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Diseases of the Pancreas

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ACUTE PANCREATITIS

Definition and Epidemiology

Acute pancreatitis is an acute inflammatory process of the pancreas that may also involve peripancreatic tissues and remote organ systems. It is the leading cause of hospitalization of patients with gastrointestinal disorders in the United States, with more than 200,000 admissions annually. This translates into an overall incidence of 1 case per 4000 people in the general population.

Pathology

The pancreas is located in the retroperitoneum and has exocrine and endocrine functions (Fig. 38-1) derived from the pancreatic acinus and the pancreatic islet, respectively. As an exocrine gland, the pancreas participates in normal digestion and nutrient absorption. The enzymes secreted by the pancreas digest starch (i.e., amylase), fats (i.e., lipase), and protein (i.e., trypsin and other proteolytic enzymes). Within acinar cells, proteolytic digestive enzymes are synthesized and packaged separately in the Golgi region into condensing vacuoles and transported in an inactive form referred to as zymogens to the apical portions of the cell. When stimulated, they are discharged into the central ductule of the acinus by exocytosis.

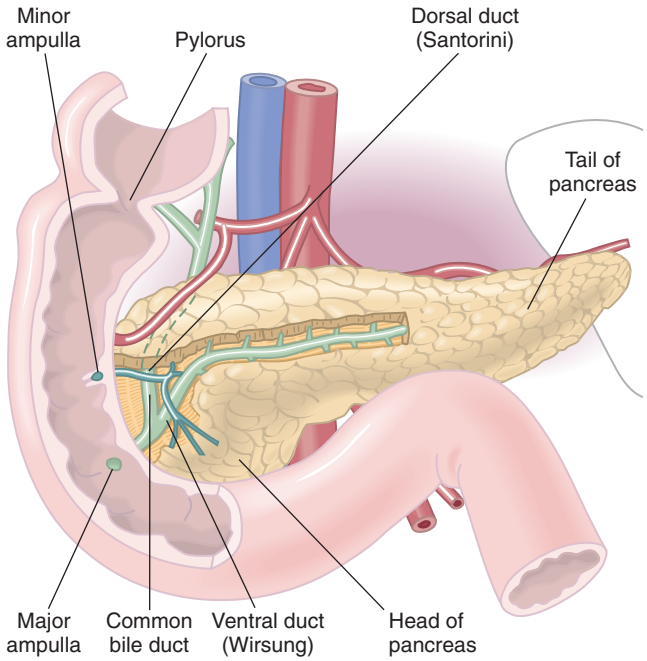

FIGURE 38-1 Normal anatomy of the pancreas.

Normal physiology involves secretion of inactive enzymes into the duodenum, where they are converted to an active form by enterokinase, a brush border enzyme secreted by small bowel enterocytes. Trypsinogen conversion to active trypsin is the trigger enzyme that subsequently converts the other zymogens to active enzymes (E-Fig. 38-1).

The pathogenesis of acute pancreatitis remains incompletely understood. Based on experimental models, the initiating event appears to involve intra-acinar activation of trypsin from trypsinogen, resulting in acute intracellular injury, pancreatic autodigestion, and the potential for profound systemic complications after activated enzymes are leaked into the bloodstream. Initiating events may include obstruction of the pancreatic duct (e.g., gallstones, pancreatic tumor), overdistention of the pancreatic duct (e.g., from endoscopic retrograde cholangiopancreatography [ERCP]), reflux of biliary or duodenal juices into the pancreatic duct, changes in permeability of the pancreatic duct, ischemia of the organ, and toxin-induced cholinergic hyperstimulation (Fig. 38-2).

During the initial hospitalization for acute pancreatitis, reasonable attempts to determine the cause are appropriate, particularly those that may affect acute management. The cause of acute pancreatitis is readily identified in 70% to 90% of patients after an initial evaluation consisting of the history, physical examination, focused laboratory testing, and routine radiologic studies. Gallstones account for 45%, alcohol for 35%, miscellaneous causes for 10%, and idiopathic causes for 10% to 20% of acute pancreatitis cases (Table 38-1).

Gallstone Pancreatitis

Among patients with gallstones, the incidence of acute pancreatitis is 0.17% per year. Gallstones increase the relative risk of pancreatitis 25- to 35-fold. It is theorized that gallstone passage causes transient obstruction of the pancreatic duct, precipitating acute pancreatitis. Acute gallstone pancreatitis should be suspected when associated with a transient elevation in liver-associated enzymes, particularly alanine aminotransferase (ALT) levels greater than 150 IU/L. Most stones pass spontaneously from the ampulla and do not require intervention (discussed later).

Alcoholic Pancreatitis

Acute alcoholic pancreatitis is the second most common cause of pancreatitis in the United States. Approximately 10% of chronic alcoholics develop attacks of pancreatitis that are indistinguishable from other forms of acute pancreatitis. Alcoholics with acute pancreatitis most commonly have underlying