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Chronic pancreatitis, a comprehensive review and update. Part II: Diagnosis, complications, and management

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Introduction

Advances in research regarding chronic pancreatitis (CP) have improved our understanding of this disease. Despite this progress, the management of CP still remains less than optimal, with many patients who are symptomatic and have poor quality of life.

Optimal management begins with an accurate diagnosis, identifying the cause, assessing the reversibility of the cause, and then the evaluation followed by treatment of symptoms and complications. This review focuses on areas with updated recent evidence.

Diagnosis of CP

Accurate diagnosis of CP is often challenging especially in early stages of the disease where patients lack classical clinical evidence of CP.

Chronic pancreatitis is usually diagnosed with historical clinical information and with the results of radiographic studies and laboratory tests of pancreatic function.

Numerous diagnostic tests have been developed. The sensitivity and specificity of many of these tests are either poor or unknown, and some older tests such as bentiromide test, dual Schillings test, and olein absorption are not commonly utilized in current clinical practice.

Is histologic fibrosis the gold standard to diagnose chronic pancreatitis?

Histologic evidence of fibrosis and parenchymal atrophy is the most specific diagnostic finding; however, it is rarely available.

In addition, pancreatic fibrosis without inflammation or parenchymal destruction may be seen in asymptomatic individuals. This “bland fibrosis,” which is found associated with alcoholism, smoking, and ageing, should be distinguished from chronic pancreatitis. While this

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bland fibrosis is clinically silent, radiologic appearances may be indistinguishable from symptomatic individuals. It is still unclear if extensive fibrosis associated with chronic alcohol use can result in functional pancreatitic insufficiency.[1]

Fibrosis and atrophy in isolation do not indicate inflammation and may be the result of normal "aging" of the pancreas. Therefore, utmost caution should be used in diagnosing chronic pancreatitis based on endoscopic ultrasound (EUS) findings alone in the absence of an appropriate clinical presentation.

Pancreatic fibrosis without pancreatitis

1. Old age
2. Smoking
3. Alcohol use

Biochemical tests

Routine lab tests are not usually helpful in diagnosing CP. Lab tests might reflect the secondary effects of chronic pancreatitis such as anemia from malabsorption, fat-soluble vitamin deficiency (low 25-hydroxy vitamin D levels and low calcium levels), and occasional elevation of alkaline phosphatase secondary to biliary compression from fibrosis-related CP.

Specific biochemical tests are less useful than imaging in diagnosing CP, although there is some evidence that these functional tests can diagnosis CP at earlier stages than imaging studies (Table 1).

Serum amylase and lipase

In contrast to acute pancreatitis, measurement of amylase and lipase are not very useful in the evaluation of CP. Amylase and lipase may be elevated, normal, or low in CP. In acute exacerbations of CP, these enzymes might be elevated; however, serum concentrations seldom reach the threshold level seen in acute pancreatitis. A very high level of amylase in a classical chronic pancreatitis patient presenting with acute abdominal pain should prompt the physician to consider evaluation for other abdominal process such as bowel perforation or obstruction.[2]

Serum trypsin

Low levels (< 20 mg/dL) of serum trypsin (a.k.a., trypsinogen) are seen in patients with advanced chronic pancreatitis who have steatorrohea.[3] Levels from 20 to 29 mg/dL are indeterminant, but sometimes represent early CP.[4] While low concentrations (< 20 mg/dL)

Table 1
Pancreatic function testing.

Enzyme assay
1. Serum trypsin
Hormone stimulation tests
1. Secretin
2. Cholecystokinin
3. Combined secretin–CCK test
Stool testing
1. 72-hr quantitative fecal fat
2. Fecal elastase
3. Fecal chymotrypsin

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