Does oxytocin have a neuro-protective impact in rats’ stroke model?

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Abstract:

Background: Stroke is a causative factor of disabilities and death. Various mechanisms involved in the cerebral ischemia-reperfusion pathophysiology, including oxidative stress along with inflammation. Aim: This research assessed the impact of oxytocin in lessening the detrimental effects of reperfusion in the cerebral ischemia/reperfusion (I/R) injury with the causal mechanisms. Materials: The cerebral ischemia-reperfusion injury was elicited by bilateral common carotid artery obstruction for 30 min followed by reperfusion for 24 h in rats. Forty eight rats were divided into: sham-operated group, oxytocin control group (underwent sham operation and given intraperitoneal oxytocin at a dose 750 µg/kg body weight), ischemia and reperfusion group and oxytocin-treated-ischemia and reperfusion group underwent I/R injury and given oxytocin 15 min before perfusion. Total antioxidant capacity, total peroxide, oxidative stress index, tumor necrosis factor-alpha and sodium/potassium-ATPase (Na+/K+-ATPase) level were measured in the cerebral homogenate. Histopathological analyses using H&E stain were carried out. Results: Administration of oxytocin lowered the ischemia-reperfusion-induced elevations in the cerebral total peroxide, oxidative stress index and tumor necrosis factor-alpha concentrations and increased total antioxidant capacity concentration and Na+/K+-ATPase level. Together, these changes were associated with alleviated histopathological alteration-induced by ischemia-reperfusion injury. Conclusion: Oxytocin has a neuro-protective impact against the deleterious effects of reperfusion via amelioration of oxidative stress, and inflammation and restoration of the declining level of the Na+/K+-ATPase. Thus, OT probably has a therapeutic impact on ischemic stroke.

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