عنوان مقاله:

Sirtuin ۳ deficiency promotes acute kidney injury induced by sepsis via mitochondrial dysfunction and apoptosis

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نویسندگان:

Heng Fan - *Department of Intensive Care Unit, Ningbo First Hospital, Ningbo, Zhejiang Province, P.R China*

Jian-wei Le - *Department of Intensive Care Unit, Ningbo First Hospital, Ningbo, Zhejiang Province, P.R China*

Min Sun - *Department of Intensive Care Unit, Ningbo First Hospital, Ningbo, Zhejiang Province, P.R China*

Jian-hua Zhu - *Department of Intensive Care Unit, Ningbo First Hospital, Ningbo, Zhejiang Province, P.R China*

خلاصه مقاله:

Objective(s): To explore the regulation mechanism of Sirtuin ۳ (SIRT۳) on the mitochondrial function and apoptosis of acute kidney injury (AKI) in septic mice.Materials and Methods: The sepsis-induced AKI model was constructed in the wild-type and SIRT۳ knockout (KO) mice, and the levels of serum creatinine (Scr) and plasma kidney injury molecule ۱ (pKIM-۱) in mice were detected by ELISA. The mitochondrial damage of kidney tubular epithelial cells (KTEC) was observed by electron microscopy, the apoptosis of KTEC was detected by TUNEL assay, and the mRNA levels of SIRT۳, Bax, Caspase-۳, and Bcl-۲ were detected by RT-qPCR.Results: SIRT۳ KO caused increased expression of Scr, pKIM-۱, and inducible nitric oxide synthase protein in the kidneys of septic mice, and decreased the levels of superoxide dismutase, catalase, and mitochondrial complex enzymes I/II/III/IV. SIRT۳ deficiency exacerbated histopathological and mitochondrial damage to the proximal tubules of the kidney. In addition, SIRT۳ KO resulted in a significantly increased apoptosis of KTEC, increased the mRNA levels of Bax and Caspase-۳, and decreased the mRNA levels of Bcl-۲. Conclusion: Our study suggests that SIRT۳ deficiency promotes sepsis-induced AKI via increasing oxidative stress, mitochondrial dysfunction, and inducing apoptosis.

کلمات کلیدی:

Acute kidney injury, Apoptosis, Mitochondria, Sepsis, Sirtuin۳