# Management of Gallstones and Associated Complications

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Cover Illustration by Scott M. Holladay
I. INTRODUCTION

Management of cholelithiasis and associated complications is a frequent problem encountered in general surgery. Cholelithiasis affects approximately 10% to 15% of adults in the United States, with an estimated 1 million new cases diagnosed annually. Although most patients with gallstones remain asymptomatic, a wide range of conditions secondary to cholelithiasis can develop, ranging from limited episodes of pain to potentially lethal cholecystitis, pancreatitis, cholangitis, or carcinoma.1 Treatment for patients with gallstones has changed dramatically during the past 10 years and may now involve multiple nonsurgical specialties. This review will present the common manifestations of cholelithiasis and discuss potential management options and outcomes. Sample board review questions with detailed answers are provided at the end of this manual for self-assessment.

II. PATHOPHYSIOLOGY OF GALLSTONE-RELATED DISEASES

Most gallstones in humans are primarily composed of cholesterol, although occasionally pigment stones can develop, which are usually related to hemolytic disease. Well-known risk factors for cholesterol stone formation include female sex, multiparity, obesity, and rapid weight loss. The incidence is also high in certain ethnic groups, such as Native Americans. Formation of cholesterol gallstones requires supersaturation of bile with cholesterol, accelerated nucleation of cholesterol crystals, and gallbladder hypomotility.2 The supersaturation of bile is usually caused by cholesterol hypersecretion, which occurs via multiple mechanisms. Cholesterol secretion into bile increases with age and after rapid weight loss. Excessive cholesterol synthesis occurs in obese or pregnant patients and is also seen with estrogen use. Pregnant women also have a decreased gallbladder emptying time. Cholesterol supersaturation, especially when coupled with excess levels of the secondary bile salt deoxycholate, causes gallbladder inflammation with secretion of procrystallizing proteins (ie, immunoglobulins and mucous glycoproteins).2 This condition leads to crystal formation, a process promoted by hypomotility of the gallbladder.

Most symptoms and clinical manifestations of gallstones are associated with an obstruction of the cystic duct or common bile duct (CBD). This blockage may be intermittent, partial, or complete. Patients with a temporary cystic duct obstruction commonly present with intermittent pain or biliary colic, believed to result from gallbladder distention secondary to the duct obstruction. Acute cholecystitis is most often associated with stagnant bile that develops from cystic duct obstruction and secondary mucosal inflammation, a release of inflammatory mediators, alterations in perfusion, and secondary infection. Cholangitis occurs with CBD obstruction accompanied by biliary stasis and eventual infection with bacteria from the portal circulation passing from the hepatic sinusoids. Gallstone pancreatitis results from passage of a CBD stone through the sphincter of Oddi and into the duodenum. It is unclear how this leads to pancreatitis; however, the condition may be related to bile reflux into the pancreatic duct and to ductal hypertension with ongoing pancreatic secretion against the obstruction.

III. MANAGEMENT OF CHOLELITHIASIS

ASYMPTOMATIC CHOLELITHIASIS

Asymptomatic cholelithiasis is a common occurrence, with most patients (70% to 80%) remaining free of symptoms during their lifetime.3 An estimated 2% to 4% of asymptomatic patients will develop symptoms each year, and only 10% of those patients present with serious complications.4 The average patient with asymptomatic cholelithiasis often does not require therapy.