complications and fluid collections along with a CT imaging template to guide reporting of findings. Local morphologic features are summarized in Table 371-1. *Interstitial pancreatitis* occurs in 90–95% of admissions for acute pancreatitis and is characterized by diffuse gland enlargement, homogenous contrast enhancement, and mild inflammatory changes or peripancreatic stranding. Symptoms generally resolve with a week of hospitalization. *Necrotizing pancreatitis* occurs in 5–10% of acute pancreatitis admissions and does not evolve until several days of hospitalization. It is characterized by lack of pancreatic parenchymal enhancement by intravenous contrast agent and/or presence of findings of peripancreatic necrosis. According to the revised Atlanta criteria, the natural history of pancreatic and peripancreatic necrosis is variable because it may remain solid or liquefy, remain sterile or become infected, and persist or disappear over time. CT identification of local complications, particularly necrosis, is critical in patients who are not responding to therapy because patients with infected and sterile necrosis are at greatest risk of mortality **(Figs. 371-1*B*, 371-2, and 371-3)**. The median prevalence of organ failure is 54% in necrotizing pancreatitis. The prevalence of organ failure is perhaps slightly higher in infected versus sterile necrosis. With single-organ system failure, the mortality is 3–10% but increases to 47% with multisystem organ failure.

ACUTE PANCREATITIS MANAGEMENT

We will briefly describe the management of patients with acute pancreatitis from the time of diagnosis in the emergency ward to ongoing hospital admission and, finally, to time of discharge, highlighting salient features based on severity and complications. It is important to note that 85–90% of cases of acute pancreatitis are self-limited and subside spontaneously, usually within 3–7 days after initiation of treatment, and do not exhibit organ failure or local complications.

The management of acute pancreatitis begins in the emergency ward. After a diagnosis has been

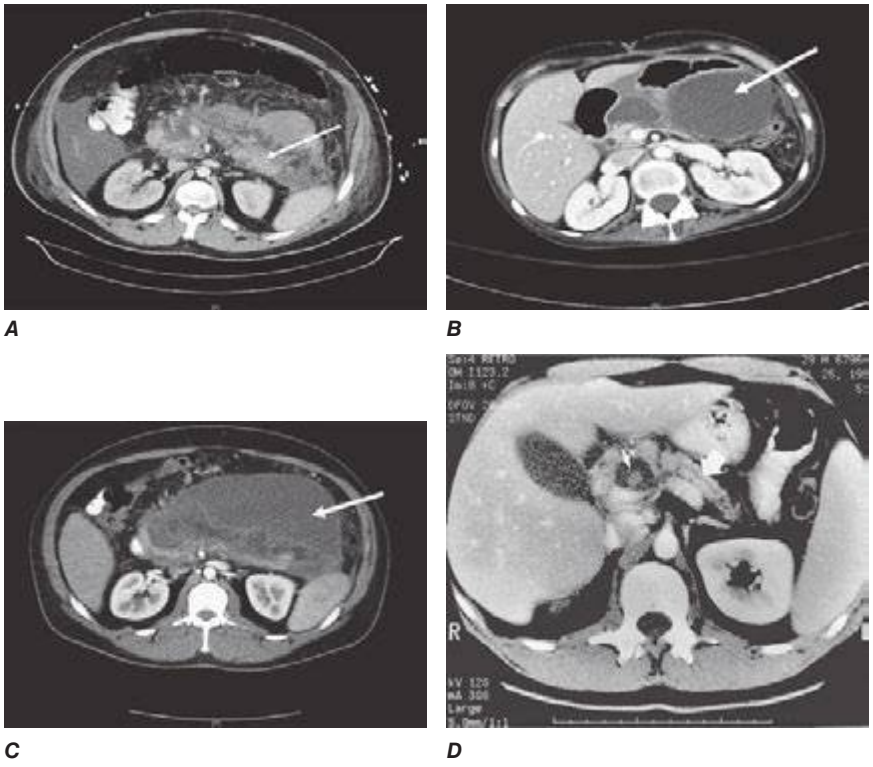

FIGURE 371-2 ***A.*** Acute necrotizing pancreatitis: computed tomography (CT) scan. Contrast-enhanced CT scan showing acute pancreatitis with necrosis. *Arrow* shows partially enhancing body/tail of pancreas surrounded by fluid with decreased enhancement in the neck/body of the pancreas. ***B.*** Acute fluid collection: CT scan. Contrast-enhanced CT scan showing fluid collection in the retroperitoneum (*arrow*) compressing the air-filled stomach arising from the pancreas in a patient with asparaginase-induced acute necrotizing pancreatitis. ***C.*** Walled-off pancreatic necrosis: CT scan. CT scan showing marked walled-off necrosis of the pancreas and peripancreatic area (*arrow*) in a patient with necrotizing pancreatitis. Addendum: In past years, both of these CT findings (Figs. 371-2*B* and 371-2*C*) would have been misinterpreted as pseudocysts. ***D.*** Spiral CT showing a pseudocyst (*small arrow*) with a pseudoaneurysm (*light area in pseudocyst*). Note the demonstration of the main pancreatic duct (*big arrow*), even though this duct is minimally dilated by endoscopic retrograde cholangiopancreatography. *(A, B, C, courtesy of Dr. KJ Mortele, Brigham and Women's Hospital; D, courtesy of Dr. PR Ros, Brigham and Women's Hospital; with permission.)*

FIGURE 371-3 ***A.*** Pancreaticopleural fistula: pancreatic duct leak on endoscopic retrograde cholangiopancreatography. Pancreatic duct leak (*arrow*) demonstrated at the time of retrograde pancreatogram in a patient with acute exacerbation of alcohol-induced acute or chronic pancreatitis. ***B.*** Pancreaticopleural fistula: computed tomography (CT) scan. Contrast-enhanced CT scan (coronal view) with *arrows* showing fistula tract from pancreatic duct disruption in the pancreatic pleural fistula. ***C.*** Pancreaticopleural fistula: chest x-ray. Large pleural effusion in the left hemithorax from a disrupted pancreatic duct. Analysis of pleural fluid revealed elevated amylase concentration. *(Courtesy of Dr. KJ Mortele, Brigham and Women's Hospital; with permission.)*