عنوان مقاله:

Deletion of protein kinase C θ attenuates hepatic ischemia/reperfusion injury and further elucidates its mechanism in pathophysiology

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نویسندگان:

Wei Li - Department of Pathophysiology, Wannan Medical College, Wuhu, China

Meng-Yuan Shen - School of Medical Imaging, Wannan Medical College, Wuhu, China

Ruo-Bing Liu - School of Clinical Medicine, Wannan Medical College, Wuhu, China

Jun-Yang Zhang - School of Medical Imaging, Wannan Medical College, Wuhu, China

Rong-Yu Li - Department of Immunology, Wannan Medical College, Wuhu, China

Guo-Guang Wang - Department of Pathophysiology, Wannan Medical College, Wuhu, China

خلاصه مقاله:

Objective(s): Hepatic ischemia-reperfusion (HIR) is a severe process in pathophysiology that occurs clinically in hepatectomy, and hepatic transplantations. The present study aimed to investigate the effect of PKC θ deletion against HIR injury and elucidate its mechanism in pathophysiology.Materials and Methods: HIR injury was induced in wild-type and PKC θ deletion mice treated with or without heme. The ALT and AST levels were determined to evaluate liver function. HIR injury was observed via histological examination. Oxidative stress and inflammatory response markers, and their signaling pathways were detected.Results: The study found that PKC θ knockout decreased serum AST and ALT levels when compared to the WT mice. Furthermore, heme treatment significantly reduced the ALT and AST levels of the PKC θ deletion mice compared with the untreated PKC θ deletion mice. PKC θ deletion markedly elevated superoxide dismutase activity in the liver tissue, reduced malondialdehyde content in the tissue, and the serum TNF-α and IL-۶ levels compared with the WT mice. Heme treatment was observed to elevate the activity of SOD and reduced MDA content and serum of TNF-α and IL ۶ in the PKC θ deletion animals. Meanwhile, heme treatment increased HO-۱ and Nrf ۲ protein expression, and reduced the levels of TLR۴, phosphorylated NF-κB, and IKB-α.Conclusion: These findings suggested that PKC θ deletion ameliorates HIR, and heme treatment further improves HIR, which is related to regulation of PKC θ deletion on Nrf ۲/HO-۱ and TLR۴/NF-κB/IKB α pathway.

کلمات کلیدی:

Gene knockout, Hepatic ischemia/reperfusion injury, Nrf۲/HO-۱ pathway Pathophysiology, Protein kinase C θ, TLR۴/NF-κB/IKB α pathway