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Melatonin Improving Myocardial Injury in Septic Rats and Relative Mechanisms

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SUMMARY. **The aim was to investigate melatonin improving myocardial injury in septic rats and relative mechanisms. Cannulation of right common carotid artery was performed 48 h after operation to detect the cardiac function of the rats in each group. HE staining was used to determine the pathological changes of myocardium. And positive apoptosis cell number were evaluated by TUNEL staining in myocardium. The serum contents of CK-MB and cTn-I, and the plasma levels of TNF-α and IL-6 of the rats in each group were measured. The protein expression of TLR4, TAK1, and NF-κB p65 in rat myocardial tissue was determined by WB. Indicators of cardiac function were significantly reduced. In morphology, the myocardial vasodilation and hyperemia, focal cell degeneration and necrosis, and inflammatory cell infiltration were observed. Biochemical indicators showed that the serum contents of CK-MB and cardiac troponin (cTn-I), and the plasma levels of tumornecrosis factor- α(TNF- α) and interleukin-6 (IL-6) were significantly increased ($p < 0.001$), apoptosis cell number significantly up-regulation and the protein expression of myocardial TLR4, TAK1, and NF-κB p65 was increased significantly ($p < 0.001$). Myocardial structure and function were improved in CLP+LD, CLP+MD and CLP+HD groups. CK-MB and cTn-I, apoptosis cell number significantly decreased and TNF-α and IL-6 were significantly reduced, and myocardial TLR4, TAK1, and NF-κB p65 was significantly reduced ($p < 0.05$, respectively). Melatonin significantly attenuates myocardial injury in the septic rats. By regulating the NF-κB inflammatory signaling pathway.**

RESUMEN. El objetivo era investigar la mejora de la melatonina en la lesión miocárdica en ratas sépticas y sus mecanismos relativos. La canulación de la arteria carótida común derecha se realizó 48 h después de la operación para detectar la función cardíaca de las ratas de cada grupo. Se utilizó tinción HE para determinar los cambios patológicos del miocardio. Y el número de células de apoptosis positivas se evaluó mediante tinción con TUNEL en miocardio. Se midieron los contenidos séricos de CK-MB y cTn-I, y los niveles plasmáticos de TNF-α e IL-6 de las ratas de cada grupo. WB determinó la expresión de proteínas de TLR4, TAK1 y NF-ϰB p65 en tejido de miocardio de rata. Los indicadores de función cardíaca se redujeron significativamente. En la morfología, se observaron vasodilatación e hiperemia del miocardio, degeneración y necrosis de células focales e infiltración de células inflamatorias. Los indicadores bioquímicos mostraron que los contenidos séricos de CK-MB y troponina cardíaca (cTn-I), y los niveles plasmáticos de factor de necrosis tumoral-α (TNF-α) e interleucina-6 (IL-6) aumentaron significativamente ($p < 0.001$), el número de células de apoptosis aumentó significativamente y la expresión de proteínas de TLR4, TAK1 y NF-ϰB p65 del miocardio aumentó significativamente ($p < 0,001$). La estructura y función del miocardio mejoraron en los grupos CLP+LD, CLP+MD y CLP+HD. CK-MB y cTn-I, el número de células de apoptosis disminuyó significativamente y TNF-α e IL-6 se redujeron significativamente, y TLR4 miocárdico, TAK1 y NF-ϰB p65 se redujeron significativamente ($p < 0,05$, respectivamente). La melatonina atenúa significativamente la lesión miocárdica en ratas sépticas. Regulando la vía de señalización inflamatoria NF-ϰB.

KEY WORDS: cecal ligation and puncture, melatonin, sepsis, myocardial injury, NF-ϰB signaling pathway

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