Drugs 2007; 67 (15): 2291
0012-6667/07/0015-2291/$44.95/0

Pharmacological Neuroprotection for Glaucoma

In their recently published interesting review on pharmacological neuroprotection in glaucoma, Chidlow et al.[1] discussed the neuroprotective effects of topically applied betaxolol. They addressed the ocular penetration issue of topical betaxolol, which is essential to the accumulation of this molecule in the target tissues of neuroprotection (retina and choroid). Unfortunately, regarding the penetration and ocular tissue concentrations of betaxolol, the authors cited only earlier data in rabbits, and did not use the recent and clinically relevant data determined on different ocular tissues of glaucoma patients and monkeys. In a study published by our group in 2006,[2] six phakic glaucoma patients and one aphakic glaucoma patient were treated with topical betaxolol 0.25% twice a day for at least 28 days before scheduled enucleation for painful blind eye. Immediately after enucleation, the ocular tissues were separated and the betaxolol concentrations were measured. Our data showed that long-term topical betaxolol medication at the clinically approved dosage results in high tissue concentrations in all anterior and posterior ocular layers, including the choroid and the retina. The concentrations measured by our group in these target tissues of neuroprotection in glaucoma were much higher than the threshold vasorelaxant concentration of betaxolol on human retinal arterioles *in vitro*.[3] Since vascular dysregulation in the posterior ocular layers is considered one of the probable reasons for retinal ganglion cell apoptosis in glaucoma, our published results are probably important in the context of the review article by Chidlow et al.[1]

Gábor Holló
Department of Ophthalmology, Semmelweis University, Budapest, Hungary

Acknowledgements

Dr Holló has acted as a consultant for Alcon, Allergan, Pfizer and Merck Sharp & Dohme.

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