Acute Ascending Cholangitis

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Pancreatic cancer is the fourth leading cause of death in men and women in the United States. In 2003, Jemal et al. estimated that 30,700 people were diagnosed with pancreatic cancer and 30,000 people died from the disease. Treatment options are limited and relatively ineffective. Pancreatic cancer has a five-year survival rate of 1%–4% (Wolff et al., 2000), with 90% of patients succumbing to their disease within the first year of diagnosis (Rosenberg, 2000). Many comorbid conditions are associated with pancreatic cancer, including acute ascending cholangitis (AAC), a bacterial infection of the biliary tract that is caused by bile stasis secondary to obstruction.

**Pathophysiology**

Under normal circumstances, bile drains from the gallbladder to the duodenum via the common bile duct. In healthy people, several mechanisms are in place to prevent infection of the biliary system. The sphincter of Oddi prevents intestinal contents from refluxing into the common bile duct, the tight junctions between hepatocytes protect from transient bacteremia, and Kupffer cells maintain sterility of the biliary system with phagocytizing organisms (Sung, Costerton, & Shaffer, 1992). Additionally, bile has antibacterial properties of its own, including immunoglobulin A and bile salts (Sung et al.), and continuous flushing of biliary conduits occurs as bile flows from the liver to the intestine (Hanau & Steigbigel, 2000).

When the biliary system becomes obstructed, most commonly from bile duct stones, infection can occur. Jean Martin Charcot, MD, first described the relationship between common bile duct obstruction and sepsis in 1877. He described the triad of fever, jaundice, and right upper-quadrant pain, which later became known as Charcot’s triad (Lipsett & Pitt, 1990).

AAC is an infection of the biliary system in which bacteria present in the bile is unable to drain adequately because of an obstruction, stones, pancreatic cancer, or reflux of bile from the jejunum into the biliary system following a choledochojejunostomy (Hanau & Steigbigel, 2000). Duodenal microorganisms from the portal vein are thought to be the primary sources of biliary infection (Sung et al., 1992). Enteric flora are the most common organisms present. Aerobes constitute the majority of infections, with E. coli comprising 59% and Klebsiella causing 13.9% of the infections (Hanau & Steigbigel). Anaerobes are rare in biliary disease, but if present, bacteroides species and Clostridia organisms are the most common (Hanau & Steigbigel).

Anaerobes tend to be associated with more serious clinical illness than purely aerobic infections (Csendes et al., 1996; Shimada, Noro, Inamasu, Urayama, & Adachi, 1981). Bile cultures are positive in 80%–100% of cases (Hanau & Steigbigel, 2000). Biliary infections are often polymicrobial (48%–61%), although blood cultures are usually positive for only one organism (Surawicz & Owen, 1995). Bacteremia occurs less frequently but is detected in 21%–83% of patients with cholangitis (Hanau & Steigbigel).

Since 1950, the etiology of AAC has shifted away from stone-induced disease to disease caused by obstruction by cancer (Lipsett & Pitt, 1990). This shift in etiology has been attributed, in part, to the use of long-term endoprostheses in patients with unresectable malignant tumors (Lipsett & Pitt). Endoprostheses, also known as endoscopic biliary stents or catheters, are used to decompress an obstructed biliary system. They are placed into the common bile duct to facilitate the drainage of bile into the jejunum, thereby relieving the pressure on the biliary system. The systemic toxicity that occurs in acute obstructive cholangitis results from entry of bacteria into the blood (Surawicz & Owen, 1995).

The primary infection mainly results from bacteria in the duodenum gaining entry and directly ascending into the biliary system, hence, ascending cholangitis (Surawicz & Owen, 1995). Increased intraductal pressure, as a result of the stricture, and bacterial growth lead to reflux of biliary contents and obstruction of the system. The toxicity, therefore, is more severe than the bacteremia from the portal vein, and the infection may spread systemically within hours.