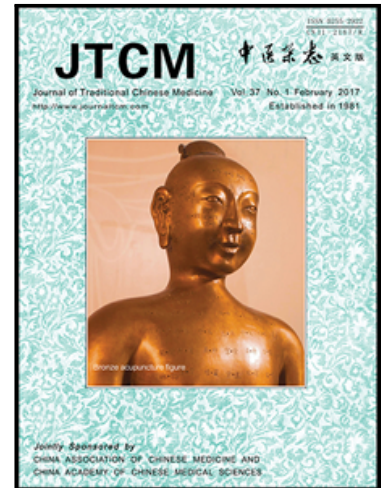


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Neuroprotective effect of Qinggan Lishui formula on retinal ganglion cell apoptosis in a microbead-induced rat chronic glaucoma model

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Abstract

OBJECTIVE: To investigate a possible mechanism for protective effects of a decoction of the Qinggan Lishui formula (QF) on retinal ganglion cells (RGCs) in a rat model of microbead-induced chronic intraocular hypertension (COH).

METHODS: The COH model was generated by injecting microbeads (superparamagnetic iron oxide) into the anterior chamber of rat eyes. QF was given by intragastric administration (gavage) once daily at a dose of 6.2 g/kg until day 28, following microbead injection. Cholera toxin B subunit (CTB) retrograde labeling and immunohistochemistry were used to evaluate changes in the number of RGCs in the retina. Terminal dUTP nick end labeling (TUNEL) staining was used to assess apoptotic changes in RGCs.

RESULTS: Microbead injection induced a steady increase in intraocular pressure (IOP) of rats. Elevated IOP resulted in a progressive reduction in the number of CTB-labeled RGCs, 2-4 weeks after microbead injection. QF administration may moderately reduce IOP in the rat COH model and attenuate reduction of the number of CTB-labeled RGCs in COH rats. Furthermore, elevated IOP resulted in a progressive increase in the number of TUNEL-positive RGCs, 2-4 weeks after microbead injection, suggestive of an increase in the extent of RGC apoptosis. There was a significant reduction in the number of TUNEL-positive signals in QF-treated COH retinas, compared with untreated COH retinas.

CONCLUSION: QF decoction may provide a protective effect for RGCs in COH retinas by reducing RGC loss; these effects may be mediated by inhibition of RGC apoptosis.

Keywords: Glaucoma; Retinal ganglion cells; Apoptosis; Qinggan Lishui formula

INTRODUCTION

Glaucoma is an optic nerve degenerative disease, which is predicted to affect > 80 million people worldwide by 2020, with approximately 10% being bilaterally blind.^{1,2} Decreasing intraocular pressure (IOP) has long been regarded as the only effective method for treatment of glaucoma.³ In addition, current available pharmacological treatments include cholinergic drugs, β -adrenergic agonists, β -adrenergic receptor blockers, α -adrenoreceptor agonists, carbonic anhydrase inhibitors, and

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