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论著

小鼠血吸虫病肝纤维化的超微结构动态观察

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摘要

目的 研究血吸虫病小鼠肝纤维化过程中几种相关细胞和肝组织超微结构动态变化 ,以探讨血吸虫病肝纤维化的可能机制。 方法 日本血吸虫尾蚴经皮肤感染小鼠建立血吸虫病肝纤维化模型。常规方法制作肝组织透射电镜标本并观察。常规 HE染色观察其病理变化。 结果 HE染色显示小鼠血吸虫病肝纤维化模型建立成功。电镜观察显示小鼠感染后 6 wk,急性肉芽肿周围的肝细胞发生坏死 ,肝窦内皮细胞窗孔减少 ,贮脂细胞 (FSC)脂滴减少 ,枯否细胞胞浆出现大吞噬体和粗面内质网。 8wk时部分肝细胞发生脂肪变性 ,少数肝细胞间隙增宽 ,间面出现微绒毛。肝窦周隙内充满大量胶原纤维 ,并形成肝窦毛细血管化。FSC胞浆出现含胶原原纤维的分泌泡 ,周围见大量胶原纤维。枯否细胞粗面内质网增加。 10 wk时FSC转变为肌成纤维细胞。 12 wk时肌成纤维细胞减少 ,成纤维细胞和纤维细胞增加。 结论 FSC被激活转化为肌成纤维细胞是血吸虫病肝纤维化发生的关键环节 ,激活的枯否细胞、损伤的肝细胞和肝窦内皮细胞与 FSC的活化密切相关 ,肝窦毛细血管化可能加速肝纤维化的发展。

关键词 血吸虫病 贮脂细胞 枯否细胞 肌成纤维细胞 纤维细胞 内皮细胞 肝纤维化 超微结构

分类号

Ultrastructural Dynamic Observation on Murine Schistosomal Hepatic Fibrosis

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Abstract

Objective To explore possible mechanisms of hepatic fibrosis by investigating the ultrastructural dynamic changes of liver tissue, especially several kinds of cells related to hepatic fibrosis.. Methods. Murine schistosomal hepatic fibrosis model was established by infecting mice with Schistosoma japonicum cercariae. Routine transmission electron microscopy was used to observe the liver tissue. H.E. staining was used for examining the pathological changes. . Results . H.E. staining showed that the model was established successfully. Ultrastructural observation showed that at the 6th week after infection, the necrosis of hepatocytes around the acute granulomas occurred; the number of sinusoidal endothelial fenestrae and vitamin A droplets in fat-storing cells decreased; large phagosomes and rough endoplasmic reticulum could be seen in the cytoplasm of Kupffer's cells. At the 8th week, steatosis was found in some hepatocytes, some microvilli emerged on a few inter-hepatocytic surfaces and the inter-hepatocytic spaces were enlarged. Large collagen fibrillar bundles filled in the perisinusoidal spaces, and capillarization of hepatic sinusoids was observed. Secretory vesicles filled with collagen fibrils appeared in the cytoplasm of fat-storing cells with large amount of collagenous fiber bundles surround the cells. Rough endoplasmic reticulum increased in Kupffer's cells. At the 10th week, fat-storing cells were activated and transformed into myofibroblasts. At the 12th week, the number of myofibroblasts decreased but that of fibroblasts and fiber cells increased. . Conclusion . Activation of fat-storing cells and transformation from fat-storing cells into myofibroblasts are the critical link in the development of hepatic fibrogenesis following schistosome infection. Kupffer's cells, necrotic hepatocytes and sinusoidal endothelial cells may relate to the activation of fat-storing cells. Capillarization of hepatic sinusoids possibly accelerates the development of hepatic fibrosis.

Key words schistosomiasis fat-storing cell Kupffer's cell myofibroblast fibroblast endothelial cell hepatic fibrosis ultrastructure.

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