

Advances in Glaucoma Treatment

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DESCRIPTION

Glaucoma is the slow and progressive degeneration of the Retinal Ganglion Cells (RGC) and the axons of the optic nerve. If not diagnosed and treated early, it can lead to irreversible blindness. Although elevated intraocular pressure is the main risk factor for glaucoma, other factors include elevated glutamate levels, changes in Nitric Oxide (NO) metabolism, changes in blood vessels, and oxidative damage caused by reactive oxygen species. Glaucoma is the second leading cause of blindness in the world, accounting for 12.3% of total blindness. Glaucoma has been broadly classified as primary or secondary open-angle or closed-angle glaucoma. The main goal of glaucoma management is to use medications, laser treatments, or conventional surgery to prevent risk factors, especially increased Intraocular Pressure (IOP). The first-line treatment of glaucoma usually starts with the use of selective or non-selective local blockers or prostaglandin analogs. The preferred second-line drugs include alpha-agonists and topical carbonic anhydrase inhibitors. Cholinergic agonists are considered a third-line treatment option. When monotherapy is not enough to reduce intraocular pressure, combination therapy is needed. In order to improve patient compliance, drug delivery systems such as electronic devices, ocular inserts, transdermal and mechanical drug delivery systems have been developed. The use of viscoelastic agents in ophthalmic preparations, emulsions, and Soluble Ophthalmic Drug Inserts (SODI) can improve patient compliance and ocular drug delivery in patients undergoing long-term glaucoma treatment. For patients who do not respond to glaucoma drugs, laser trabeculoplasty and incision surgery are recommended. Several nutrients and botanicals have shown promise in the treatment of glaucoma, but most studies are

preliminary and larger-scale controlled studies are needed. The future direction of the development of new glaucoma therapies may point to glutamate inhibition, NMDA receptor blockade, exogenous neurotrophic, open channel blockers, antioxidants, protease inhibitors, and gene therapy. Epinephrine is a direct-acting sympathomimetic amine. Adrenaline stimulates alpha- and beta-adrenergic receptors in the eye. It reduces intraocular pressure by increasing the aqueous output of the trabecular meshwork and uveoscleral pathway. However, recent studies have shown that topical epinephrine does not significantly affect uveoscleral outflow or extra scleral venous pressure. anxiety. Dipivephrine is a prodrug of epinephrine, formed by the desferification of epinephrine and pivalic acid. Adding two ester groups (pivaloyl) to the adrenaline molecule improves its lipophilicity and promotes its penetration into the anterior chamber. Since it penetrates the cornea more easily, a lower dose is required. Dipivuline is as effective as epinephrine in lowering intraocular pressure. The released adrenaline works by reducing water production and improving exit facilities. Dipivuline takes effect about 30 minutes after treatment and is effective in about 1 hour.

Dipivephrine is more effective than adrenaline; it penetrates the cornea approximately 17 times faster than adrenaline. It is suitable for initial treatment or as an adjunct to other intraocular pressure-lowering drugs. Reduces intraocular pressure by 20-24%. Topical 0.1% dipivuline can be used to reduce intraocular pressure in patients intolerant to epinephrine. The most common side effects of dipivephrine include burning pain, tingling, follicular conjunctivitis, blurred vision, headache, and allergic reactions

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