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Ultra structural study of the pathogenesis of liver fibrosis in rats

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Liver fibrosis is a significant health problem developed as a response to a wound-healing process in injured liver characterized by excessive deposition of fibers and extracellular matrix. Despite extensive studies, the ultra-structural events are not well elucidated in liver fibrosis. The aim of this study was to clarify the ultra-structural events that govern the ECM deposition and fibrosis progression using dimethyl nitrosamine DMN induced liver fibrosis in rat's model. Two groups of male rats were assigned in this study: Control and DMN. Rats were administered DMN intra-peritoneally (10 mg/kg, 3 days/week for 21 days). Administration of DMN induced significant body weight loss and severe pathological alterations in hepatic cells. All hepatic cells suffered specific changes. The hepatocytes were injured and went through apoptosis. The sinusoidal endothelial cells lost their fenestrae. The Kupffer cells as well as the lymphocytes proliferated and contributed to the inflammation. In addition, the quiescent hepatic stellate cells (HSCs) activated; they lost their retinoid and acquired large nucleus, attained large amount of fibers around it. HSCs were transformed into myo-fibroblasts phenotype synthesized extracellular matrix (ECM) proteins and produced fibrous scar. Furthermore, portal fibroblast PFs proliferated and produced large amount of fibers in portal and periportal area. Lymphocytic infiltration, necrosis, hepatocyte steatosis, cholangiocyte proliferation all these contributed to liver fibrosis in this study. In conclusion, the most distinctive features of the cellular events of hepatic fibrosis in this study were focal deposition of ECM and collagens, primarily in portal and periportal areas as well as bizzare and extensive fibrous appearance of mitochondria in hepatocytes. This guides us to believe that activated portal fibroblasts contributed highly to fibrosis.

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