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Commentary

General Application about Hepatic Bile Duct

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DESCRIPTION

The right and left hepatic bile ducts emerge from the liver and unite at the portal hepatics to form the common hepatic duct, they are joined by the cystic duct from the gall bladder to form the common bile duct, large bile ducts are lined by columnar epithelium with thicker fibrous walls, in Primary Sclerosing Cholangitis (PSC) and IgC4 related cholangitis, the large bile ducts or extra hepatic bile duct is stenosis.

The human liver arises from hepatic diverticulum of the foregut during the third or fourth week of gestation, the left and right vitelline veins around the foregut communicate to each other and form sinusoids, the umbilical veins pass on each side of the liver and connect to the hepatic sinusoids, as the embryo develops, blood supplying this region, delivers nutrients from the yolk sac, placenta, and gut, hepatocyte precursors, hepatoblasts, arise from endodermal cells at the front of the diverticulum and invade the mesoderm of septum transversum.

Hepatoblast cords develop into anastomosing tubular structures with central bile canaliculi that eventually communicate with the bile ducts, most hepatoblasts produce Alpha Fetoprotein (AFP) and differentiate into hepatocytes, numerous hematopoietic cells are found in the sinusoids even at birth and surround immature hepatocytes, but they are largely gone from the liver by 4 weeks of age.

Hepatoblasts adjacent to the portal mesenchyme differentiate into a layer of duct progenitors called the ductal plate; the ductal plate gradually becomes bilayer and forms ductal segments with lumina.

Remodeling of the embryonic ductal plate causes neonatal billary atresia, congenital hepatic fibrosis, Caroli's disease, microhamartoma, choledochal cyst, and polycystic disease in which there is a genetic abnormality in cholangiocytic cilia leading to disruption of fluid transport and cholangiocyte proliferation, the common bile duct, left and right hepatic ducts, and gallbladder develop in the stalk region of the hepatic diverticulum, these ducts are connected with the ductal plate at the cranial end of the diverticulum, the liver occupies most of the abdominal cavity in the third month of gestation.

The cytoskeleton supporting the hepatic structure consists of microtubules, microfilaments, and intermediate filaments, microtubules that contain tubulin control subcellular motility, vesicle movement, and secretion of plasma protein or glycoprotein, microfilaments made up of actin are cotractile and are important for the motility of bile canaliculus and for the bile flow, intermediate filaments consisting of cytokeratins are essential for the stability and special organization of hepatocytes.

Hepatic stellate cells are known as fat storing cells, stellate cells, Ito cells, or lymphocytes, they lie within the sub endothelial space, cytoplasmic droplets contain abundant vitamin A in the form of retinol palmitate, which can be identified by their immune reactivity to smooth muscle actin, when the droplets are scanty, they resemble fibroblasts, which contain actin and myosin and contract in response to endothelin-1 and substance P to regulate blood flow and to influence portal pressure, in hepatocellular injury, hepatic Kupffer cells are activated, releasing many cytokines, stellate cells transform to myofibroblast.

Pit cells are natural killer lymphocytes attached to the sinusoidal surface of the endothelium and are numerous in sinusoids of zone I, they are short lived cells that are renewed from circulating large granular lymphocytes, they have characteristic granules which contain perforin injuring cell membrane and have a role for killing tumor cells and virus infected hepatocytes.

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