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Aminoguanidine reduces infarct volume and improves neurological dysfunction in transient model of focal cerebral ischemia in rat

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Abstract:

Focal cerebral ischemia (Stroke) is the cessation or severe reduction of blood flow to an area of the brain that through activation of a complex cytotoxic cascade results in neuronal cell death. The present study was designed to examine the effects of post-ischemic treatment with aminoguanidine (AG) on cortical, striatal infarct volume as well as neurological dysfunctions. Rats (n=23) were allocated to sham, saline or AG (300 mg/kg)-treated groups. Ischemia was induced by 90 minutes middle cerebral artery occlusion, followed by 24 hrs reperfusion. Saline or AG was administered intraperitoneal at one hour after induction of ischemia. At the end of 24hrs reperfusion, neurological deficit score was tested and cortical, striatal infarct volumes were determined by Triphenyltetrazolium chloride staining. Administration of AG (300 mg/kg) at one hours after ischemia resulted in a significantly lower cortical (85±25 vs. 210±13 mm3), striatal (35±5 vs. 58±10 mm3) infarct volumes, and neurological deficit score (1.88±0.23 vs. 2.67±0.21). Our findings indicate that aminoguanidine is a potent neuroprotective in rat model of transient focal cerebral ischemia. The future studies are required to clear cerebroprotective mechanism of aminoguanidine and possible use of this agent as a therapeutic target in stroke patients.

Keywords:

Aminoguanidine ، Focal cerebral ischemia

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