Effects of isoflurane preconditioning in the delayed phase on myocardial tumor necrosis factor alpha levels and caspase-3 protein expression in a rabbit model of ischemia-reperfusion injury

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Received June 3, 2014
Accepted February 20, 2015
Published July 3, 2015
DOI http://dx.doi.org/10.4238/2015.July.3.1

ABSTRACT. This study aimed to investigate the protective effects and the mechanisms underlying these effects of isoflurane preconditioning in the delayed phase of myocardial ischemia-reperfusion injury. We randomly divided 30 healthy male New Zealand white rabbits into three groups with 10 rabbits in each group as follows: sham operation group (C group), ischemia-reperfusion group (I/R group), and 2.0% isoflurane preconditioning group (S group). Rabbits in the C group received thoracotomy for 160 min. Rabbits in the I/R group underwent left coronary artery occlusion for 40 min and reperfusion for 120 min. Rabbits in the S group received inhalation of 2.0% isoflurane and 100% oxygen for 2 h; after 24 h, rabbits in this group
received the same treatment as that administered to rabbits in the I/R group. We examined the tumor necrosis factor alpha (TNF-α) levels in each group 20 min before occlusion of the left coronary, 20 and 40 min after occlusion of the left coronary artery, and 1 and 2 h after myocardial reperfusion. After reperfusion, immunoblotting was used to measure the myocardial caspase-3 expression levels, and the infarct size was measured using Evans blue and tetrazolium chloride staining. The levels of TNF-α and caspase-3 were lower in the S group than in the I/R group, and the myocardial infarct size decreased in the S group. Thus, isoflurane preconditioning in the delayed phase exerted protective effects by decreasing the myocardial caspase-3 expression and TNF-α production in a rabbit model of ischemia-reperfusion injury.

Key words: Isoflurane; Preconditioning in delayed phase; Myocardial ischemia reperfusion; Caspase-3; Tumor necrosis factor-α