Commentary

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22.1 Relevant Basic Information, Indications, and Contraindications

The diagnosis of acute pancreatitis is generally straightforward. The pillars of diagnostic evaluation are the clinical history (abdominal pain, nausea, vomiting) and serum amylase and lipase determination. We perform ultrasonography primarily to assess for gallstones as the cause of the episode of pancreatitis, and to look for bile duct dilation as a possible sign of ongoing choledocholithiasis – however, ultrasonography is of limited utility in evaluating the pancreas itself.

We agree that contrast-enhanced computed tomography (CT) is the most specific imaging modality available for diagnosing acute pancreatitis. CT is rarely necessary for diagnosis, though CT may be valuable for excluding other potential sources of abdominal pain, assessing biliary obstruction, identifying necrosis at an early stage, and for prognostication. We do not, however, perform routine "initial CT assessment" of patients in whom we confidently diagnose acute pancreatitis for several reasons. First, patients with severe pancreatitis often present with acute kidney injury which may be worsened by the administration of intravenous contrast agents. Second, there is some experimental and clinical evidence that intravenous contrast may contribute to the worsening of pancreatic necrosis. Finally, in our experience, many patients who will go on to develop extensive necrosis may lack obvious non-enhancement of the pancreatic parenchyma and have only edema evident on a CT performed at the time of presentation, as the authors allude to in Table 22.1 where they state, "early CT may underestimate the ultimate severity of pancreatitis." Most of these patients will require a repeat CT early in their course (such as at 4-5 days, as the authors suggest) to determine the extent of necrosis. We find that an initial CT evaluation rarely alters our management during this initial period, while carrying some risk of possible harm. Recent studies have stressed the overuse of CT in necrotizing pancreatitis and outlined the very real dangers with regard to radiation exposure and financial impact.

As the authors point out, antibiotic prophylaxis remains a matter of debate. Historically, the vast majority of our patients have received antibiotic prophylaxis, usually with a carbepenem. Nevertheless, more recently we have trended away from the routine use of antibiotics because of the lack of proven efficacy in Level 1, randomized studies. We agree that endoscopic retrograde cholangiography (ERC) should not be used routinely in cases of gallstone pancreatitis, but ERC is indicated if there is evidence of biliary obstruction due to a retained gallstone in the common bile duct.

Management throughout the early phase of the systemic inflammatory response syndrome (SIRS) that accompanies acute pancreatitis so frequently

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	Number (%)
	Nulliber (70)
Patients	67
Etiology	Alcoholic 23 (34 %)
	Biliary 20 (30 %)
	Unknown 9 (13 %)
	Hypertrigyceridemia 5 (8 %)
	Post-ERCP 4 (6 %)
	Post-operative 4 (6 %)
	Other 2 (3 %)
Indications	Infection 47 (71 %)
	SIRS 9 (13 %)
	Persistently unwell 6 (9 %)
	Persistent pancreatitis 4 (6 %)
	Hemorrhage 1 (1 %)
Microbiology	Infected necrosis 52 (77 %)
	Sterile necrosis 15 (23 %)
Reoperation	11 (16 %)
Mortality	6 (9 %)

 Table 22.1
 Pancreatic débridements for acute necrotizing pancreatitis performed from 2006 to 2010

ERCP endoscopic retrograde cholangiopancreatography, *SIRS* systemic inflammatory response syndrome

Indications: All patients had demonstrated necrosis. If infection was proven or strongly suspected, that was considered the primary indication for débridement. SIRS was considered the indication in cases where the decision to operate was based on the presence of necrosis without demonstrable infection in a patient with escalating, lifethreatening SIRS. Persistent pancreatitis denotes repeated episodes of acute pain with increases in serum amylase and lipase after the primary episode of necrotizing pancreatitis. The persistently unwell patient usually has chronic, low grade, but unresolving symptoms (e.g. intolerance of oral feeds, nausea, weight loss, abdominal pain, or fever) after an episode of necrotizing pancreatitis

is generally non-operative. We attempt to use enteral nutrition whenever possible because its use has been associated with decreased rates of infected pancreatic necrosis. If nutritional needs cannot be met enterally, then parenteral nutritional support is utilized. The surgeon must remain involved closely throughout this phase of the illness because infected necrosis or abscess may intervene, and plans for treatment need to be made with an eye toward possible eventual operative intervention should a less invasive approach not be indicated.

Proven infected necrosis remains the one consensus indication for some formal necrosectomy in acute pancreatitis. Infected necrosis is demonstrated typically by either CT findings of extraluminal gas within areas of pancreatic necrosis or by staining and culture of specimens from CT-guided fine needle aspiration (FNA) of areas of pancreatic or peripancreatic necrosis. It is important to note, however, that CT-guided FNA may have a 20-25 % false negative rate and even occasional false positives. We cannot overemphasize the clinical observation that when faced with a patient with known pancreatic necrosis who is failing to improve, either in the critically ill phase (persistent organ failure, SIRS) or even as an outpatient (low grade fever, failure to tolerate oral feeding, the so-called "persistent unwell") the diagnosis of infected necrosis must be entertained and necrosectomy considered strongly, even after a negative FNA. Many of these patients will have infection demonstrated from the operative samples; in addition, we have found that many with sterile necrosis will nonetheless improve clinically. As the authors point out, localized areas of infected necrosis can be treated sometimes with a combination of endoscopic and percutaneous drainage. More commonly, however, with extensive peripancreatic necrosis, we maintain that operative necrosectomy is required.

Even in patients who have documented infected necrosis early in their course, we prefer to wait 4 weeks if possible from the onset of pancreatitis until operation. This delay in necrosectomy allows the necrotic tissue to completely demarcate from viable pancreatic and retroperitoneal tissue, minimizing the risks of incomplete débridement, bleeding, and post-operative pancreatic insufficiency. We developed this general policy after a review of our own data demonstrated an optimal composite outcome score (including death, intensive care utilization, need for further operative or percutaneous procedures, and other major complications) if débridement was performed at 27 days. Waiting for a greater period of time did not confer added advantage.

22.2 Operative Technique

It is critical that a recent CT, preferably with oral and intravenous contrast, be available in the operating room to ensure that all areas with necrosis



Fig. 22.1 Blunt débridement of the lesser sac through the transverse mesocolon (Reproduced with permission from: Fernández-del Castillo et al. (1998))

or fluid recognized preoperatively are recognized and addressed intraoperatively. Manual blunt necrosectomy with closed packing is our preferred technique. We usually begin with a midline incision.

Our primary approach to the lesser sac is through the transverse mesocolon (Fig. 22.1). We believe this approach offers a number of advantages over an anterior approach into the lesser sac:

- 1. It avoids a difficult and time-consuming dissection of the stomach and omentum off the transverse colon, which is often densely adherent due to inflammation in the lesser sac.
- 2. By avoiding the risk of even small serosal tears to the transverse colon in a setting of infection and possible pancreatic fistula, this approach may decrease the risk of colo-cutaneous fistula.
- 3. Entry through the base of the mesocolon allows drains to be placed in a dependent position posterior in the lesser sac.

Commonly, the mesocolon to the left of the ligament of Treitz is thinned, allowing easy entry through this usually avascular region into the area of necrosis and fluid collection. The middle colic vessels are often thrombosed, but if they are patent and present an impediment, they can be divided, usually without sequelae. If left-sided collections in the retrocolic or pararenal spaces cannot be reached by this approach, they may require medial mobilization of the splenic flexure of the colon. If collections or necrosis surrounding the head of the pancreas cannot be reached via this left-sided approach, a second opening may be made in the right side of the transverse mesocolon. Care should be taken to remain oriented to the position of the superior mesenteric vessels relative to the areas of necrosis when this approach is used. Right sided collections that cannot be reached via the transmesocolic approach can be exposed by mobilizing the hepatic flexure of the colon medially with the second and third portions of the duodenum as necessary.

While this anterior, transperitoneal, transmesocolic approach is our primary technique, it is worth noting that in selected patients with a localized retroperitoneal area of necrosis or fluid collection, a primary retroperitoneal approach can be simpler and yield excellent results. In cases fitting this description, we prefer, if possible, to have a percutaneous drain placed by a totally retroperitoneal access route. If this does not resolve the infection and débridement is required, a more limited incision can be made over the skin access point of the drainage catheter, and the catheter can then be followed into the area of necrosis. This operative approach to necrosectomy can also be done videoscopically.

Once areas of fluid and necrosis are exposed by any approach, fluid should be drained and devitalized tissue débrided bluntly. Dissection with fingers combined with use of blunt, circular sponge clamps and vigorous irrigation allows separation of necrotic tissue from still viable tissue. All necrotic tissue should be removed and sent for microbiologic analysis. Sharp dissection should be avoided. Bleeding from cavity walls may be from granulation tissue or from major vascular structures. Bleeding from major vessels should be controlled with sutures if possible, but if exposure is difficult, packing may be required.

Once all areas of necrosis have been drained and débrided thoroughly, we pack any resulting cavities with ³/₄ in. Penrose drains stuffed with gauze, and then place soft, silicone-rubber closedsuction drains into each major extension of the cavity (Fig. 22.2). Each drain, Penrose or closed



Fig. 22.2 Packing of the cavity with stuffed Penrose and Jackson-Pratt drains (Reproduced with permission from: Fernández-del Castillo et al. (1998))

suction, is brought out through a separate stab incision in the abdominal wall and sutured to the skin.

If indicated, cholecystectomy is performed at the time of débridement. Bowel resection or diversion may be required if enteric fistula or perforation is present. Occasionally involvement of the splenic vessels may result in splenic infarction necessitating splenectomy. Rarely, decompressive gastrostomy or feeding jejunostomy is also performed.

22.3 Additional Treatments and Procedures

Postoperative antibiotics are tailored to the culture results as they become available. We continue antibiotics usually for 10–14 days after débridement. Enteral feeding is preferred. Many patients can tolerate an oral diet. In those who cannot, nasogastric or nasojejunal tubes are placed as needed, or gastrostomy or jejunostomy feeding can be instituted.

The stuffed Penrose drains are left in place for 1 week and then removed, usually at the rate of

one drain every other day. Removal of these drains allows the packed cavity to close gradually and allows large particulate matter a route of egress. Closed suction drains are left in place until output is minimal, and there is no evidence of ongoing pancreatic fistula. Low output (<100 mL/day) fistulas may be managed by sequentially withdrawing the closed suction drain by 2 cm every week. This lengthening of the fistulous tract encourages closure of the fistula. If fever, abdominal pain, or inability to tolerate oral intake occurs during the process of sequential drain withdraw, abdominal CT is performed seeking an intraabdominal collection. Regardless of the presence of a fistula, any patient who does not continue to improve after débridement should undergo abdominal CT scanning. In our experience, 30 % of patients will require subsequent percutaneous drain placement after débridement, so residual fluid collections should be sought actively if clinical progress is poor.

22.4 Results

Results from 2006 to 2010 are included in Table 22.1. We have reported our results previously for 1990–2005 using the technique described above Rodriguez et al. (2008). For that longer time period, our overall mortality in 167 patients undergoing operative débridement was 11 % (20 % in patients operated before 28 days vs. 5 % in patients operated after 28 days, P=0.002). The reoperation rate was 13 % and the rate of post-operative percutaneous intervention was 30 %. The median post-operative duration of hospital stay was 19 days (range 4–195 days).

22.5 Summary

A comparison of our method for operative treatment of necrotizing pancreatitis with that of the authors both demonstrates areas of growing consensus, but also illustrates points of divergence in contemporary management of this disease. CT imaging for the identification of necrosis and planning of operative therapy, infected necrosis as the primary indication for débridement, and delaying débridement to 4 weeks after the onset of symptoms whenever possible have taken hold as accepted principles of management. Nonetheless, considerable variability in practice likely persists regarding the role of débridement in the persistently unwell patient without preoperatively proven infected necrosis, the role of minimally invasive drainage or débridement techniques, and the technical approaches to open débridement. These areas require further research to optimize patient care in the interesting and challenging disease.

References

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- Rodriguez JR, Razo AO, Targarona J et al (2008) Débridement and closed packing for sterile or infected necrotizing pancreatitis: insights into indications and outcomes in 167 patients. Ann Surg 247(2):294–299