

Possible Neuroprotective Mechanism of Remote Limb Ischemic Post Conditioning against Global Cerebral Ischemic Injury

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Abstract : Background and purpose: Recent investigations on ischemia and reperfusion injury postulate that transient ischemia of remote organs after a prolonged ischemic insult confers neuroprotection. However, the molecular mechanisms of the remote limb ischemic post-conditioning (RIPOC) are yet to be elucidated. The current study was designed to investigate the protective mechanism of RIPOC against cerebral ischemic injury using global model of stroke. Materials and methods: Global ischemic reperfusion injury (IR) was achieved by 30 minutes ischemia of cerebral artery, followed by reperfusion for 24 hours. Induction of global ischemia was followed by 4 brief episodes (30 seconds each) of ischemia and reperfusion of femoral artery to accomplish RIPOC. 5-Hydroxy Decanoic acid (5-HD), a KATP channel blocker (20 mg/kg) was administered after induction of global ischemia and RIPOC intervention. Results: IR injury ensue significant behavioural deficits as manifested by rotarod performance and spontaneous locomotor activity when compared to sham control. Furthermore, IR injury significantly increased oxidonitrative stress and infarct volume as evidenced by biochemical parameters (MDA, GSH, Nitrite, SOD) and 2,3,5-triphenyltetrazolium chloride (TTC) staining respectively. Moreover, RIPOC intervention ameliorated the behavioural performance, attenuated the oxidative stress and infarct volume when compared to IR injury group. However, administration of 5-HD increased the oxidative stress and infarct size while deteriorating the behavioural parameters when compared to RIPOC group. Conclusions: In a nutshell, cerebral IR injury has significantly induced the neuronal damage, whereas RIPOC intervention decreased the neuronal injury. Moreover, 5-HD abolished the neuroprotection offered by RIPOC indicating the putative role of KATP channel opening in RIPOC against cerebral ischemic injury.

Keywords : RIPOC, cerebral injury, KATP channel, neuroprotection

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