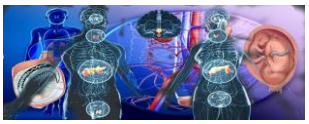
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The Management of Inflammation of The Exocrine Pancreas: Acute Pancreatitis

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Abstract

Acute pancreatitis is the leading cause of gastrointestinal-related hospitalization in the United States, and its frequency continues to rise in the United States and worldwide. The severity of the disease varies widely, from mild disease needing conservative treatment to severe and complicated disease with high morbidity and mortality. The most common causes of acute pancreatitis include gallstones, alcohol use, and hypertriglyceridemia. The rate of occurrence of each etiology of acute pancreatitis varies across geographic regions and socioeconomic strata.

Keywords: Acute pancreas; edematous pancreatitis; peri-pancreatic tissue; exocrine and endocrine secretions

Introduction

Acute pancreatitis is an acute response to injury of the pancreas. Chronic pancreatitis can result in permanent damage to the structure and endocrine and exocrine functions of the pancreas. In the United States, about 200,000 hospital admissions annually are due to acute pancreatitis, and this number has been increasing.

Etiology

The two most common causes of acute pancreatitis in the United States are gallstones (35% to 40% of cases) and alcohol use (30% of cases). However, the causes are extensive and include, but are not limited to, the following: autoimmune pancreatitis, hypertriglyceridemia, post-endoscopic retrograde cholangiopancreatography (ERCP), genetic risk (gain of function mutations in PRSS1, mutations in CFTR and SPINK1 genes), pancreatic duct injury and medications.

The most common cause of chronic pancreatitis is ethanol abuse. Smoking is also an important risk factor. There are several other causes which include tropical pancreatitis, idiopathic pancreatitis, ductal obstruction (pancreas divisum, pseudocysts, stones, tumors, and trauma), mutations in cystic fibrosis genes, hereditary pancreatitis and systemic diseases such as systemic lupus erythematosus.

Pathophysiology

The initiating event in AP is due to acinar cell injury and impaired secretion of zymogen granules and involves extracellular neural and vascular mechanisms as well as intracellular mechanisms (such as intracellular enzyme activation, calcium accumulation and heat shock protein activation). Increased calcium transients potentiate co-localisation of zymogen and lysosome granules and ultimately premature conversion of typsinogen to trypsin. Medications that may cause AP through acinar cell injury include azathioprine, corticosteroids and thiazide diuretics. Ethanol-induced pancreatitis has different pathophysiological mechanisms. Ethanol is directly toxic to the acinar cell, leading to inflammation and membrane destruction. There is also evidence that ethanol increases pancreatic ductal pressure favouring retrograde flow and intra-pancreatic enzymatic activation. It is likely that the ischaemia-reperfusion injury plays a role in the development of AP, which is supported by the importance of early aggressive fluid resuscitation.

Diagnosis

Presentations of pancreatitis include epigastric or diffuse abdominal pain (80–95%), nausea and vomiting (40–80%), abdominal distension, fever, breathlessness, irritability, and impaired consciousness, with pyrexia, low oxygen saturation, tachypnoea, tachycardia, hypotension, abdominal guarding, ileus and/or oliguria. The medical history should include careful enquiry directed at aetiology including gallstones, obesity, alcohol excess, smoking, hyperlipidaemia, and drugs that can induce the disease, recognising that more than one precipitant may cause the disease.

Hypertriglyceridaemia

Hypertriglyceridaemia was found to be the cause of acute pancreatitis in ~ 9% in a recent global systematic review, making it the third most common cause. One high-volume centre in China reported acute pancreatitis secondary to hypertriglyceridaemia in 33% of cases, the second most common cause in this cohort. The Endocrine Society differentiates primary (genetic) from secondary (e.g. metabolic syndrome, diabetes mellitus, alcohol or pregnancy) hypertriglyceridaemia as mild (150–500 mg/dL; 1.7–5.6 mmol/L), moderate (500–1000 mg/dL; 5.6–11.3 mmol/L) or severe (> 1000 mg/dL; > 11.3 mmol/L) dependent on serum triglyceride levels; much is polygenic in nature . There is an approximate 4% increase in the incidence of acute pancreatitis for every 100 mg/dL rise in serum triglyceride levels above 1000 mg/dL level frequently used to define hypertriglyceridaemia as the cause of acute pancreatitis.

Drugs

Although a well-recognised category accounting for up to 5% of acute pancreatitis, possibly with higher frequencies as co-factors, there are relatively few studies of drug-associated acute pancreatitis. A large multi-national collaboration is underway to develop biomarkers that may help to address this deficit.An important focus of this collaboration is to develop novel biomarkers for accurate identification and prognostication of pancreatic injury induced by drugs in early-phase trials, to determine whether to continue, modify or abandon drug development. Most published reviews to date focus on acute pancreatitis associated with drugs already licensed for other indications, the strongest evidence for which is recurrence of acute pancreatitis if the implicated drug is reintroduced, after withdrawal and recovery from a prior attack.

Endoscopic Retrograde Cholangiopancreatography (ERCP)

ERCP is associated with a significant risk of acute pancreatitis, assessed in one systematic review of > 100 randomised clinical trials at ~ 9% and up to 14% in high-risk patients, although there are effective strategies to reduce this risk. Patients at greatest risk are young women with small or normal bile ducts and sphincter of Oddi dysfunction.

Autoimmune Pancreatitis

Autoimmune pancreatitis is a rare, distinct form of chronic pancreatitis that may present acutely. It was initially described as chronic pancreatitis with hypergammaglobulinaemia, prior to introduction of the term 'autoimmune pancreatitis. It was then variously described as chronic pancreatitis with autoimmune features, non-alcoholic duct-destructive chronic pancreatitis, lymphoplasmocytic sclerosing pancreatitis with cholangitis, chronic sclerosing pancreatitis, pseudotumorous pancreatitis and duct-narrowing chronic pancreatitis. The current definition of two types is based on histopathology: type 1 for lymphoplasmacytic sclerosing pancreatitis and type 2 for idiopathic duct centric chronic pancreatitis or autoimmune pancreatitis with granulocytic epithelial lesions.

Conclusion

The management of acute pancreatitis involves interprofessional teams including hospitalists, gastroenterologists, surgeons, nurses, pharmacists, addiction specialists, and dietitians. The management of acute pancreatitis should involve specific counseling geared toward the etiology of pancreatitis. If a patient is noted to have several readmissions with pancreatitis, and the cause is known to be alcohol abuse, for instance, this should be specifically targeted via intensive counseling by the healthcare team.

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