INTRODUCTION

Glaucoma is a neurodegenerative disease characterized by progressive loss of retinal ganglion cell axons and their cell bodies in the retina.

(IOP) was considered to be the major risk factor associated with the development of this neuropathy; but a randomized controlled clinical trials have demonstrated that in some patients the disease progresses, even with low IOP.

So, it is accepted now that IOP is not the major cause for glaucomatous visual loss in every patient. Although lowering of the IOP is now the only way to treat glaucoma (primarily by pharmacological means and to a lesser extent by surgery) however, the inadequacy of these methods are increasingly being recognized.

Therefore, the need to have a drug that preserves visual function irrespective of the cause of glaucomatous optic neuropathy is the aim of all researches now. This drug must interact with intra-retinal components to preserve, protect, and rescue the RGCs whatever the cause of their loss is; hence the name *NEUROPROTECTION* appeared which is defined as a therapeutic paradigm for slowing or preventing death of neurons, to maintain their physiological function.

Neuroprotection has been proposed as a therapeutic approach that aims to promote survival of retinal ganglion cells (RGCs), rather than addressing the often unknown underlying causes.

RGC survival has been demonstrated following a number of interventions including blockade of apoptosis pathways, prevention of glutamate-induced RGC excitotoxicity and administration of various neurotrophins.

The identification of the agents that mediate secondary injury and much more understanding of the pathways of apoptosis have raised the possibility of preventing the death of neurons that escaped the primary insult (by elevated IOP).So the hope is to rescue cells from apoptosis to continue functioning by neutralization or genetical alteration of the toxic products and secondary mediators of apoptosis after glaucoma related damage.