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# **Introductory Chapter: Biliary Tree**

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Hesham Abdeldayem

Additional information is available at the end of the chapter

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## **1. The biliary tree**

Bile flows through canaliculi formed by the walls of the hepatocytes, then into successively larger ducts: the term biliary tree is derived from the arboreal branches of the bile ducts. The liver produces 500–1000 mL of bile per day. The bile ducts, gallbladder, and sphincter of Oddi modify, store, and regulate the bile flow [1].

## **2. Biliary stones**

Biliary stones are formed as a result of failure to maintain biliary solutes (primarily, cholesterol and calcium salts) in a soluble state. The pathogenesis is multifactorial and involves cholesterol supersaturation, crystal nucleation, and gallbladder dysmotility. The vast majority of patients are asymptomatic, often discovered at laparotomy or during abdominal imaging. Over time, asymptomatic gallstones can progress to symptomatic disease. Prophylactic cholecystectomy is not generally indicated in patients with asymptomatic gallstones. Prophylactic cholecystectomy is considered for children with gallstones, patients with sickle cell disease (as cholecystitis can precipitate a crisis with substantial operative risks), and large gallstones (>2.5 cm), porcelain gallbladder (calcified gallbladder wall). Acute cholecystitis results from a stone impaction at the gallbladder-cystic duct junction. The extent and the progression of inflammation are related to the duration and degree of obstruction. In severe cases, this process can lead to ischemia and necrosis of the gallbladder wall. More frequently, the gallstone is dislodged, and the inflammation gradually resolves. Intrahepatic stones are more prevalent in Asia. They are associated with prolonged partial BD obstruction, as in sclerosing cholangitis, benign and malignant biliary strictures, choledochal cysts, and biliary parasites. Mirizzi syndrome is a form of obstructive jaundice, first described by Mirizzi in 1948, caused

by a stone or stones impacted in the neck of the gallbladder or the cystic duct, such that the common hepatic duct (CHD) is narrowed. It occurs in about 0.1–0.7% of patients who have gallstones [2].

Gallstone ileus results from fistula formation from the biliary tract to the intestine. It is mainly a disease of the elderly and of women. The advanced age and medical comorbidities contribute to the high morbidity and mortality. Clinical suspicion for this entity must exist. It is often a consequence of inflammation of the gallbladder, adhesions to adjacent bowel, with subsequent pressure and ischemia causing a gallstone to erode into the bowel, resulting in fistula formation. Most of the stones pass without consequence. Obstruction occurs if the stone is of large enough size, mostly bigger, greater than 2–2.5 cm. The point of obstruction is most often in the terminal ileum because of its smaller diameter, but it can occur throughout the GI system [3].

### **3. Biliary strictures**

Biliary strictures are usually caused by inflammatory conditions such as chronic pancreatitis, cholelithiasis and choledocholithiasis, primary sclerosing cholangitis, stenosis of the sphincter of Oddi, duodenal ulcer, and Crohn's disease. Drugs follow injury at a primary biliary operations as laparoscopic cholecystectomy. Strictures also can complicate open cholecystectomy, common bile duct exploration, gastrectomy, and hepatic resection. Ischemia of the bile duct, unnecessary dissection around the bile duct during cholecystectomy or bile duct anastomosis can divide or injure the major arteries of the bile duct that run in the 3 o'clock and 9 o'clock positions. Marked local inflammatory response can develop in association with bile leakage, which occurs with many bile duct injuries. This results in fibrosis and scarring in the periductal tissue, contributing to stricture formation [4].

### **4. Biliary fistulas**

Biliary fistulas are classified by the etiology as spontaneous, posttraumatic, iatrogenic, or postoperative. They can be classified by the site of exit as internal fistulas (the most frequent site is to the GI tract, particularly the duodenum) and external (commonly postoperative) [4].

### **5. Cholangiocarcinoma**

Cholangiocarcinoma is defined as primary malignancy originating from BD epithelium. It is the second most common primary hepatic neoplasia. These cancers tend to grow perpendicularly to, and horizontally along, the bile duct, and therefore, tumors that are detected by imaging tend to be underestimated. The anatomic relationship of the distal bile duct to the pancreas, duodenum, portal vein, and hepatic artery can also make removal of these tumors

technically challenging. Although these tumors can occur at any level of the biliary tree, nearly two-thirds occur at the bifurcation of the bile duct (hilar cholangiocarcinoma), where they are often referred to as Klatskin tumors. Surgery remains the primary curative modality. Most cases cannot undergo curative resection because of patient-related causes (medical comorbidities), local anatomic causes (local tumor extension), and tumor biology (metastatic disease). Three main subgroups are (1) hilar tumors involving the confluence of the left and right HDs and the CHD, (2) mid-duct tumors involving the supraduodenal CBD, and (3) distal involving the intraduodenal bile duct. This classification is based upon on the technique required for curative resection. Hilar tumors require excision of the CHD and frequently concomitant hepatic parenchymal resections; mid-duct tumors rarely require concomitant hepatic resections; distal tumors necessitate pancreaticoduodenectomy [4].

## Author details

Hesham Abdeldayem

Address all correspondence to: [habdeldayem64@hotmail.com](mailto:habdeldayem64@hotmail.com)

National Liver Institute, Menoufia University, Egypt

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