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Neuroprotective effect of minocycline on cognitive impairments induced by transient cerebral ischemia/reperfusion through its anti-inflammatory and anti-oxidant properties in male rat

Running title : Neuroprotective effect of minocycline after cerebral ischemia

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Highlights

- Inflammation plays an important role in post-ischemic neuronal death.
- Minocycline reduced death of hippocampal CA1 pyramidal cells following ischemia.
- Minocycline has anti-inflammatory and antioxidant properties.
- Minocycline improves learning and memory deficits induced by ischemia/reperfusion

Abstract

Memory deficit is the most visible symptom of cerebral ischemia that is associated with loss of pyramidal cells in CA1 region of the hippocampus. Oxidative stress and inflammation may be involved in the pathogenesis of ischemia/reperfusion (I/R) damage. Minocycline, a semi-synthetic tetracycline derived antibiotic, has anti-inflammatory and antioxidant properties. We evaluated the neuroprotective effect of minocycline on memory deficit induced by cerebral I/R in rat. I/R was induced by occlusion of common carotid arteries for 20 min. Minocycline (40mg/kg, i.p.) was administered once daily for 7 days after I/R. Learning and memory were assessed using the Morris water maze test. Nissl staining was used to evaluate the viability of CA1 pyramidal cells. The effects of minocycline on the microglial activation was also investigated by Iba1 (Ionized calcium binding adapter molecule 1) immunostaining. The content of malondialdehyde (MDA) and pro-inflammatory cytokines (IL-1β and TNF-α) in the hippocampus were measured by thiobarbituric acid reaction substances method and