Pancreatitis

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KEYWORDS

• Pancreatitis • Epidemiology • Management

KEY POINTS

- Acute pancreatitis is an inflammation of the glandular parenchyma of the retroperitoneal organ that leads to injury with or without subsequent destruction of the pancreatic acini. This inflammatory process can either result in a self-limited disease or involve life-threatening multiorgan complications.
- In contrast, chronic pancreatitis is a syndrome that consists of endocrine and exocrine gland dysfunction that develops secondary to progressive inflammation and chronic fibrosis of the pancreatic acini with permanent structural damage.
- Recurrent attacks of acute pancreatitis can result in chronic pancreatitis; it is thought that acute and chronic pancreatitis are 2 different diseases with 2 separate morphologic patterns.
- Acute pancreatitis has an estimated annual incidence of 4.9 to 40 cases per year per 100,000, which has been increasing over the last several decades, albeit with a decreasing mortality.

INTRODUCTION

Acute pancreatitis (AP) is an inflammation of the glandular parenchyma of the retroperitoneal organ that leads to injury with or without subsequent destruction of the pancreatic acini. This inflammatory process can either result in a self-limited disease or involve life-threatening multiorgan complications. In contrast, chronic pancreatitis (CP) is a syndrome that consists of endocrine and exocrine gland dysfunction that develop secondary to progressive inflammation and chronic fibrosis of the pancreatic acini with permanent structural damage. Recurrent attacks of AP can result in CP; it is thought that AP and CP are 2 different diseases with 2 separate morphologic patterns.[1–4] AP has an estimated annual incidence of 4.9 to 40 cases per year per 100,000, which has been increasing over the last several decades, albeit with a

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decreasing mortality.[5,6] The incidence of CP ranges from 5 to 12 cases per year per 100,000, with an estimated prevalence of 50 per 100,000.[6,7]

Peery and colleagues[8] reported in 2012 that AP was the most common gastrointestinal admission diagnosis, with an inpatient cost estimate of $2.6 billion per year. AP is reported to carry a mortality risk of approximately 1%; however, a subset of patients can have a more severe form of the disease with a mortality approaching 30%.[8,9] CP was reported to have a mortality as high as 20% to 25%.[10,11] Hence, proper management of those diseases requires a multidisciplinary team approach with the involvement of gastroenterologists, endocrinologists, pain specialists, psychiatrists, surgeons, and support groups.

Cause

Acute pancreatitis

The cause of AP can be readily identified in most patients, with gallstones and alcohol being the leading causes (40%–70% and 25%–35% respectively).[12,13]

Gallstone size negatively correlates with an increased risk of pancreatitis. Smaller stones are more likely to migrate down the bile or pancreatic duct and cause an obstruction, which in return increases ductal pressure and unregulated digestive enzymatic activity.[14] Because gallstones have a high prevalence, the best way to evaluate for cholelithiasis is by performing abdominal ultrasonography on all patients with AP. However, only 3% to 7% of patients who have gallstones develop AP.[15] Moreover, men with gallstones have a higher risk of developing AP, whereas women have a higher incidence of gallstone pancreatitis because of a higher prevalence of gallstones.

AP associated with alcohol consumption is usually considered in patients with AP who have a history of 5 years or more of heavy drinking. Because clinically identified AP is only prevalent in 5% of heavy drinkers, there may be additional genetic and environmental factors that contribute to a person's sensitivity to the negative effects of alcohol (eg, failure to inhibit trypsin activity, tobacco use).[12,14,16]

Other infrequent causes of AP include hypercalcemia secondary to hyperparathyroidism, hypertriglyceridemia, endoscopic retrograde cholangiopancreatography (ERCP), drugs, and infections. Primary or secondary hypercalcemia can lead to calcium deposition in the pancreatic duct or activation of trypsinogen in the pancreas.[17] However, this only occurs inconsistently in 1% to 4% of AP cases.[12,14,18] Similarly, hypertriglyceridemia accounts for 1% to 4% of AP cases, potentially resulting in AP attacks following a serum triglyceride concentration more than 1000 mg/dL.[12] Hypertriglyceridemia can be acquired by many factors (eg, obesity, diabetes mellitus, hypothyroidism) or caused by inherited lipoprotein metabolism disorders.

ERCP can potentially cause AP because of various associated risks. After the procedure, 35% to 70% of the patients develop asymptomatic hyperamylasemia.[14,19] If the hyperamylasemia presents with persistent severe abdominal pain, nausea, and vomiting, it is generally diagnosed as post-ERCP pancreatitis.[14,20] The risk for AP associated with ERCP is higher when it is used to treat sphincter of Oddi dysfunction (25%) but is much lower if used for removal of gallstones (5%). In addition, diagnostic ERCP is only associated with AP in 3% of the patients.[14,21] Further ERCP-induced AP-associated risks include young age, female sex, and poor emptying of the pancreatic duct after the procedure.[14] To prevent post-ERCP pancreatitis in patients at higher risk, a temporary pancreatic stent may be placed during the procedure.[22] In addition, prophylactic nonsteroidal antiinflammatory drugs were also reported to be effective in preventing post-ERCP pancreatitis.[23]

Several reported cases show that various medications (eg, 6-mercaptopurine, aminosalicylates, sulfonamides, diuretics, valproic acid) are associated with inducing