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TETRANDRINE CONTROL PRO-INFLAMMATORY FACTOR TO REDUCE RAT MYOCARDIAL ISCHAEMIC/ REPERFUSION INJURY

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Objectives To investigate how tetrandrine through regulate the pro-inflammation factors TNF- α , IL-1 β , IL-6 to attenuate rat ischaemic/reperfusion injury.

Methods 80 male Sprague-Dawley (SD) rats were randomly divided into 4 group: Sham group, ischaemia/reperfusion (I/R) group, Tetrandrine group (Tet) and simvastatin group (Sim). The SD rat underwent 30 min of left anterior descending (LAD) coronary occlusion and 24 h reperfusion to make ischaemia/reperfusion (I/R) injury model in vivo. Sham group were not subjected to occlusion of artery. Tet group were injected tetrandrine to abdominal cavity 20 min before ischaemia starting. The rat in Sim group was administered simvastatin 2 mg/kg/l intragastrically every day, administering drugs lasted 14 days. The other procedures were same to the I/R group. Samples were collected after 24 h reperfusion. The expression level of TNF- α , IL-1 β , IL-6 protein in serum and myocardial tissue was detected by ELISA. LDH and CK were detected too. The neutrophil infiltration degree in myocardium was determined by using measuring the activity of myeloperoxidase (MPO) method. Cardiac function which includes FS%, EF and E/A was measured by using ultrasound. EB/TTC (Azovan Blue/2,3,5-Triphenyl-2H-Tetrazolium Chloride) dyeing method was used to measure the infarction size.

Results

1. The effect of Tet on the release of myocardial enzyme during ischaemia/reperfusion: the LDH and CK were significantly higher in I/R, Tet and Sim groups compared with Sham group ($p < 0.01$), but it were much lower in Tet and Sim groups compared with I/R group.
2. The effect of Tet on the cardiac function and infarction size: the cardiac function of systolic and dilator in experimental group was decreased significantly compared with normal heart's function. In Tet and Sim group, which was experienced pharmacological preconditioning their cardiac function were significant higher than I/R group ($p < 0.01$), but no significant difference between Tet and Sim on EF and E/A.
3. The effect of Tet on the neutrophil infiltration: the activity of MPO was significantly increased after reperfusion, its activity in experimental groups were much higher than Sham group ($p < 0.01$), notwithstanding its activity in Tet nad Sim groups were significantly lower than I/R group ($p < 0.01$). No significant difference was found between Tet and Sim group.
4. The effect of Tet on the release of proinflammatory factors (TNF- α , IL-1 β , IL-6): The similar results were obtained after measuring the serum and tissue concentration of TNF- α , IL-1 β and IL-6, that was in Tet and Sim group the expression of proinflammatory factors (TNF- α , IL-1 β , IL-6) were significant lower compared with I/R group ($p < 0.01$) and significant higher than shame group ($p < 0.01$).

Conclusions Tet can attenuate myocardial ischaemia/reperfusion injury. It achieves this pharmacologic action through reduce the harmful cytokine TNF- α and IL-6, IL-1 β .