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Inhibition of nitric oxide synthase activity improves focal cerebral damage induced by cerebral ischemia/reperfusion in normotensive rats

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Abstract

Introduction: Nitric oxide seems to play a dual role in ischemia/reperfusion injury. Few studies have investigated whether it exacerbates or improves brain edema. In the present study, we inhibited the activity of nitric oxide synthase by L-NAME and evaluated the cerebral infarct volume, tissue swelling and brain edema, alongside the measurement of blood flow of the ischemic region.

Methods: Transient focal cerebral ischemia was induced by 60 min middle cerebral artery occlusion followed by 12 hours reperfusion in rat. Experiments were performed in three groups of rats (n=12 each); Sham, control ischemic, and L-NAME pretreated (1 mg/kg IP). Laser Doppler flowmetry was used to measure the regional blood flow. After neurological deficit score (NDS) testing, the brains were prepared for TTC staining or brain water content technique to measure the infarct volume and brain edema.

Results: Pretreatment with L-NAME significantly reduced NDS (3.66 ± 0.33 to 1.5 ± 0.34), infarct volume of cortex (374 ± 34 to 160 ± 41 mm3) and striatum (158 ± 15 to 87 ± 16 mm3), tissue swelling (7.35 ± 1.27% to 4.05 ± 0.91%) and brain edema (3.5 ± 0.48% to 1.6 ± 0.6%) without significant alteration of blood flow of the ischemic region.

Conclusion: The findings of this study indicate that inhibition of nitric oxide synthase activity reduces infarct volume and brain edema of the ischemic region induced during 60 min middle cerebral artery occlusion. This effect is not accompanied with any alteration in the blood flow of the ischemic region.

Key words: Ischemia/reperfusion injury; Nitric oxide synthase; L-NAME; Regional blood flow; Neurological deficit; Brain edema

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