

HOSPITAL PHYSICIAN®

GASTROENTEROLOGY BOARD REVIEW MANUAL

STATEMENT OF EDITORIAL PURPOSE

The *Hospital Physician Gastroenterology Board Review Manual* is a peer-reviewed study guide for fellows and practicing physicians preparing for board examinations in gastroenterology. Each manual reviews a topic essential to the current practice of gastroenterology.

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Chronic Pancreatitis

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Chronic Pancreatitis

Phillip P. Toskes, MD, and John G. Lieb, II, MD

INTRODUCTION

Chronic pancreatitis is a progressive inflammatory disease that is associated with significant morbidity and mortality. It causes 86,000 hospital admissions per year in the United States.¹ Patients with chronic pancreatitis have a mortality rate that is 3.6 times that of the general population, with 10- and 20-year survival rates of 70% and 45%, respectively. Mortality in these patients predominantly is due to comorbidities, including atherosclerotic disease, cirrhosis, continued alcohol and narcotic use, pancreatic and other cancers, and postoperative complications.²

Despite continued investigative efforts, chronic pancreatitis remains difficult to diagnose, particularly in its early stages. Classically, the diagnosis requires a pancreatic tissue sample demonstrating irreversible structural damage. However, histologic diagnosis is limited by the difficulty of obtaining pancreatic tissue and the patchy distribution of histologic changes in early disease. The secretin stimulation assay represents an alternative diagnostic approach, but it is not widely available and is poorly tolerated by patients. Therefore, clinicians often rely on presentation, radiologic and endoscopic findings, and biochemistry to diagnose chronic pancreatitis. This manual reviews the causes of chronic pancreatitis and discusses current diagnostic and therapeutic options.

ETIOLOGY

The myriad causes of chronic pancreatitis (**Figure 1** and **Table 1**) center around 2 fundamental physiologic events. First, bicarbonate secretion may decrease due to obstruction, genetic factors, or direct toxins. Reduced bicarbonate secretion is the primary mechanism for steatorrhea because active pancreatic lipase requires a neutral pH. Second, premature activation of the proteolytic cascade within the acini releases inflammatory cytokines, which eventually cause gland fibrosis, duct distortion, and altered secretion of pancreatic enzymes.

COMMON CAUSES

Alcohol

Approximately 70% of chronic pancreatitis cases

in the Western world are caused by alcohol. Although no amount of alcohol is safe, it is believed that in most persons at least 5 years of intake exceeding 150 g/day is required to cause pancreatitis. However, it has been suggested that intake as little as 50 g/day can cause chronic pancreatitis.³ For unclear reasons, only 5% to 15% of heavy drinkers develop chronic pancreatitis.⁴ Alcohol also may hasten the course of idiopathic chronic pancreatitis.⁵ Very rarely, alcohol can cause acute pancreatitis without evidence of chronicity, but in some patients, recurrent attacks of alcohol-induced acute pancreatitis lead to chronic pancreatitis.⁶ Several mechanisms of alcohol-induced injury to the pancreas have been proposed: increased sphincter of Oddi relaxation with bile or duodenal reflux, inappropriate activation of trypsin, increased production of proteolytic enzymes,⁷ increased protein concentration in pancreatic juice leading to small ductular obstruction,⁸ direct toxicity to acinar cells,⁹ or, more likely, a combination of these mechanisms.

Acute Pancreatitis

Although acute pancreatitis is not classically included as a cause of chronic pancreatitis, clinicians have known for decades that a severe attack of acute pancreatitis, such as from an impacted biliary stone, can lead to chronic pancreatitis. In these scenarios, chronic pancreatitis is more likely to occur when a great portion of the gland has undergone necrosis. A recent analysis of nondrinkers who presented with acute pancreatitis due to choledocholithiasis (n = 26) or following endoscopic retrograde cholangiopancreatography (ERCP; n = 8) showed that 5 years after the event none had recurrent attacks, but 78% of those with severe acute pancreatitis (per the Atlanta criteria) and 24% of those with mild acute pancreatitis had evidence of endocrine or exocrine insufficiency.¹⁰ This study is all the more remarkable considering that rather insensitive testing was used to measure pancreatic function (bentiromide test, 72-hr fecal fat).

Idiopathic

No cause can be identified in approximately 20% of patients with chronic pancreatitis. These idiopathic cases fall into 2 categories: a variant characterized by prominent early-onset pain in the early twenties with very slow progression to calcification, diabetes, and