**TABLE 371-2 REVISED ATLANTA DEFINITIONS OF MORPHOLOGIC FEATURES OF ACUTE PANCREATITIS**

| Morphologic Feature | Definition | Computed Tomography Criteria |
|---|---|---|
| **Interstitial pancreatitis** | Acute inflammation of the pancreatic parenchyma and peripancreatic tissues, but without recognizable tissue necrosis | Pancreatic parenchyma enhancement by IV contrast agent<br>No findings of peripancreatic necrosis |
| **Necrotizing pancreatitis** | Inflammation associated with pancreatic parenchymal necrosis and/or peripancreatic necrosis | Lack of pancreatic parenchymal enhancement by IV contrast agent and/or presence of findings of peripancreatic necrosis (see below—ANC and WON) |
| **Acute pancreatic fluid collection** | Peripancreatic fluid associated with interstitial edematous pancreatitis with no associated peripancreatic necrosis. This term applies only to areas of peripancreatic fluid seen within the first 4 weeks after onset of interstitial edematous pancreatitis and without the features of a pseudocyst. | Occurs in the setting of interstitial edematous pancreatitis<br>Homogeneous collection with fluid density<br>Confined by normal peripancreatic fascial planes<br>No definable wall encapsulating the collection<br>Adjacent to pancreas (no intrapancreatic extension) |
| **Pancreatic pseudocyst** | An encapsulated collection of fluid with a well-defined inflammatory wall usually outside the pancreas with minimal or no necrosis. This entity usually occurs >4 weeks after onset of interstitial edematous pancreatitis. | Well circumscribed, usually round or oval<br>Homogeneous fluid density<br>No nonliquid component<br>Well-defined wall; that is, completely encapsulated<br>Maturation usually requires >4 weeks after onset of acute pancreatitis; occurs after interstitial edematous pancreatitis |
| **Acute necrotic collection (ANC)** | A collection containing variable amounts of both fluid and necrosis associated with necrotizing pancreatitis; the necrosis can involve the pancreatic parenchyma and/or the peripancreatic tissues. | Occurs only in the setting of acute necrotizing pancreatitis<br>Heterogeneous and nonliquid density of varying degrees in different locations (some appear homogeneous early in their course)<br>No definable wall encapsulating the collection<br>Location—intrapancreatic and/or extrapancreatic |
| **Walled-off necrosis (WON)** | A mature, encapsulated collection of pancreatic and/or peripancreatic necrosis that has developed a well-defined inflammatory wall. WON usually occurs >4 weeks after onset of necrotizing pancreatitis. | Heterogeneous with liquid and nonliquid density with varying degrees of loculations (some may appear homogeneous)<br>Well-defined wall; that is, completely encapsulated<br>Location—intrapancreatic and/or extrapancreatic<br>Maturation usually requires 4 weeks after onset of acute necrotizing pancreatitis |

***Source:*** Modified from P Banks et al: Gut 62:102, 2013.

an increased output of adrenal glucocorticoids and catecholamines. *Hypocalcemia* occurs in ~25% of patients, and its pathogenesis is incompletely understood. Although earlier studies suggested that the response of the parathyroid gland to a decrease in serum calcium is impaired, subsequent observations have failed to confirm this phenomenon. Intraperitoneal saponification of calcium by fatty acids in areas of fat necrosis occurs occasionally, with large amounts (up to 6.0 g) dissolved or suspended in ascitic fluid. Such "soap formation" may also be significant in patients with pancreatitis, mild hypocalcemia, and little or no obvious ascites. *Hyperbilirubinemia* (serum bilirubin >68 mmoL or >4.0 mg/dL) occurs in ~10% of patients. However, jaundice is transient, and serum bilirubin levels return to normal in 4–7 days. Serum alkaline phosphatase and aspartate aminotransferase levels are also transiently elevated, and they parallel serum bilirubin values and may point to gallbladder-related disease or inflammation in the pancreatic head. *Hypertriglyceridemia* occurs in 5–10% of patients, and serum amylase levels in these individuals are often spuriously normal (Chap. 370). Approximately 5–10% of patients have *hypoxemia* (arterial $PO_2$ ≤60 mmHg), which may herald the onset of ARDS. Finally, the electrocardiogram is occasionally abnormal in acute pancreatitis with ST-segment and T-wave abnormalities simulating myocardial ischemia.

An abdominal ultrasound is recommended in the emergency ward as the initial diagnostic imaging modality and is most useful to evaluate for gallstone disease and the pancreatic head.

The revised Atlanta criteria have clearly outlined the morphologic features of acute pancreatitis on computed tomography (CT) scan as follows: (1) interstitial pancreatitis, (2) necrotizing pancreatitis, (3) acute pancreatic fluid collection, (4) pancreatic pseudocyst, (5) acute necrotic collection (ANC), and (6) walled-off pancreatic necrosis (WON) (Table 371-2 and Fig. 371-1). **Radiologic studies useful in the diagnosis of acute pancreatitis are discussed in Chap. 370 and listed in Table 370-1.**

## DIAGNOSIS

Any severe acute pain in the abdomen or back should suggest the possibility of acute pancreatitis. The diagnosis is established by two of the following three criteria: (1) typical abdominal pain in the epigastrium that may radiate to the back, (2) threefold or greater elevation in serum lipase and/or amylase, and (3) confirmatory findings of acute pancreatitis on cross-sectional abdominal imaging. Patients also have associated nausea, emesis, fever, tachycardia, and abnormal findings on abdominal examination. Laboratory studies may reveal leukocytosis, hypocalcemia, and hyperglycemia. Although not required for diagnosis, markers of severity may include hemoconcentration (hematocrit >44%), admission azotemia (BUN >22 mg/dL), SIRS, and signs of organ failure (Table 371-3).

The *differential diagnosis* should include the following disorders: (1) perforated viscus, especially peptic ulcer; (2) acute cholecystitis and biliary colic; (3) acute intestinal obstruction; (4) mesenteric vascular occlusion; (5) renal colic; (6) inferior myocardial infarction; (7) dissecting aortic aneurysm; (8) connective tissue disorders with vasculitis; (9) pneumonia; and (10) diabetic ketoacidosis. It may be difficult to differentiate acute cholecystitis from acute pancreatitis, because an elevated serum amylase may be found in both disorders. Pain of biliary tract origin is more right sided or epigastric than periumbilical or left upper quadrant and can be more severe; ileus is usually absent. Ultrasound is helpful in establishing the diagnosis of cholelithiasis and cholecystitis. Intestinal obstruction due to mechanical factors can be differentiated from pancreatitis by the history of crescendo-decrescendo pain, findings on abdominal examination, and CT of the abdomen showing changes characteristic of mechanical obstruction. Acute mesenteric vascular occlusion is usually suspected in elderly debilitated patients with brisk leukocytosis, abdominal distention, and bloody diarrhea, confirmed by CT or magnetic resonance angiography. Vasculitides secondary to systemic lupus erythematosus and polyarteritis nodosa may be confused with pancreatitis, especially because pancreatitis may develop as a complication of these diseases. Diabetic ketoacidosis is often accompanied by abdominal pain and elevated total serum amylase levels, thus closely mimicking acute pancreatitis. However, the serum lipase level is not elevated in diabetic ketoacidosis.

## CLINICAL COURSE, DEFINITIONS, AND CLASSIFICATIONS

The Revised Atlanta Classification (1) defines phases of acute pancreatitis, (2) defines severity of acute pancreatitis, and (3) clarifies imaging definitions as outlined below.

**Phases of Acute Pancreatitis** Two phases of acute pancreatitis have been defined, early (<2 weeks) and late (>2 weeks), which primarily