Uncomplicated Acute Pancreatitis

Evidenced-Based Management Decisions

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KEYWORDS

• Pancreatitis • Acute • Severity • Management

KEY POINTS

- Acute pancreatitis is among the most common gastrointestinal disorders requiring hospitalization.
- Early goal-directed fluid resuscitation with lactated Ringer solution remains the cornerstone of therapy the management of mild acute pancreatitis.
- Non-opioid analgesics should be considered in the management of pain in acute pancreatitis.
- A low-fat, low-residue diet can be used for initial re-feeding after resolution of nausea, emesis and abdominal pain.
- There is no role of prophylactic antibiotics in the setting of necrotizing pancreatitis.

INTRODUCTION AND EPIDEMIOLOGY

Acute pancreatitis (AP) is among the most common gastrointestinal disorders requiring hospitalization worldwide with an annual incidence of 13 to 45 cases per 100,000 persons.[1] In the United States, AP resulted in 275,000 hospitalizations in 2012 with aggregate costs of $2.6 billion.[2] Recent National Hospital Discharge Surveys suggests that although there has been an increase in AP admissions, the overall mortality rate has remained around 2%.[3]

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DIAGNOSIS OF ACUTE PANCREATITIS

According to the revised Atlanta classification, AP is diagnosed if two or more of the three clinical features listed at the top of **Table 1** are present.[4] There are additional considerations listed under each of these clinical features that are importance to the practitioner (see **Table 1**).

ELEVATION OF PANCREATIC ENZYMES WITHOUT ACUTE PANCREATITIS

Lipase is elevated to three times the upper limit of normal in many nonpancreatic conditions as summarized in **Table 2**.[5–7] In a large study of cardiovascular safety in patients with type 2 diabetes, 22.7% were noted to have asymptomatic amylase and lipase elevation.[8] Abdominal imaging should be considered when a patient without clear risk factors for AP presents with upper abdominal pain and elevated pancreatic enzymes because neither are specific for AP.

EVALUATION OF THE CAUSE OF ACUTE PANCREATITIS

Establishing the cause of AP ensures appropriate management and proper health care resource use. The two most common causes of AP are biliary (40%–70%) and alcohol use (25%–35%).[1,9] Other causes include metabolic factors, such as hypertriglyceridemia (HTG), hypercalcemia, drug induced, autoimmune, hereditary/genetic, and anatomic abnormalities.

Biliary Tract Stones and/or Sludge

Biliary tract stones and/or sludge are the most common cause of AP. Approximately 7% of the US adult population has gallstones but only 0.1% to 0.3% develop associated complications including acute cholecystitis, choledocholithiasis, or acute biliary pancreatitis.[10,11] All patients presenting with their first episode of AP should undergo abdominal ultrasonography.[12] If abdominal ultrasonography does not identify stones or sludge, an alanine aminotransferase greater than three times the upper limit of normal has greater than 95% positive predictive value for acute biliary pancreatitis.[13] Patients with elevated liver enzymes on Day 1 were found to have low risk of AP recurrence after cholecystectomy (9%), but the risk of AP recurrence is higher among those without elevated liver enzymes (34%) or in those without gallbladder stone/sludge (61%).[14]

The risk of pancreatitis among heavy users of alcohol ranges from 2% to 5%.[15,16] It should be highlighted that most heavy drinkers do not develop pancreatitis, which suggests that there are other factors that drive risk. Alcohol can modify the risk of AP from other etiologies including genetic mutations, hyperlipidemia, and drug-induced pancreatitis.[1] There is a dose-dependent relationship between alcohol and the risk of AP with various reports describing an increased risk of pancreatitis in individuals consuming greater than 14 beers per week.[16,17]

Smoking Tobacco

Smoking tobacco has been identified to be independent risk factor for pancreatitis. The risk is dose dependent, particularly with a 15 pack-year history.[18] However, with two decades of smoking cessation, the risk is noted to be reduced to the levels of never smokers.[19] Smoking is also known to modify risk of AP from other etiologies and combined use of smoking and alcohol can synergistically increase the risk of AP.[1]