Small Domestic Animal Paper - Sample Long Answer Question

Cholecystitis and cholangiohepatitis are part of a spectrum of inflammatory disorders of the hepatobiliary system in cats.

(a) Differentiate between the terms cholecystitis, cholangitis and cholangiohepatitis, and explain an anatomical feature of the cat that predisposes to combined biliary and pancreatic involvement (6 marks)

Cholecystitis: inflammation confined to the gallbladder (1). Cholangitis: inflammation of the bile duct(s)/biliary epithelium (1). Cholangiohepatitis: extension of ductal inflammation into the adjacent hepatic parenchyma (1), such that cholangitis and cholangiohepatitis form part of a continuum (1). In the cat, the common bile duct and the major pancreatic duct share a common entry into the duodenum (1), so infectious/inflammatory disease can spread between the biliary tract and the pancreas, and concurrent pancreatitis is common (1).

(b) Describe the two main recognised forms of feline cholangitis/cholangiohepatitis, including for each the proposed pathogenesis and characteristic histologic features (8 marks for each).

Neutrophilic (suppurative/exudative) cholangitis-cholangiohepatitis:

Relatively common in cats (1), resulting from ascending bacterial infection of the biliary tree (1), often in association with extrahepatic bile duct obstruction, pancreatitis, or inflammatory bowel disease (1) (Triaditis). Escherichia coli is the most common isolate; Bacteroides, Klebsiella, streptococci and clostridia are also reported (1). Acute stage: portal oedema and a neutrophilic infiltrate, with neutrophils within and emigrating through bile duct epithelium (1). Progression may lead to inflammatory cells within hepatic lobules, periportal hepatocellular necrosis, and suppuration/abscessation (1). A mixed subacute stage shows neutrophils, lymphocytes and plasma cells in portal areas with bile duct proliferation and periportal to bridging fibrosis (1); chronic disease shows concentric periportal fibrosis and pseudo-lobule formation (1).

Lymphocytic cholangitis (lymphocytic portal hepatitis):

A slowly progressive disease of cats (1), with a proposed immune-mediated pathogenesis (1) portal infiltrates are predominantly T-cell, often with accompanying B-cell aggregates (1). Histologically: lymphocytic infiltration of the portal areas (1), with variable bile duct/oval cell proliferation (1) and peribiliary, portal-to-bridging fibrosis (1). It progresses slowly over months to years (1), in contrast to the more acute/fulminant course that may be seen with neutrophilic disease (1).

(c) Describe the gross (3 marks) and histologic changes (3 marks) that may be found in a cat with severe suppurative cholangiohepatitis and outline the pathogenesis of ascending bacterial infection of the biliary tract (7 marks).

Gross: the liver is swollen, soft and pale (1); few to many small, non-encapsulated, sometimes miliary suppurative foci are visible beneath the capsule and on cut surface (1); lesions elsewhere reflect septicaemia and jaundice/icterus (1).

Histologic: larger bile ducts contain neutrophilic exudate/bacterial colonies (1); neutrophilic infiltration and degeneration of bile duct epithelium (1); periportal hepatocyte necrosis and suppuration/microabscess formation in severe cases (1).

Pathogenesis of ascending bacterial infection: Bile itself contains antimicrobial factors (e.g. beta-defensins, bile acids) that are normally inhospitable to most bacteria (1). Enteric bacteria, typically coliforms, ascend the biliary tree from the duodenum (1), a process facilitated by bile stasis due to mechanical or functional obstruction, e.g. cholelithiasis, extrahepatic obstruction, or gallbladder mucocoele (1). Inflammatory and proliferative responses within the ducts themselves further interfere with bile flow (1), creating a self-perpetuating cycle that promotes ductular spread of infection (1). Bacteria may also reach the liver haematogenously and localise in the peribiliary vascular plexus or spread from sinusoidal Kupffer cells or foci of hepatic necrosis by direct extension into ducts or via portal lymphatics (2).