the enteral route or those in whom enteral access cannot be maintained.

Management of Recurrence and Necrosis

Gallstone Pancreatitis

The risk of gallstone pancreatitis recurrence is as high as 50% to 75% within 6 months of the initial episode, and cholecystectomy before discharge is recommended for patients with mild attacks of pancreatitis. Cholecystectomy is often delayed in patients with severe pancreatitis to allow for better exposure of the ductal anatomy at the time of surgery. Urgent ERCP (Video 38-1) with identification and clearance of bile duct stones is recommended for patients with evidence of ongoing biliary obstruction, as suggested by imaging and laboratory data. Biliary sphincterotomy leaving the gallbladder in situ is considered an effective alternative for those who are not candidates for cholecystectomy.

Sterile Pancreatic and Extrapancreatic Necrosis

Sterile pancreatic necrosis usually is treated with supportive medical care during the first several weeks, even in patients with multiple organ failure. After the acute pancreatic inflammatory process has subsided and coalesced into an encapsulated structure (e.g., walled-off pancreatic necrosis), débridement may be required for intractable abdominal pain, vomiting caused by extrinsic compression of stomach or duodenum, or persistent systemic toxicity. Débridement is delayed for at least 4 to 6 weeks after the onset of pancreatitis and can be performed by a combination of endoscopic, radiologic, and surgical techniques. Asymptomatic pancreatic necrosis does not warrant intervention, regardless of the extent and location.

Infected Pancreatic and Extrapancreatic Necrosis

Infected pancreatic necrosis is best treated with drainage or débridement, or both. In most cases, the diagnosis of infected necrosis is confirmed by fine-needle aspiration before intervention, but because false-negative results can occur, débridement warrants consideration when infected necrosis is suspected, even if infection is not documented. The consensus is that the best outcomes are achieved when surgery is delayed for a minimum of 4 weeks after the onset of disease to allow liquefaction of necrotic tissues and a fibrous rim to form around the necrosis (i.e., walled-off pancreatic necrosis). Patients with infected necrosis are initially treated with broad-spectrum antibiotics and medical support to allow encapsulation of the necrotic collections, which may facilitate intervention and reduce complications of bleeding and perforation. When there is dramatic clinical deterioration, delay is not feasible, and early intervention is required.

Traditional management of infected pancreatic necrosis has been open surgical necrosectomy with closed irrigation by indwelling catheters, necrosectomy with closed drainage without irrigation, or necrosectomy and open packing. The open surgical approaches are associated with a high morbidity (34% to 95%) and mortality (11% to 39%) rates. A more conservative step-up approach using percutaneous catheter drainage as the initial treatment has gained favor. If the percutaneous approach fails, it is followed by a less invasive, video-assisted retroperitoneal débridement (VARD) or endoscopic transluminal drainage with or without necrosectomy (Video 38-2), provided expertise is available.

CHRONIC PANCREATITIS

Definition and Epidemiology

Chronic pancreatitis is characterized by inflammation, fibrosis, and irreversible loss of acinar (exocrine) and islet (endocrine) cell function. This disorder contrasts with acute pancreatitis, which is usually nonprogressive. The two conditions may overlap because recurrent attacks of acute pancreatitis may lead to chronic pancreatitis, and individuals with chronic pancreatitis may experience exacerbations of acute pancreatitis. The annual incidence of chronic pancreatitis ranges from 5 to 12 cases per 100,000 people, and the prevalence is about 50 cases per 100,000 people.

Pathology

Chronic pancreatitis can be classified as nonobstructive or obstructive (Table 38-3). The most common cause of the nonobstructive type is chronic alcoholism (70%). Alcohol can cause episodes of acute pancreatitis, but at the time of the initial attack, structural and functional abnormalities often indicate underlying chronic pancreatitis.

Because most alcoholics do not develop pancreatitis, the presumption is that unidentified genetic, dietary, or environmental influences must coexist with alcohol abuse. For example, epidemiologic data have implicated smoking as a causal, dose-dependent cofactor in chronic pancreatitis. The combined effect of smoking and alcohol is synergistic and contributes profoundly to the development and progression of the disease.

If alcoholism is excluded, 20% of U.S. patients have chronic pancreatitis with no immediately demonstrable cause. Gallstone pancreatitis, the major cause of acute pancreatitis, rarely leads to chronic pancreatitis. Calcific pancreatitis is a major cause of chronic pancreatitis in South India and other parts of the tropics. Autoimmune pancreatitis, genetic mutations *(CFTR, SPINK1, PRSS1, CTRC, CASR)*, obstruction (e.g., tumors, sphincter of Oddi dysfunction, pancreas divisum), hypertriglyceridemia, and hypercalcemia are potential causes of cases initially labeled idiopathic.

Clinical Presentation

Most patients with chronic pancreatitis experience episodic or continuous pain. Occasionally, patients exhibit exocrine or

TABLE 38-3 CAUSES OF CHRONIC PANCREATITIS

NONOBSTRUCTIVE CAUSES	
Alcohol	Metabolic: hypertriglyceridemia, hypercalcemia
Idiopathic (10%-20% of total cases)	**OBSTRUCTIVE CAUSES**
Tropical	Benign obstruction: sphincter of Oddi dysfunction, pancreas divisum, posttraumatic
Genetic (cationic trypsinogen, *CFTR*, chymotrypsin C, *SPINK1*)	Neoplastic obstruction: tumors of the ampulla or ductal system
Traumatic	
Autoimmune	
Vascular ischemia	

CFTR, Cystic fibrosis transmembrane conductance regulator; SPINK1, serine peptidase inhibitor Kazal type 1.