Changes of platelet function in experimental biliary cirrhosis: intracellular calcium homeostasis

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Introduction: In liver cirrhosis, several haemostatic abnormalities occur, mostly episodes of bleeding, but there is a 9-20 % risk of thrombosis. Some extrinsic and intrinsic factors are altered in these patients and their role in the development of these alterations is not completely known.

Objectives: To investigate the role of homocysteine (Hcy), bile acids, nitric oxide (NO) and folic acid on platelet function of rats with experimental cirrhosis.

Methodology: We have used platelets of rats with bile duct ligation (BDL), to measure aggregation, Ca2+-intracellular levels and its regulation ([Ca2+]i), reactive oxygen species-(ROS) and the expression of P-selectin.

Conclusions: LCB rats without ascites show greater platelet aggregation, mobilization of [Ca2+]i, ROS production and expression of P-selectin and less capacitative calcium entry (ECC). In platelets of LCB with ascites, many of these alterations are absent. The absence of NO and/or presence of bile acids reduce ECC in platelets of LCB animals. Folic acid can reverse the hyperaggregation, overexpression of P-selectin, reduce the release of Ca2+ stores and normalize ROS production.

Keywords: Bile duct ligation (BDL), homocysteine (Hcy), nitric oxide (NO), reactive oxygen species (ROS), capacitative Ca2+ entry (ECC), intracellular calcium ([Ca2+]i), deoxycholic acid (DC) and chenodeoxycholic (CDC).