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Infections of the Biliary Tree

Series Editor: Stephanie Nagy-Agren, MD

*Assistant Professor of Internal Medicine, University of Virginia School of Medicine, Charlottesville, VA
Chief, Division of Infectious Diseases, Veterans Affairs Medical Center, Salem, VA*

Contributors:

Radu Clincea, MD

Chief of Infectious Diseases, Veterans Affairs Healthcare Center, Orlando, Florida

Himabindu Gaddipati Chalasani, MD

Resident in Internal Medicine, University of Virginia, Roanoke-Salem Program, Roanoke, VA

Kishore V. Gaddipati, MD

Resident in Internal Medicine, University of Virginia, Roanoke-Salem Program, Roanoke, VA

Table of Contents

Introduction	2
Cholecystitis	2
Cholangitis	4
Liver Abscesses	7
AIDS-Related Biliary Tract Infections	10
References	11

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next page ↓

Infections of the Biliary Tree

Radu Clincea, MD, Himabindu Gaddipati Chalasani, MD, and Kishore V. Gaddipati, MD

INTRODUCTION

Infections of the biliary tree are frequent causes of acute and chronic abdominal pain syndromes. Acute cholecystitis and ascending cholangitis are primary inflammations of the gallbladder and bile ducts, respectively, that can be caused by infection. Pyogenic liver abscesses often occur after obstruction of the common bile duct and the development of subsequent ascending (suppurative) cholangitis. Both cholecystitis and cholangitis are of particular concern in immunosuppressed hosts, particularly patients with AIDS. This manual reviews infections of the biliary tree by focusing on these topics. Case examples will be used on occasion to illustrate major points.

CHOLECYSTITIS

DEFINITION, EPIDEMIOLOGY, AND ETIOLOGY

Acute cholecystitis is an inflammation of the gallbladder resulting from obstruction of the cystic duct and subsequent bacterial invasion and overgrowth. In the United States, cholelithiasis is the cause of cystic duct obstruction in more than 90% of cases of acute cholecystitis, with women being affected 2 times more often than men.¹ The pathologic process of cholecystitis may be acute, chronic, or—more often than not—a combination of both types. For example, it has been shown that approximately 95% of gallbladders excised for acute cholecystitis exhibit fibrosis and other signs of chronic inflammation.²

Under physiologic conditions, the bile is sterile, and bactibilia is always a secondary event. Gallstone impaction of the cystic duct or of the common bile duct is thought to alter the local microenvironment, allowing the overgrowth of bacteria in the duodenum and invasion of the biliary tree through the ampulla of Vater. Moreover, previous endoscopic manipulations or surgical interventions involving the biliary tract predispose patients to polymicrobial infections, including infections by anaerobes or *Pseudomonas aeruginosa*.

The inflammatory reaction/infection of cholecystitis may involve the full thickness of the gallbladder wall and may lead to ischemia with transmural necrosis, empyema, gangrenous cholecystitis, emphysematous cholecystitis, perforation with pericholecystic or intra-peritoneal abscess formation, and frank peritonitis. Perforation of the gallbladder occurs in 10% to 15% of cases of acute cholecystitis² but generally remains contained locally by the omentum and serosa of contiguous viscera. However, rupture into an adjacent viscus is not uncommon.

Acalculous cholecystitis tends to occur in critically ill patients in the setting of an intensive care unit (ICU) after surgery; in patients with extensive burns, systemic sepsis, or trauma; or in patients receiving total parenteral nutrition for more than 3 weeks with no oral intake. The pathophysiologic mechanisms leading to acalculous cholecystitis are not understood but may involve transmural ischemia with subsequent necrosis of the gallbladder. Acute acalculous cholecystitis represents between 2% and 12% of cases of acute cholecystitis¹ and carries higher morbidity and mortality rates than does the calculous type.

Microbiologically, the pathogens involved in cases of cholecystitis are aerobic, enteric, gram-negative bacilli (eg, *Escherichia coli*; *Klebsiella*, *Enterobacter*, *Proteus* species) or aerobic gram-positive organisms (eg, *Enterococcus*, *Streptococcus*, *Staphylococcus* species). Anaerobes (eg, *Bacteroides*, *Clostridium*, *Fusobacterium* species) are identified in as many as 15% of isolates from patients with cholecystitis,³ generally as part of polymicrobial infections. *Clostridium perfringens* can be identified in approximately 45% of cases of emphysematous cholecystitis,⁴ which is characterized by the presence of gas within the gallbladder wall or lumen and occurs most often in elderly patients or in male patients with diabetes mellitus. Infection with *Salmonella* or *Campylobacter* species can result in acute acalculous cholecystitis.

CLINICAL MANIFESTATIONS

Acute cholecystitis most often presents with pain, presumably resulting from distention of the gallbladder wall, and can lead to vascular compromise and necrosis. The pain may start in the midepigastria region but is