

Portal Hypertension Model in Pigs

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Every year in western countries millions of people are diagnosed with portal hypertension (PHT) which rapidly complicates hepatic cirrhosis and causes esophageal varices, ascites and encephalopathy.

It determines higher risk of morbidity and mortality and contributes to elevate costs for the Health Care System. PHT is defined as the elevation of the portal vein pressure gradient over 5-10 mmHg. Common causes are: Pre-hepatic such as portal vein thrombosis or congenital atresia; intra-hepatic as liver cirrhosis, hepatic fibrosis and less commonly non-cirrhotic causes such as schistosomiasis, massive fatty change and granulomatous diseases; post-hepatic including obstruction that occurs at any level between liver and right heart (i.e., Budd Chiari syndrome and veno-occlusive disease VOD).

The pathophysiology of portal hypertension is explained by the increasing of vascular resistance into the blood flow of the liver; as consequence the hepatic micro vasculature is compressed by regenerative nodules and fibrotic scars of the cirrhosis.

When the portal circulation is obstructed, whether it is within or outside the liver, a remarkable collateral circulation develops to carry portal blood into the systemic veins. Such picture occurs in the setting of the portal vein thrombosis or in case of congenital atresia. In adults causes are more often due to malignancies or hypercoagulable status while in pediatric patients, the portal vein thrombosis is usually originated by the obliteration of the umbilical vein and of the Arantius duct. In the childhood, secondary cause of PHT might be the atresia of the portal trunk which leads to the formation of a "portal cavernoma".

Considering the intra-hepatic causes, HBV and HCV related cirrhosis represent the most frequent etiology of end stage liver disease which is associated to PHT in Western countries. Alcohol-induced

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induce PHT in pigs. This preclinical model is useful in the investigation of splanchnic blood flow and showed to be a reliable option to further

enquire the clinical picture of the small for size syndrome after partial liver transplantation and of the post hepatectomy liver failure.

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