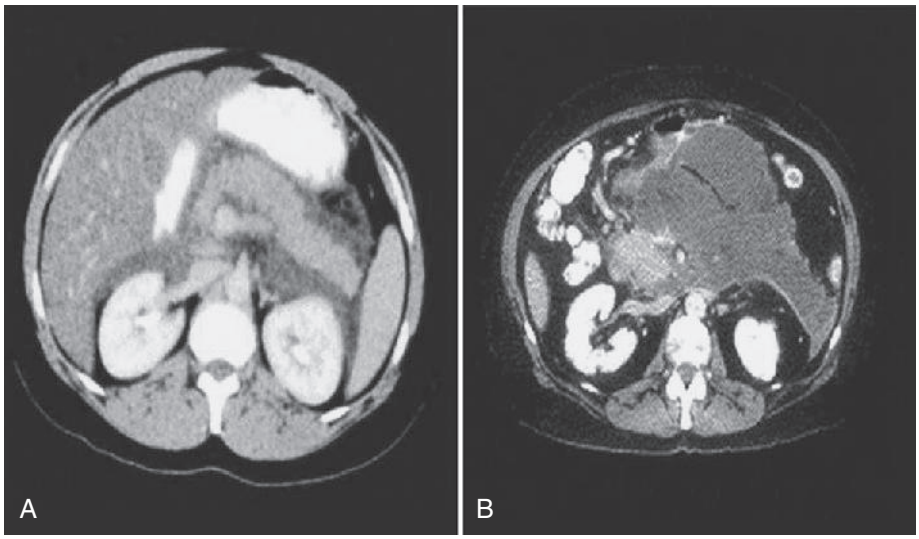


**FIGURE 38-3** Contrast-enhanced computed tomography demonstrates interstitial pancreatitis (**A**) and necrotizing pancreatitis (**B**).

## Prognosis

The distinction between interstitial and necrotizing acute pancreatitis has important prognostic implications (Fig. 38-3). *Interstitial pancreatitis* is characterized by an intact microcirculation and uniform enhancement of the gland on CECT. *Necrotizing pancreatitis* is characterized by disruption of the pancreatic microcirculation so that large areas (>3 cm or >30%) of pancreatic parenchyma do not enhance on CECT. Approximately 20% to 30% of patients with acute pancreatitis have necrotizing pancreatitis.

The finding of pancreatic necrosis predicts a more severe course, particularly infection in the necrotic pancreatic tissue, also called *infected necrosis*. Infection is a strong determinant of the severity of illness and accounts for a large percentage of the deaths from acute pancreatitis. Infected necrosis develops in 30% to 50% of patients with acute necrotizing pancreatitis but not in those with interstitial disease. Infected necrosis should be suspected in patients with persistent systemic inflammatory response syndrome (SIRS) or organ dysfunction. The diagnosis can be made if extraluminal gas is seen on CECT. More commonly, CT-guided needle aspiration is obtained for Gram stain and culture of necrotic material, or antibiotics are given empirically based on clinical suspicion after appropriate cultures are obtained. Antibiotics that penetrate pancreatic tissue, including cephalosporins, carbapenems, quinolones, and metronidazole, are used for treatment of infected necrosis.

Risk assessment should be performed for all patients to stratify the severity of illness. The current classification includes mild, moderate, and severe forms. Mild acute pancreatitis, the most common form, is characterized by the absence of organ failure and pancreatic necrosis. Mild pancreatitis usually does not require pancreatic imaging, and patients recover within several days with restoration of normal pancreatic function and gland architecture. Patients with mild acute pancreatitis account for 80% of all attacks and less than 5% of the overall mortality rate.

Moderately severe pancreatitis is transient organ failure or local or systemic complications in the absence of organ failure. Local complications include pancreatic necrosis (with or without infection) and acute peripancreatic fluid collections. Death from moderately severe pancreatitis is much less common than in cases of severe pancreatitis.

Severe acute pancreatitis is defined by persistent organ failure extending for more than 48 hours. Severe acute pancreatitis occurs in 15% to 20% of patients. Most individuals with persistent organ failure have underlying necrotizing disease. The respiratory, cardiovascular, and renal systems are most commonly affected. Early deaths (within the first week) are most often the result of multiple organ failure caused by the release of inflammatory mediators and cytokines. Late deaths are more likely to result from local or systemic infection. The risks of infection and death correlate with disease severity and pancreatic necrosis.

Despite the importance of recognizing severe disease, most patients are initially admitted to the hospital without necrosis or organ failure, and methods to predict individuals more likely to progress to severe disease during the initial several days of hospitalization have been defined. A combination of clinical assessment, scoring systems, serum markers, and CECT scanning provides the most useful prognostic information (Table 38-2). Regardless of the prognostic factor chosen, there are significant limitations in predicting disease severity.

Clinical predictors of a poor outcome include severe comorbid illnesses, older age, and obesity. Laboratory findings associated with increased mortality include blood urea nitrogen elevation on admission or a rise during the first 24 hours of admission, hemoconcentration from third spacing of fluids reflected by an elevated hematocrit of 44 or greater on admission, and serum markers reflecting a robust systemic inflammatory response, such as a C-reactive protein level greater than 150 mg/dL (sensitivity of 80%, specificity of 76%, positive predictive value of 67%, and negative predictive value of 86%). Imaging studies predicting a severe outcome include a pleural effusion seen on chest radiography within the first 24 hours or pancreatic imaging identifying necrosis.

Severe pancreatitis is predicted by organ dysfunction, including shock (systolic blood pressure <90 mm Hg), respiratory failure ($Pao_2$ ≤60 mm Hg), and acute renal injury (creatinine