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Protocatechuic Acid Induced Pharmacological Postconditioning Ameliorates Cerebral Ischemia Reperfusion Injury in MICE By Modulation of Nuclear Factor Erythroid 2-Related Factor Pathway

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Introduction

*Ischemia/Reperfusion Injury is the tissue damage caused when blood supply returns to tissue after a period of ischemia or lack of oxygen.

*Pharmacological postconditioning is a strategy that either involves hindering the deleterious pathway or inducing a modest stress level which triggers the intracellular defense pathway to sustain more vigorous insult leading to conditioning.

*At present, there is no proven neuroprotective drug against the cerebral ischemia-reperfusion injury

*Protocatechuic acid, a naturally occurring phenolic acid, reported to possess various pharmacological effects: antioxidant, anti-inflammatory, anti-apoptotic properties, and antimicrobial properties. PCA protected ischemic-reperfusion rats by reducing the MDA formation and restored the depleted antioxidants

Material & Methods

Induction of global cerebral ischemia

Cerebral global ischemia of 17 min was induced, and then bilateral carotid artery was occluded

Experimental design:

Mice were randomly divided into 7 groups: each group comprised of 8 animals

Group I: (Sham group) ; Group II: (I/R Group) ; Group III: (Trigonelline per se) ; Group IV: I/R+ Protocatechuic acid (50 mg/kg) ; Group V: I/R+ Protocatechuic acid (100 mg/kg) ; Group VI: Trigonelline (10 mg/kg) +I/R+Protocatechuic acid (100 mg/kg) ; Group VII: Trigonelline (20 mg/kg)+I/R+Protocatechuic acid (100 mg/kg)

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Discussion

*We employed elevated plus-maze test & Morris water maze test to assess memory and rota-rod test for the evaluation of motor coordination and TBARS and Glutathione for oxidative stress.

*In our study, global cerebral ischemia/ reperfusion (I/R) produced a significant rise in infarct size and induced impairment of memory as well as of motor coordination.

*pPoCo was observed to prevent ischemia and reperfusion-induced cerebral infarct size and impairment of memory and motor incoordination, which is in concordance with earlier findings.

Conclusion

*It is concluded that PCA mediated pharmacological postconditioning exerts neuroprotective effect, possibly mediated through the activation of Nuclear Factor Erythroid 2-Related Factor signaling pathway

*Nevertheless, further studies are needed to affirm this biochemical transduction system that might be ultimately leading to this Nuclear Factor Erythroid 2-Related Factor signaling pathway based protection served by pPoCo of the brain.