

Melatonin Suppresses the Brain Injury after Cerebral Ischemia/Reperfusion in Hyperglycemic Rats

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Abstract : Diabetes mellitus (DM) is known to exacerbate cerebral ischemic injury. The present study aimed to investigate the anti-oxidant and anti-inflammatory effects of oral supplementation of melatonin (MLN) on cerebral injury caused by middle cerebral artery occlusion and reperfusion (MCAO/Re) in streptozotocin (STZ)-induced hyperglycemic rats. Hyperglycemia was induced by a single injection of STZ (55mg/kg; i.p.), six weeks later the cerebral injury was induced by MCAO/Re. Twenty-four hours after the MCAO/Re the MLN (10 mg/kg) was injected for 14 consecutive days. Results of the present study revealed that MCAO/Re in STZ-induced hyperglycemia in rats causes an increase in the oxidative stress biomarkers; it increased brain lipid peroxidation (measured as malondialdehyde; MDA) and brain level of nitric oxide (NO). Moreover, MCAO/Re produces a prominent increase in the brain inflammatory markers viz. interleukin-6 (IL-6), interleukin-1 β (IL-1 β) and tumor necrosis nuclear factor-alpha (TNF- α). Oral treatment of MCAO/Re in STZ-induced hyperglycemic rats with MLN (10 mg/kg) for two weeks restored the brain levels of MDA, GSH, NO, IL-6, IL-1 β and the TNF- α . MLN succeeded to suppress the exacerbation of damage in the brain of hyperglycemic rats. These results suggest that daily intake of MLN attenuates the exacerbation of cerebral ischemic injury in a diabetic state, which may be attributed to anti-oxidant and anti-inflammatory effects in the brain.

Keywords : melatonin, brain injury, cerebral ischemia/reperfusion, hyperglycemia, rats

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