for the diagnosis of chronic pancreatitis are 75% to 90% and 85%, respectively.

ERCP can reliably evaluate structural abnormalities of the pancreatic ductular system. ERCP allows detection of pancreatic duct changes, including ductal dilation, strictures, abnormal side branches, communicating pseudocysts, and ductal stones and leaks. ERCP is highly effective for visualizing these ductal and duct-related findings, with a sensitivity for the diagnosis of chronic pancreatitis of 71% to 93% and a specificity of 89% to 100%. The major limitation of ERCP is the development of procedure-related acute pancreatitis in up to 5% of patients. ERCP should not be used for diagnostic purposes but instead reserved for patients with established chronic pancreatitis when endoscopic therapy is recommended (discussed later).

Magnetic resonance cholangiopancreatography is a noninvasive diagnostic imaging modality that provides visualization of the pancreatic duct with images similar to those of ERCP but without the risk of precipitating acute pancreatitis. However, limited visualization of pancreatic duct side branches makes this test useful only for patients with advanced changes of the main pancreatic duct. A modification of MRI imaging with secretin stimulation enhances side branch visualization, but whether it aids in the diagnosis of chronic pancreatitis remains to be determined.

Endoscopic ultrasound (EUS) as a diagnostic imaging study for chronic pancreatitis relies on quantitative and qualitative parenchymal tissue and ductal findings. EUS appears to be equally or more sensitive than other tests of structure and function. An international consensus panel proposed the Rosemont criteria for diagnosing chronic pancreatitis. Major criteria include hyperechoic foci with shadowing that indicates pancreatic duct calculi and parenchymal lobularity with honeycombing. Minor criteria include cysts, a dilated main duct (≥3.5 mm in diameter), irregular pancreatic duct contour, dilated side branches (≥1 mm in diameter), hyperechoic duct wall, parenchymal strands, nonshadowing hyperechoic foci, and lobularity with noncontiguous lobules. In the absence of any of these criteria, chronic pancreatitis is unlikely, whereas with detection of four or more criteria, the disease is likely, even when other imaging and pancreatic function tests may still be normal.

Treatment

Malabsorption

Treatment of pancreatic exocrine insufficiency is best achieved with pancreatic enzyme supplementation. Most commercial preparations consist of pancreatin, which is the shock-frozen powdered extract of porcine pancreas containing lipase, amylase, trypsin, and chymotrypsin. For most patients, the recommended dose depends on the size and nature of the meal (i.e., fat content), residual pancreatic function, and therapeutic goals (i.e., elimination of steatorrhea, reduction in the abdominal symptoms of maldigestion, or improvement in nutrition).

Approximately 90,000 USP units of lipase per meal are needed for optimal fat absorption. Due to residual pancreatic lipase secretion and physiologic gastric lipase secretion, it is appropriate to begin therapy with at least 50,000 USP units of lipase with each meal and one half of that amount with snacks. Administration of acid-stable, encapsulated microspheres or microtablets filled with pancreatic enzymes has greatly increased the efficacy of enzyme supplementation.

In cases of gastric hyperacidity, proton pump inhibitors (PPIs) or histamine (H_2)-receptor antagonists should be used to reduce enzyme inactivation. If these methods do not provide improvement, the next step is to decrease dietary fat intake to less than 50 g per day and to substitute medium-chain triglycerides (MCTs), which do not require hydrolysis before absorption. Although clinically effective, patients usually do not like MCT fat because of poor palatability.

Other factors may accentuate steatorrhea, including concomitant small bowel bacterial overgrowth, which can occur in up to 25% of patients with chronic pancreatitis. Bacterial overgrowth may be caused by hypomotility due to inflammatory diseases of the head of the pancreas or chronic use of narcotic analgesics.

Pain

The greatest challenge in treating chronic pancreatitis is controlling abdominal pain. Pain may improve over time, but the course is not predictable, and improvement may take years. Therapy targets the mechanisms responsible for pancreatic pain, including pancreatic hyperstimulation, ischemia, obstruction of ducts, inflammation, and neuropathic hyperalgesia. Pain can develop in the early stages of chronic pancreatitis before morphologic changes can be demonstrated on imaging studies. Patients with chronic pancreatitis are at increased risk for pancreatic cancer, which may cause a change in the pain pattern, and extrapancreatic causes of pain must always be considered.

Pain management should proceed in a stepwise fashion and begin with lifestyle modifications such as alcohol and tobacco abstinence, a low-fat diet, and pancreatic enzyme supplementation, followed by a sequentially more aggressive and invasive approach for symptomatic failures, although it should be recognized that placebo alone is effective for up to 30% of patients. Several approaches can be considered for chronic pain relief.

1. Tobacco and alcohol abstinence. Abstention may decrease the frequency of painful attacks and reduce the likelihood pancreatic function deterioration and development of pancreatic cancer.
2. Nutrition and hydration. Small meals that are low in fat may help to some degree. Although the evidence is anecdotal, encouraging hydration may prevent exacerbations of pancreatitis. Supplementation of dietary fat with MCTs may be of benefit, possibly as a result of antioxidant effects of MCTs or the minimal increases in plasma cholecystokinin (CCK) levels associated with MCTs compared with other sources of dietary fat.
3. Analgesics. Most patients with pain require analgesics. Tramadol, a less potent opioid, is commonly used initially. The risk of addiction to opioids is not known, but studies of opioid use in other chronic pain syndromes suggest rates of less than 20%. Patients with previous addictive behaviors such as substance abuse (alcohol and tobacco included) are at greater risk for analgesic abuse and addiction.
4. Secretion suppression. Oral pancreatic enzyme replacement is thought to blunt pain by reducing endogenous CCK