Effect of morphine preconditioning in the delayed phase on the expression of p38 mitogen-activated protein kinase in a rabbit model of myocardial ischemia-reperfusion injury

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ABSTRACT. This study aimed to investigate the protective effects of delayed morphine preconditioning on myocardial ischemia-reperfusion injury. We randomly divided 30 rabbits into three groups with 10 rabbits in each group as follows: sham operation group (C group), ischemia-reperfusion group (I/R group), and morphine pretreatment group (M group). Rabbits in C Group received left coronary without blocking for 160 min. The left descending artery of rabbits in the I/R group was blocked for 40 min and reperfused for 120 min. Rabbits in the M group received intravenous administration of 1.0 mg/kg morphine; after 24 h, rabbits in this group received the same treatment as that administered to the I/R group. We determined tumor necrosis factor alpha (TNF-α) levels in blood samples from the internal carotid artery of rabbits in
each group 20 min before occlusion of the left descending coronary artery, 20 and 40 min after occlusion of the left descending coronary artery, and 1 and 2 h after myocardial reperfusion. After 120 min of reperfusion, immunoblotting was used to measure the activity levels of myocardial p38 mitogen-activated protein kinase (MAPK); in addition, the infarct size was measured. Compared to the I/R group, the M group showed a significant decrease in TNF-α levels, p38 MAPK activity, and the myocardial infarct size (I/R group 37.8% ± 1.7% vs 21.5% ± 2.4%; P < 0.05). Thus, morphine preconditioning in the delayed phase may exert protective effects on myocardial I/R injury by inhibiting myocardial p38 MAPK activity and decreasing TNF-α production.

Key words: Morphine; Myocardial ischemia-reperfusion injury; Delayed preconditioning; p38 Mitogen-activated protein kinase