Influence of minocycline preconditioning on myocardial ischemia and reperfusion injury and HMGB1 expression in rats

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[Foundation Project]: This Work is Financially Supported by Special Foundation for Clinical Studies from Committee of Medical Journal of Chinese Universities (112210174)

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Received: 2011-12-13 Revised: 2012-04-10 JHMC, 2012; 18(6): 740-743

View from specialist: It is creative, and of certain scientific and educational value.

[ABSTRACT] Objective: To investigate the protection role of minocycline and the mechanism by which minocycline protects rats against myocardial ischemia and reperfusion (I/R) injury in rats. Methods: Anesthetized male rats were treated with minocycline (45 mg/kg, i.p.) 1 h before ischemia, and then subjected to ischemia for 30 min followed by reperfusion for 4 h. The lactate dehydrogenase (LDH), creatine kinase (CK), malondialdehyde (MDA) and superoxide dismutase (SOD) were measured. Infarct size was measured by TTC and the myocardial tissue apoptosis was assessed by TUNNEL assay. HMGB1 expression was assessed by Western blot. Results: Comparing with the control group, minocycline could significantly decrease the infarct size, myocardium apoptosis and the levels of LDH and CK (all P<0.05). Minocycline could also significantly inhibit the increase in the MDA level and the decrease in the SOD level (both P<0.05). Meanwhile, minocycline could also significantly inhibit the HMGB1 expression during myocardial I/R. Conclusions: Minocycline preconditioning could reduce myocardial ischemia and reperfusion injury and myocardial tissue apoptosis by inhibiting HMGB1 expression.

[KEY WORDS] Minocycline; High mobility group box 1 protein; Apoptosis; Myocardial ischemia; Reperfusion