**Etiologies of Cholangitis**

**Cholangitis** Characterized by Charcot’s triad of fever, jaundice and abdominal pain, present in 50-75% of patients. Addition of confusion and hypotension -> Raynaud’s pentad. Primarily caused by bacterial infection.

->**Most important predisposing factor** is biliary obstruction and stasis. Elevated intrabiliary pressure 1) promotes migration of bacteria from portal circulation into biliary tract and 2) impairs intrinsic hepatic host defense mechanisms such as hepatic tight junctions, Kupffer cell function, bile flow, and IgA production.

->**Bacteriology**: gram negatives and enterococci. E coli 20-25%, Klebsiella 15-20%, Enterobacter 5-10%, Enterococcus 10-20%, anaerobes (Bacteroides and Clostridia) present as mixed infection but are rarely the sole infecting organisms (unclear what role anaerobes play in pathogenesis of acute cholangitis).

->**Diagnosis** U/S is first line to look for CBD dilatation and presence of stones. Will be negative 10-20% of cases.

->**Treatment** Biliary drainage via ERCP or percutaneous drainage, antibiotics to cover gram negatives and anaerobes, as well as enterococcus. Quinolones are excreted into bile, amp/gent/flagyl provides excellent coverage.

**Post instrumentation** Instrumentation of the sphincter of Oddi post ERCP, stenting, or choledochal surgery compromised the sphincter’s function and may allow for pathogenic bacteria to enter the biliary system.

**Choledocholithiasis**- Most common cause of cholangitis in the US. Presence of foreign body such as GS acts as a nidus for bacterial colonization of organisms that pass through a competent sphincter in small numbers. GS in common bile duct much more likely to have positive culture of biliary fluid. Stones may be retained post cholecystectomy.

**Carcinoma of the ampulla of Vater, pancreas, GB or common duct**

**Extrinsic compression of CBD** Usually by metatstatic can (breast or GI tract) involving porta hepatitis lymph nodes. Rarely, compression from large duodenal diverticulum.

**Choledochal cyst** Cystic dilatation of CBD, usually congenital. At higher risk of cholangiocarcinoma.

**Biliary Stricture** Result from surgical trauma in 95% of cases. Reamineder caused by blunt external injury to abdomen, pancreatitis, or erosion of the duct by a GS.

**Biliary Fistula or biliary enteric anastomosis**

**Biliary parasitosis :**

- **Ascariasis:** Globally, one of the most common helminthic parasites. Can cause biliary colic, cholangitis, acute cholecystitis, pancreatitis and rarely, hepatic abscess. Worms may be visible by U/S in the common bile duct. Large biliary worms can be removed with ERCP.

- **Echinococcus** Adult worm lives in intestines but hydatid cyst can form in the liver. Up to 25% of these cyst can rupture into the biliary tree, leading to obstructive jaundice, cholangitis and occasionally cholangiolytic abscesses. Can also rupture into peritoneal cavity or other
Opisthorchis sinensis “Chinese liver fluke”, previously called Clonorchis- trematode found in China, Japan, Korea, Taiwan, Vietnam. Infxn from eating raw fresh water fish. Can cause obstructive jaundice, cholangitis, intrahepatic calculi. Chronic infection can lead to cholangiocarcinoma. Meacercariae (infective stage) attach to CBD and migrate to intrahepatic ducts. Prefer smaller branches of left lobe of liver. Migration causes trauma to the bile duct epithelium leading to ulceration and desquamation -> thickening and scarring of biliary system (“encapsulating fibrosis”). Cirrhosis can develop in late infection. Opisthorchis causes similar disease.

Fasciola Hepatica – “sheep liver fluke” Infxn after eating raw vegetable in sheep raising areas. Worms penetrate duodenum, migrate through peritoneum, and enter biliary system through liver capsule and parenchyma. Larvae in bile duct cause inflammation and fibrosis. Causes cystic dilatation, obstruction, and periportal cirrhosis. Eggs be nidus for stone formation.

Oriental cholangiohepatitis” Also known as “recurrent pyogenic cholangiohepatitis” Characterized by intrahepatic stone formation. In patients from Southeast Asia, but has been reported in India, Mexico, Central and South America. Etiology unknown but may be due to prior infection of biliary system with Opisthorchis and Ascaris which induce ductal injury an stricture from exuberant inflammatory response and may lead to subsequent secondary bacterial infection. Bacterial colonization of biliary tree from portal vein also plays causative role - enteric bacteria possess B-glucuronidase activity which causes deconjugation of bilirubin glucuronide -> deconjugated bilirubin precipitates with calcium in the bile and forms pigment stones, which then obstruct intrahepatic ducts leading to cholangitis However, parasites recovered from stool in only 5-25% of pts (See NEJM CPC on Recurrent Pyogenic Cholangiohepatitis 345:817-823, 2001)

Caroli’s Disease Rare disease resulting from congenital dilation of the intrahepatic bile ducts. Usually silent for 5-20 years after birth but manifests with episode of bacterial cholangitis. GS can form withing affected dilated ducts.

Primary Sclerosis Cholangitis Can present with acute cholangitis.