J Clin Trials 2017, 7:5 (Suppl)
DOI: 10.4172/2167-0870-C1-020

Possible role of JAK-2 in attenuated cardio-protective effect of ischemic preconditioning/post conditioning in hyperlipidemia rat heart

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The present study has been designed to investigate the possible role JAK-2 in the attenuated cardio protective effect of ischemic preconditioning (IPC) and ischemic post conditioning (IPOC) in hyperlipidemic rat heart. Rats were administered high fat diet freely for 28 days to produce experimental hyperlipidemia. Isolated Langendorff's perfused normal and hyperlipidemic rat heart were subjected to global ischemia for 30 min followed by reperfusion for 120 min. Rat heart infarct size was assessed macroscopically using triphenyltetrazolium chloride (TTC) staining. CK-MB and LDH release was analyzed from coronary effluent to assess the degree of cardiac injury. Moreover, the oxidative stress in heart was assessed by measuring TBARS, GSH and SOD. The ischemia-reperfusion (I/R) injury has been observed to induce oxidative stress by increasing TBARS and decreasing reduced form of glutathione and SOD in normal and hyperlipidemic rat heart. Moreover, I/R induced myocardial injury, which assessed in terms of increase in myocardial infarct size, CK-MB and LDH release in coronary effluent and decrease in coronary flow rate in normal and hyperlipidemic rat heart. In addition, the hyperlipidemic rat hearts showed enhanced I/R-induced myocardial injury with high degree of oxidative stress as compared with normal rat heart subjected to I/R. Four episodes of IPC (5 min each) and six cycle of IPOC (10 sec each) afforded cardio protection against I/R-induced myocardial injury in normal rat heart which was assessed in terms of improvement in coronary flow rate and decrease in CK-MB, LDH, oxidative stress and myocardial infarct size. On the other side, IPC and IPOC mediated myocardial protection against I/R-injury was abolished in hyperlipidemic rat heart. Treatment via Langendorff's perfusion apparatus with Tyrphostin AG490 (5 μM), a selective inhibitor of JAK-2 markedly restored the cardioprotective effects of IPC and IPOC in hyperlipidemic rat heart. At last, it is suggested that the high degree of oxidative stress produced in hyperlipidemic rat heart during reperfusion and consequent activation of JAK-2 may be responsible to attenuate the cardioprotective effects of IPC and IPOC against I/R induced myocardial injury.

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