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Alterations of Mast Cells and TGF-β1 on the Silymarin Treatment for CCl_4-Induced Hepatic Fibrosis

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Silymarin is a potent anti-oxidant, anti-inflammatory and anti-fibrogenic agent in the liver, which is mediated by alteration of hepatic Kupffer cell function, lipid peroxidation, and collagen production. Especially, in hepatic fibrogenesis, mast cells are expressed in chronic inflammatory conditions, and promote fibroblast growth and stimulate production of the extracellular matrix by hepatic stellate cells. We examined the inhibitory mechanism of silymarin on CCl_4-induced hepatic cirrhosis in rats. At 4, 8, and 12 weeks, liver tissues were examined histopathologically for fibrotic changes and observed fibrotic process by silymarin treatment. In the silymarin with CCl_4-treated group, increase of hepatic stellate cells and TGF-β1 production were lower than that of the CCl_4-treated group at early stages. Additionally, at the late fibrogenic stage, expressions of TGF-β1 were weaker and especially not expressed in hepatocytes located in peripheral areas. Moreover, the number of mast cell in portal areas gradually increased and was dependent on the fibrogenic stage, but those of CCl_4 with silymarin-treated group were decreased significantly. In conclusion, anti-fibrotic and anti-inflammatory effects of silymarin were associated with activation of hepatic stellate cells through the expression of TGF-β1 and stabilization of mast cells. These results suggest that silymarin prevent hepatic fibrosis through suppression of inflammation and hypoxia in the hepatic fibrogenesis.

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