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INOS/NO ACTS AS TRIGGERS AND EFFECTORS IN THE MECHANISMS OF LTA PROTECTION AGAINST MYOCARDIAL ISCHAEMIA/REPERFUSION INJURY IN SPONTANEOUS HYPERTENSIVE RAT

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Objectives To explore the effects of lipoteichoic acid (LTA) induced delayed preconditioning on myocardial ischaemia/reperfusion injury (I/R) in spontaneous hypertensive rat (SHR) and whether iNOS/NO were participated the mechanisms.

Methods I/R model was induced by left anterior descending coronary artery ligation for 30 min, followed by 60 min reperfusion. Myocardial apoptosis on the left ventricle at the end of reperfusion were detected by TUNEL staining in situ, and the changes of Bcl-2 and Bax protein, were detected by western blot. Meanwhile, the expression of iNOS mRNA and protein of left ventricle were detected.

Results Pretreated with LTA (1 mg/kg) 24 h before the experiment could obviously decrease ventricular arrhythmia and reduce CK-MB and LDH release in serum during ischaemia and reperfusion. LTA preconditioning significantly decreased myocardial apoptosis index and the expression of Bax protein was obviously decreased but markedly increased the expression of Bcl-2 protein. The mean relativity expression of iNOS mRNA in LTA preconditioning group was increased 0.71 times and the expression of iNOS protein was increased 0.96 times compared with that of in I/R group. There are no desperations no matter pretreatment of the rats with the inhibitor of iNOS, aminoguanidine (AG) before LTA preconditioning, or 30 min before ischaemia or pretreatment with AG alone.

Conclusions LTA induced delayed preconditioning could obviously decreases myocardial necrosis and apoptosis induced by I/R in SHR. iNOS/NO acts as triggers and subsequently as effectors play a key role in the mechanisms of LTA protection.