

Necrosectomy for Acute Necrotizing Pancreatitis

Michael G. Sarr and Gregory G. Tsiotos

Necrotizing pancreatitis (NP), the most severe form of acute pancreatitis (AP), accounts for 3% to 5% of all patients who have AP and 5% to 15% of patients in tertiary referral centers (Tsiotos et al. 1998). A better understanding of the pathophysiology of NP and the subsequent pathogenesis of "superinfection" leading to "infected necrosis" has led to improved treatment modalities and outcomes in the past three decades. Critical evaluation of the relevant literature, however, has been hampered by lack of universally accepted definitions of NP's clinical and radiographic characteristics. Today, most pancreatologists have accepted the Atlanta classification (Bradley 1993), an international consensus on terminology. Use of the Atlanta classification allows for a uniform approach to the diagnosis, management, reporting, and understanding of these conditions, and two entities with clear and precise definitions are described: acute fluid collections and pancreatic necrosis. In this chapter, the definition and management of each entity are presented separately with special effort to highlight previous misinterpretations in the literature. Our preferred operative techniques are discussed in detail, and important points on pre- and postoperative management are highlighted. Although necrosectomy (operative débridement) of the necrotic pancreatic parenchyma and peripancreatic tissues is the crucial element in the operative management of patients who have NP, equally important are the optimal timing of intervention and the appropriate nonoperative management that precedes (and may prevent the need for) necrosectomy. For these reasons, emphasis is also directed at initial nonoperative management of NP.

Acute Fluid Collections

Acute fluid collections (occasionally referred to as *pseudopseudocysts*) occur frequently in patients who have severe AP (30% to 50%), develop early in the course of AP, and are characterized by the lack of a wall of granulation or fibrous tissue (Bradley 1993; Yeo et al. 1994). They have no apparent communication with the pancreatic ductal system and represent a serous or exudative reaction to the pancreatic inflammation (their fluid does not contain high concentrations of pancreatic enzymes, and the electrolyte concentration is similar to that of plasma). Acute fluid collections do not manifest symptoms and are usually discovered on imaging studies. These fluid collections, which are often multiple and have an irregular shape, require no specific treatment and generally resolve spontaneously. This natural history is important to bear in mind when interpreting studies (mainly in radiology literature) on successful percutaneous drainage of pseudocysts; many such articles describe patients who really had acute fluid collections (not true pseudocysts), which would have regressed without treatment.

Although most of these acute fluid collections resolve spontaneously, some (approximately 10%) persist as true pseudocysts 6 weeks later—that is, peripancreatic fluid collections that communicate with the pancreatic ductal system and contain a high concentration of pancreatic enzymes. Early in the course of AP, these acute fluid collections (that go on to eventually into a pancreatic pseudocyst) appear quite similar to those fluid collections that resolve spontaneously; obviously, how-

ever, their pathogenesis differs. Ultimately, differentiation of acute fluid collections from pancreatic pseudocysts requires a certain time period (classically, 6 weeks) after onset of AP; persistent peripancreatic fluid collections represent pancreatic pseudocysts, because the acute fluid collections lacking communication with the pancreatic ductal system would have resolved by this point in time.

Necrotizing Pancreatitis

Dynamic contrast-enhanced computerized tomography (CT) is the mainstay for the diagnosis of pancreatic parenchymal and peripancreatic necrosis. Enhancement of the pancreatic parenchyma by bolus intravenous contrast accurately discriminates viable regions from necrotic regions of the gland and defines its extent. Contrast density lower than 50 HU within the pancreas and heterogeneous densities in the peripancreatic fat represent parenchymal necrosis and a combination of fat necrosis and fluid collections, respectively (Bradley 1993). Numerous laboratory parameters have been used to predict the course of NP. Increased C-reactive protein, polymorphonuclear leukocyte activation, early increase of polymorphonuclear-elastase, certain interleukins, and a host of other early response agents are associated with more severe disease. Evaluation based on clinical staging systems, however, is equally effective and has had a more widespread application. Ranson's and Glasgow (Imre's) criteria have been largely replaced in clinical practice by the acute physiology and chronic health evaluation (APACHE-II) score—a physiologic score that is not limited to the early time frame of the dis-