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Effect of Rosiglitazone, on infarct volume and neurological deficits in local brain ischaemia

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Background

Stroke is accompanied by a robust inflammatory response, glutamate-mediated excitotoxicity, release of reactive oxygen species and apoptosis. Thiazolidinediones, which target the nuclear receptor peroxisome proliferator-activated receptor (PPAR)-g, have been reported recently to exhibit potent anti-inflammatory and anti-oxidant actions and inhibit both neural excitotoxicity and apoptosis.

Materials and methods

The present study was conducted to determine whether rosiglitazone, a potent thiazolidinedione for PPAR-g, would show efficacy against the cerebral infarction and neurological dysfunctions induced by embolic middle cerebral artery (MCA) occlusion in the rat. Focal ischaemic injury was induced by embolizing a preformed clot into the MCA. Rosiglitazone was dissolved in dimethyl sulphoxide and injected i.p. 1 h before MCA occlusion at doses of 0.033, 0.1, 0.3 or 1 mg/kg. Forty-eight hours after MCA occlusion, brains were removed, sectioned and stained with a 2% solution of 2,3,5-triphenyltetrazolum chloride and analysed using a commercial image-processing software program.

Results

When rosiglitazone was administered 1 h before embolization, it significantly reduced infarct volume by 48.2, 68.4% and 70.3% at doses of 0.1, 0.3 and 1 mg/kg, respectively ($P < 0.001$). Rosiglitazone-treated rats also demonstrated improved neurological functions. However, there were no statistically significant differences between control and treated groups in terms of brain oedema at 48 h after ischaemic injury.

Conclusions

The findings of the present study may support the idea of a potential benefit of thiazolidinediones in the management of ischaemic stroke.

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