importance of enteral nutrition for patients with severe AP.[12,13] Enteral therapy results in fewer infections and a shorter hospital stay. A systematic review that included 348 patients from 8 trials also showed that enteral nutrition decreased the risk of death (odds ratio [OR], 0.50; 95% confidence interval [CI], 0.28 to 0.91), multiple-organ failure (OR, 0.55; 95% CI, 0.37 to 0.81), systemic infection (OR, 0.39; 95% CI, 0.23–0.65), operative interventions (OR, 0.44; 95% CI, 0.29–0.67), local septic complications (OR, 0.74; 95% CI, 0.40–1.35), and length of hospital stay (reduced by 2.4 days).[13] Most importantly, in patients with severe AP, enteral nutrition decreased the risk of death (RR, 0.18; 95% CI, 0.06–0.58) and multiple-organ failure (RR, 0.46; 95% CI, 0.16–1.29), suggesting that patients should receive enteral over parenteral nutrition.[13] It is also relevant to note that the traditional dogma mandating nil per os status in patients with AP because of a fear of pancreatic stimulation is currently believed to be unfounded. High-volume observations and numerous studies now confirm that there is virtually no way of predicting which patients will tolerate full oral intake versus those who will require an alternative route of nutritional access. A randomized trial of early enteral nutrition based on hunger alone in patients with severe acute pancreatitis showed that oral feeding is extremely successful and shortens the length of hospital stay compared with tube feeding.[14] Similarly, if tube nutrition is required, then the simpler nasograstric route has also been shown to be comparable in pain, intestinal permeability, and endotoxemia to more distal nasojejunal feeding.[15] The nutritional progression should therefore begin with oral intake with transition to nasogastric, nasojejunal, and finally parenteral nutrition as needed, with the expectation that the proportion of patients in each successive tier will be fewer and fewer.

The use of prophylactic antimicrobial agents in patients with severe AP has a long and confrontational history. Eleven randomized trials have been reported. Unfortunately, the methodology and specific antimicrobials have varied across most studies.[16–18] When taken as a whole, however, it becomes clear that prophylactic antimicrobial therapy should be avoided.[16] Although the bacteria resident within infected necrosis were traditionally reported to be *Escherichia coli*, *Pseudomonas*, and anaerobic species,[17–19] the most common flora have now changed considerably. Gram-positive organisms are the most frequent bacteria within secondary pancreatic infections in any patient cohort treated with preceding antibiotics (52%).[20] This shift in bacteriology is also reflective of overall improvements in critical care and our ability to maintain life support in these patients for prolonged periods (i.e., enhanced opportunity for secondary infections). It remains clear however that resistant bacterial infections caused by preceding antimicrobial therapy lead to an increased length of stay, reoperation, and readmission. Fungal infections are also clearly linked to increased hospital stays, reoperations, as well as overall morbidity and mortality. The general recommendation is to stop all antibiotics if a given patient has been previously started on an empiric/prophylactic basis before transfer to the acute care surgery service. It also prudent to note that although the observation of gas within the necrosum on cross-sectional imaging (or upon a rarely performed percutaneous aspirate) may indicate the presence of infection, this finding in itself should not directly alter the clinician's plans relating to antimicrobial therapy, nutritional access, or other core principles. In other words, although prophylactic antibiotics are not required, the use of therapeutic antimicrobials is reliant on the clinical condition of the patient and is only mandated in the presence of significant *clinical* deterioration.

Nonsurgical Interventions

Additional topics of note include the role of early endoscopic retrograde cholangiopancreatography (ERCP) in the

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context of choledocholithiasis. This has been evaluated in numerous prospective studies.[21–26] Among 153 patients in a multicenter prospective study, patients were divided into two groups (with and without signs of cholestasis).[27] Although ERCP was associated with fewer complications compared with the observation group (25% vs. 54%, $p = 0.02$) in patients with signs of cholestasis, mortality was not significantly lower (6% vs. 15%, $p = 0.2$). In addition, ERCP reduced neither complications (45% vs. 41%, $p = 0.8$) nor mortality (14% vs. 17%, $p = 0.7$) in patients without cholestasis, suggesting that ERCP should be indicated only in selected patients with *persistent* cholestasis.[21,26] Trials evaluating probiotic use reported that prophylaxis did not reduce the risk of infectious complications but actually increased the risk of mortality in patients with severe AP.[28] Similarly, another trial of 302 patients with moderate-to-severe AP who received either octreotide or placebo had similar rates of mortality, complications, duration of pain, surgical interventions, and length of hospital stay. This suggests that octreotide should not be used in severe AP.[29]

Surveillance for complications and the improvement/progression of necrotizing pancreatitis is most commonly obtained via cross-sectional (computed tomography [CT]) imaging with intravenous contrast. As clinicians however, we must be cautious to avoid the overuse of ionizing radiation in patients where our pretest probability of altering the patient's therapeutic course is low. More specifically, in patients with severe or critical AP, only 31% of CT scans were found to alter management in a highly experienced pancreatitis critical care unit.[30] Perhaps, even more importantly, the risk of acute kidney injury related to the use of intravenous contrast medium is also a significant concern.

Prognosis

Objectively defining the severity and prognosis of AP has a long history. Multiple intricate scoring systems have aimed to predict both organ dysfunction (Ranson's criteria, Acute Physiology and Chronic Health Evaluation score [APACHE], multiple-organ failure assessment score, modified Glasgow Scale, simplified acute physiology score (SAPS) 3, mortality probability model III [MPM]) and local complications (Balthazar score, body mass index, hematocrit).[31] Each of these scoring systems has specific advantages and provides insight into the severity of AP (Table 4). Despite their variability, Ranson,[32] APACHE,[33] and Balthazar[34] scores are commonly used within North America and remain helpful indicators of both local and systemic complications (including mortality). It must be stated, however, that the best early indicators of severity remain the clinical signs and symptoms of potential organ failure (tachycardia, hypotension, tachypnea, hypoxemia, oliguria, encephalopathy). Less commonly used serum markers for the severity of AP also include trypsinogen activation peptide, C-reactive protein, amyloid A, and an assortment of cytokines.[35,36]

Mortality secondary to AP has improved substantially during the past few decades. More specifically, mortality associated with necrotizing pancreatitis has decreased from 65% in 1960 to 15% in 2000.[37] This radical improvement is related to two dominant alterations in management. The first is epic advances in nearly all areas of critical care. The second, which has special relevance to acute care surgery teams, is the increasing recognition of the optimal timing of operative intervention and therefore a more nuanced understanding of the natural course of AP itself. It is now clearly understood that the most common cause of patient death within the first 24 hours and first 7 days following admission (95% and 94%, respectively), remains respiratory failure.[38] Beyond the 7-day mark however, sepsis becomes an increasingly dominant etiology. This not only reflects the intricate relationship between organ dysfunction and prognosis but also elucidates the bimodal distribution of timing and mortality associated with AP itself.[39] Deaths within the first 3 weeks to 4 weeks of AP are typically related to multiorgan failure, whereas those deaths in the 4-week to 7-week range are more often a consequence of sepsis.

TABLE 4. Predictors of Adverse Outcomes in Scoring Systems for Severe AP

Predictors of organ dysfunction
APACHE score ≥ 8
Multiple Organ Dysfunction Score > 3 at 72 h
Ranson score ≥ 3 at 48 h
Modified Glasgow Scale ≥ 3
Sequential Organ Failure Assessment score > 4 at 48 h
Predictors of local complications
Balthazar C, D, E CT grade at 7 d
C, inflammation of the pancreas or peripancreatic fat
D, single fluid collection
E, ≥2 fluid collections and/or retroperitoneal air
Body mass index > 30
Hematocrit > 44%

Timing of Operative Intervention

Given the relationship between death and surgical timing, it became clear through the 1980s and 1990s that a policy of delayed operative intervention represented a safer approach. More specifically, as surgery was delayed over time, both the incidence of intervention (68% in 1980–1985 to 33% in 1991–1997) and subsequent mortality (39% in 1980–1985 to 12% in 1991–1997) decreased dramatically.[37] This epiphany was most clear following the publication of a randomized controlled trial of early (<72 hours) versus late (>12 days) necrosectomy.[40] Among 36 patients, the mortality rate decreased from 56% in early interventions to 27% in late operations. Beyond mortality itself, it is also clear that intraoperative blood loss is substantially reduced by a delayed approach. Early resections (<7 days) have been associated with mean hemorrhage volumes as high as 5,700 mL.[41] Finally, despite initial beliefs to the contrary, it is also evident that early debridements have no positive impact on the systemic inflammatory response and therefore on the driving factor for early death.[42] As high-volume pancreatitis services continue to improve on a clear volume-outcome relationship (i.e., akin to pancreatic resections), mortality associated with necrotizing pancreatitis has now been reported to be as low as 4% with an overall decreased hospital length of stay approximating 26 days.[43] Similarly, morbidity (50%) and the rate of return to the operating theater (20%) are also reduced. These impressive gains in centers such as Indiana University[43] were recently echoed in a large nationwide analysis in 2011.[44]

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The required patience before operative intervention in patients with necrotizing pancreatitis often leads to frustration on the part of team members because it seems that ongoing organ physiologic support leads to few "big wins" and movements forward for a given patient. There are two notable exceptions to the rule of delayed intervention however. In cases where a patient displays rapid and progressive deterioration despite maximal physiologic support, the clinician must contemplate the occurrence of ischemia of the gallbladder and/or colon. Each of these organs is at particular risk for vascular inflow insufficiency because of both the geographic variation of the pancreatic necrosum itself and pharmacology-induced low-flow states. These two scenarios are the exceptions that may mandate early and focused operative intervention. As a result, it is ischemia of these two organs that must be ruled out if a critically ill patient continues to worsen, as opposed to focusing on the status of the pancreas gland itself (infected or not). If the pancreatic necrosum is not mature (i.e., 4–6 weeks), then any operative intervention should remain exclusively directed at removing the ischemic organ and avoiding any interaction with the pancreas.

Interventions and Technique

Interventional guidelines are always undertaken in the context of comprehensive and precise multidisciplinary critical care based on the severity of disease. The surgical treatment of patients with severe AP has evolved dramatically and now includes open, laparoscopic, percutaneous, and endoscopic techniques of debridement and drainage.[45,46] These approaches may be used alone or in combination (i.e., hybrid). Once the patient is stabilized and the pancreatic necrosum is mature, operative therapies may involve both minimally invasive (laparoscopic cystgastrostomies and debridements, use of percutaneous drains as access guides for rigid scope debridement, step-up procedures, endoscopic transmural debridements) and/or open (transperitoneal, retroperitoneal-flank) approaches.[47–51] The best choice among these options is based on patient anatomy and the specific location(s) of the necrosum within that given patient. In addition to this long list of potential techniques to remove necrotic tissue, an equally dominant consideration for the ACS surgeon is one of timing. As mentioned earlier, almost every patient should be physiologically supported without major intervention until the 28-day mark. Patients with severe AP follow a predictable pattern of early systemic inflammatory response syndrome and potentially multiorgan failure. Unfortunately, this interval observation is often misinterpreted as sepsis requiring treatment with antimicrobial therapy or major intervention. Within the first 7 days to 10 days, very few of these patients have infected necrosis (and therefore do not require antimicrobial therapy).

Numerous core surgical principles must be highlighted. Operating too early in the course of AP leads to an inability to differentiate between "viable," "nonviable," and "potentially viable" inflamed pancreatic tissues. It also results in clinicians missing patients who will resolve spontaneously (10%) with no required active surgical intervention (i.e., only medical support). It should be noted that inadequate debridement leads to persistent sepsis and "unwellness." An unrecognized disconnected pancreatic duct (i.e., disconnected left pancreatic remnant and therefore "end" fistula) often results in an uncontrolled pancreatic fistula and subsequent mortality.[52] The guiding principle in effective necrosectomies (beyond appropriate timing) remains precisely targeted removal of the majority of necrosum in the most minimally invasive manner possible. Achieving this goal relies on a detailed preoperative assessment using cross-sectional imaging that outlines the specific location of the necrosum and subsequent operative approach (i.e., is the necrosum limited to the lesser sac only [and well opposed to the posterior gastric wall], or does it involve one or both paracolic gutters, or is it within the leash of the superior mesenteric artery [SMA] and vein?). As a result, approaches may require the use of preoperative percutaneous catheters as route guides, flank/retroperitoneal incisions, and/or combined minimally invasive and open modalities.

In general, a surgical transgastric cyst-gastrostomy with concurrent necrosectomy is the preferred approach to many of these patients.[53–55] This lone procedure is typically successful in removing nearly all pancreatic necrosum, addressing ongoing drainage from a disconnected left pancreatic remnant, and essentially converts a traditionally multiprocedure approach into a single operative intervention. In cases where necrosum within the lesser sac extends down either paracolic gutter and/or the SMA/superior mesenteric vein leash and the connecting fistulous tract to the lesser sac remains patent, the vast majority of necrosis can still be removed through the cystgastrostomy. In scenarios where the communicating tract has scarred down and therefore created large "islands" of necrosis that are not contiguous with the lesser sac, additional approaches must be used to remove all of the necrosum in a single sitting. Preoperative percutaneous insertion of drainage catheters (as large as possible) into these islands followed by intraoperative enlargement of the tracts and use of either open or rigid endoscope debridement will lead to removal of the remaining necrotic material. If the islands of necrosis are small enough, they may not require any additional debridement beyond the initial cystgastrostomy however. If auxiliary percutaneous catheters are required, it is essential that the acute care surgeon be intimately involved with the interventional radiologist in determining the precise location of insertion for these tubes. They must facilitate subsequent surgical access (i.e., posteriorly placed catheters make this notoriously difficult). Synchronous removal of the gallbladder (if safe) is also advocated to reduce the risk of future recurrences.

It should be noted that although controversy remains with regard to the initial intervention of choice,[56] every approach possesses inherent limitations. This choice is also influenced by both surgeon experience and hospital resources. More specifically, many high-volume surgeons believe that percutaneous drainage alone is frequently unsuccessful because of an inability of a relatively small drain to remove the thick solid (peri)pancreatic necrosum from the patient. Furthermore, if a percutaneous drain fails in the context of a primarily liquid collection, the patient is often left with a persistently draining pancreatic fistula in a location that is often problematic with regard to both patient comfort and subsequent surgical approaches (e.g., fistulojejunostomy). Similarly, endoscopic cystgastrostomies lack durability and are also limited by their inability to remove thick solid necrosum and by the

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requirement for prolonged indwelling hardware (i.e., large bore catheters/tubes should remain in place until they naturally fall out). Endoscopic drainage can be helpful however in alleviating some symptoms (i.e., pain and obstruction) in patients who meet absolute contraindications for surgical intervention and have primarily fluid-filled collections.

The recent publication popularity highlighting percutaneous techniques as a lone treatment for pancreatic necrosis deserves specific mention. CT-guided drainage followed by repeated irrigation procedures in the context of ever increasingly larger drains placed by involved and committed radiologists may improve the clinical course in up to 75% of patients.[57,58] It has also been shown to resolve the necrotic collection in 45% of cases. It must be emphasized that achieving success in removing enough of the necrosum with this technique requires incredible dedication from the surgeon and especially the interventional radiologist. More specifically, these patients require repeated trips to the interventional radiology suite over prolonged periods to remove large quantities of solid necrosum. Although appropriate head-to-head comparisons are limited at this early juncture, institutional commitment with regard to equipment, physician, and inpatient resources is substantial and must be carefully considered and organized by a dedicated multidisciplinary group. Finally, it must also be emphasized that this procedure is fundamentally distinct from the placement of a percutaneous drain as a guide for subsequent operative debridement as described previously.

In addition to the operative resection of pancreatic necrosum and previously mentioned ischemic gallbladder and/or colon, further operative interventions most commonly surround relief of the ACS. Although this scenario is increasingly uncommon secondary to personalized goal-directed resuscitation and ongoing support with the minimization of crystalloid fluids, it may still occur. Once diagnosed and following the failure of percutaneous attempts at removing any large volumes of exudative and/or pancreatic ascites, decompression via laparotomy remains the standard of care. Following a generous laparotomy incision (i.e., to prevent recurrent ACS), the patient should generally be left with an open abdomen/temporary abdominal closure to facilitate the correction of extremis physiology. As always in the context of the open abdomen, the surgeon must aim to close the patient's fascia as soon as possible following physiologic improvement.

It can be concluded that although the list of subtle technical pearls associated with operative pancreatic debridement/necrosectomy is extensive, two overarching principles remain critical to success. First, extreme care is essential with regard to tissue handling. A soft touch not only with pancreatic and peripancreatic tissues but also with all tissues throughout the peritoneal cavity is absolutely essential (omentum, bowel, gallbladder, stomach, and spleen). These tissues are uniformly edematous and do not tolerate rough handling. They also bleed frequently when this principle is violated. This is particularly relevant to tissues within the lesser sac because they often include a thrombosed cord-like splenic vein, a thin walled portal vein, a freely hanging SMA, and/or multiple arterial branches resident within the splenic hilum. Each of these vessels has the ability to cause catastrophic hemorrhage both during the operative intervention and within the postoperative setting if injuries are missed and/or not adequately controlled. Control of venous hemorrhage is almost always surgical (pressure, packing, and suture/clip ligation), while arterial bleeding is often best arrested via percutaneous embolization techniques. Second, the surgical treatment of pancreatitis is frequently more challenging than pancreatic oncology cases. This reality highlights the extreme helpfulness of a surgeon with extensive experience in the surgical intervention of these patients. Whether this experience arises from a high-volume pancreatitis fellowship or a long tenure as the local "pancreatitis surgeon," these experts can be lifesaving.

In summary, as long as a pancreatic fistula/leak is well controlled in the acute setting, most severe AP scenarios associated with necrosum can be treated in the long term by an experienced surgeon. As a result, broad and effective drainage following nearly all necrosectomies (except successful cystgastrostomies) is mandatory. In these scenarios, externalized (i.e., closed suction drain) and controlled pancreatic fistulas have the opportunity to close on their own without additional intervention (side fistula: mean, 22 weeks; end fistula: mean, 28+ weeks).[59–61] If they do not close, roux-en-y fistula-jejunostomies or completion distal pancreatectomies remain strong options.[52]

It should be emphasized that the need to manage significant and quality of life–altering complications following successful emergency care provided by the acute care surgery team to patients with severe/infected pancreatitis is common. This reality is also often the most challenging component of their overall management. More specifically, much of the therapy for these patients begins after discharge from the acute care hospital and surrounds complications such as the disconnected pancreatic duct (with or without fistulae), enteric (including duodenal) and colonic fistulae, and ubiquitous incisional hernia. Even in the context of optimal and timely acute care management, the surgeon must follow these patients for a prolonged period. Similar to oncology, this further emphasizes the need for a truly multidisciplinary team that is engaged in both the early phases and the postdischarge segments of this disease.

CONCLUSION

In conclusion, the successful treatment of patients with severe and critical AP will require all of your skills as an acute care surgeon. These include but are not limited to an evidence-based knowledge of the literature, advanced critical care, technical expertise, and perhaps most importantly, patience. Acute care surgeons remain the best trained workforce to treat these patients given their high frequency, chronic nature, and requirement for "nonboutique" surgery using core general surgical and critical care principles.

DISCLOSURE

The authors declare no conflicts of interest.

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