Surgery of acute pancreatitis

Edward L. Bradley III, M.D.

Department of Clinical Sciences, Florida State University College of Medicine, 1600 Baywood Way, Sarasota, FL 34231, USA

Abstract

Within the past two generations, changing indications for surgical intervention, coupled with improvements in surgical techniques, have led the way to remarkable reductions in the morbidity and mortality of acute pancreatitis and its complications. Moreover, parallel advances in supportive intensive care and an improved understanding of the pathophysiology of acute pancreatitis also have contributed significantly to increased patient survival. Now that the pancreas has finally emerged from the dim recesses of the retroperitoneum to gain its rightful place of importance among the other organs, we may anticipate a rapid increase in our knowledge of the gland, ultimately leading to cures of its conditions and one day, perhaps even to the final goal of all medicine: prevention of disease. © 2007 Excerpta Medica Inc. All rights reserved.

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Historical Aspects

It is now almost 120 years since Reginald Fitz, a Harvard anatomist, delivered to the New York Pathologic Society the first systemic description of fatal acute pancreatitis [1]. In the conclusions of his clinicopathologic analysis, he strongly advised that surgical intervention not be performed in necrotizing pancreatitis. This opinion was vigorously challenged by both American [2] and Continental European surgeons [3–5], who believed, despite few actual successes, that surgery represented the only hope for salvation in these desperately ill patients.

The subsequent development of the serum amylase assay in 1929 [6] revealed that milder forms of acute pancreatitis existed that had not been previously recognized. Furthermore, once the spectrum of acute pancreatitis was enlarged from autopsy studies to include nonfatal cases, spontaneous recovery from acute pancreatitis was recognized to be the rule and not the exception. Accordingly, surgical intervention was demonstrated to be unnecessary in the majority of patients. When these observations of recovery in the vast majority of patients were combined with the significant mortality and morbidity from resection of the inflamed and necrotic pancreas, the therapeutic pendulum swung away from operative intervention for severe acute pancreatitis in the 1930s and 1940s [7,8].

However, noting that patients with the more severe forms of acute pancreatitis continued to die with nonoperative

Corresponding author. Tel.: +1-941-923-1331; fax: +1-941-587-7560. *E-mail address:* ed.bradley@med.fsu.edu

management, Pollack reopened the issue in 1959 by describing successful pancreatic resections in several of these seriously ill patients [9]. Thereafter, expanded pancreatic resections for severe acute pancreatitis began to be deployed in earnest. Successful total pancreatectomy for "fulminant" acute pancreatitis was first reported by Watts in England [10] and later advocated by Rives et al [11], Devic et al [12], and Hollender et al [13] in France. Pancreatectomy for severe acute pancreatitis was subsequently adopted by many other continental surgeons, including Alexandre and Guerri [14], Kivilaakso et al [15], and Aldridge et al [16], despite mortality rates often in excess of 60%. Presumably, their reasoning was that without surgical intervention, mortality would approach 100%. Once again, the pendulum had swung toward the surgical camp.

Assisted in the clinical recognition of pancreatic necrosis by the application of contrast-enhanced computed tomography (CECT), in 1985 Beger and his colleagues from Ulm emphasized the importance of the presence of pancreatic necrosis to the mortality in severe acute pancreatitis and to the development of complications [17]. Recognition that a course of severe acute pancreatitis could frequently be correlated with the presence of pancreatic necrosis was a significant step forward and underlies our clinicopathologic approach to the disease today. In a second major contribution, Professor Beger and his coworkers subsequently demonstrated the technical superiority of debridement of pancreatic and peripancreatic necrosis combined with continuous postoperative retroperitoneal lavage, rather than employing excessively morbid attempts at pancrea-

tectomy or pancreatoduodenectomy in the presence of extensive inflammation and obscuring edema. In subsequent prospective clinical trials, they were able to achieve mortality rates of less than 15% with this approach [18,19]. This was a remarkable improvement over the previous mortality rates of 40% to 60% associated with urgent resection. Debridement of pancreatic and peripancreatic necrosis was believed to be necessary for removing the toxic broth of retroperitoneal proteases released by the dead and dying tissues, whether or not the necrotic tissues were secondarily infected. Although the proposition that noninfected necrotic tissues should be removed was "intuitive" to many surgeons, this indication for surgery, in fact, had never been studied, as no populations of nonoperated controls with sterile pancreatic necrosis had been encountered.

Current Approaches

Sterile pancreatic necrosis

Over the 3-year period from 1986 to 1989, we studied 194 patients with documented acute pancreatitis in a prospective, longitudinal fashion, separating those with pancreatic necrosis (36/194) from those with interstitial pancreatitis by means of CECT. Surgical intervention was performed only in those 25 patients with pancreatic necrosis in whom the clinical course was compatible with pancreatic infection and needle aspiration bacteriology demonstrated bacteria or fungi in the necrotic tissue. Patients with documented sterile necrosis were managed solely by intensive medical support. Each of the 11 patients with sterile necrosis, including 6 with multiorgan failure, survived with conservative management alone [20]. We concluded from these observations that the vast majority of patients with acute pancreatitis do not require surgical intervention and that sterile pancreatic necrosis, even when accompanied by organ failure, was not an absolute indication for surgery. Subsequent prospective studies of sterile pancreatic necrosis have confirmed our initial findings, demonstrating mortality rates lower than 10% with nonoperative management [21–23]. When it became apparent that mortality rates for supportive management of sterile pancreatic necrosis were uniformly lower than rates in comparable patients treated operatively, once again the therapeutic pendulum reversed, and conservative management of patients with sterile pancreatic necrosis became widely adopted by the international medical and surgical communities [24].

Recently, there has been considerable rapprochement between the aggressive and the conservative surgical camps, in that we have recognized that *some* patients with sterile necrosis will continue to require operative therapy but that the percentage of these patients will be quite small. As a case in point, an occasional patient with sterile necrotizing pancreatitis will have recurrent abdominal pain and hyperamylasemia with repeated attempts at reinstituting oral feeding, despite full recovery from the systemic effects of pancreatitis. This complication, variably referred to as "refeeding pancreatitis" [25], "persistent pancreatitis" [26], or "disconnected duct syndrome" [27], is often the result of necrotic disruption of the pancreatic duct, with subsequent complete obstruction of the flow of pancreatic juice from the distal segment of the gland. The condition responds well

to distal pancreatectomy or roux-en-Y drainage. Other delayed surgical complications of sterile pancreatic necrosis need to be more widely recognized, such as necrosis-induced stricture of the main pancreatic duct with resultant "upstream" chronic pancreatitis [28], small and large bowel fibrotic obstructions due to ischemia [29], and strictures of the common bile duct [30]. These complications may become more evident as our experience expands with the remote consequences of conservatively treated necrotizing pancreatitis.

Infected pancreatic necrosis

In contrast to the now largely resolved controversies surrounding sterile necrosis, there has been relatively little disagreement regarding the necessity for surgical debridement and drainage of infected pancreatic necrosis. The principal area of discussion in infected necrosis has centered over the precise form of surgical drainage after necrosectomy—whether it should be open [25], semiclosed [18], or closed [31]. In the absence of any controlled trials addressing the issue of the optimal surgical technique, it is sufficient to note that each of the techniques is capable of providing good results in skilled hands. More recently, operative alternatives to the traditional transabdominal approach for debridement and drainage of infected pancreatic necrosis have been described. Both retroperitoneal [32] and laparoscopic approaches [33] have been reported, with acceptable results in selected cases. As we move forward, it will be important to carefully note the advantages and limitations of each new technique, in order to more appropriately match the surgical approach to the specific conditions exhibited by individual patients. Choosing unilateral retroperitoneal approaches in patients with extensive bilateral retroperitoneal necrosis will limit access to necrotic material due to normal anatomic restrictions, such as the vena cava or the spinal column. Moreover, attempting to apply limited access procedures to those patients exhibiting extensive retrocolic extensions of infected necrosis can hardly be expected to result in satisfactory outcomes.

It is well known that failure to timely remove infected pancreatic necrosis resulted in historically excessive mortality rates, frequently in the range of 70% [34]. With the advent of programmatic debridement and drainage for documented infected pancreatic necrosis, surgical mortality rates for this condition were lowered to less than 15% [18,22,25,31]. Although most experienced pancreatic surgeons can describe occasional cases of documented infected necrosis in patients that were successfully treated with antibiotics and transcutaneous catheter drainage alone, as opposed to formal surgical debridement, most of these patients harbored significant comorbid conditions, such as acute myocardial infarction, and were not acceptable surgical candidates. Recently, noting the anecdotal survival of a few patients with infected pancreatic necrosis treated without surgical debridement, several internists have questioned the dictum of mandatory surgical debridement whenever infected necrosis is documented [35]. While it is undoubtedly true that some cases of infected pancreatic necrosis can, in fact, be successfully managed without surgical debridement, it will be necessary for proponents of nonoperative man-

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