EFFECT OF MANNITOL AND URIC ACID ON THE OXIDATIVE STRESS BIOMARKERS IN ALBINO RATS INDUCED WITH TRAUMATIC BRAIN INJURY

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Abstract

Introduction and Objective
Traumatic brain injury (TBI) causes massive production of reactive oxygen species (ROS) with resultant oxidative stress (OS). Oxidative stress is caused by excessive generation of reactive oxygen species with consequent depletion of endogenous antioxidants. This study investigated the effect of mannitol and uric acid in the management of induced TBI in albino rats.

Materials and Methods
TBI was induced by closed head injury in albino rats of winstar strain using accelerated impact device by weight drop method. The rats were orally administered 22.5mg/kg, 45mg/kg and 60mg/kg of mannitol and uric acid in different groups for 14 days after TBI induction. Blood and brain tissues were collected and analyzed for oxidative stress biomarkers and histopathological changes.

Results and Conclusion
The results showed that TBI significantly increased (P<0.05) malondialdehyde level and creatine kinase activity. Supplementation with uric acid and mannitol, however reversed the trend. Histological changes indicated interparenchymal hemorrhages in brain tissues of traumatized non treated rats and normal brain tissue in uric acid and mannitol supplemented rats groups. Supplementation might have replenish the antioxidant defense system and have therefore reduced the impact of oxidative stress that is associated with increased mortality in TBI. This study highlighted the potentials for the use of antioxidants in the management of TBI.

Keywords: Traumatic brain injury, Oxidative Stress, Antioxidants