



Glaucoma Pathogenesis: Mechanisms of Optic Nerve Damage

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Citation: Peter N (2025) Glaucoma Pathogenesis: Mechanisms of Optic Nerve Damage. Int J Ophthalmic Pathol 13: 061

Received: 01-Jun-2025, Manuscript No. iopj-26-182519; Editor assigned: 4-Jun-2025, Pre-QC No. iopj-26-182519 (PQ); Reviewed: 19-Jun-2025, iopj-26-182519; Revised: 26-Jun-2025, Manuscript No. iopj-26-182519 (R); Published: 30-Jun-2025, DOI: 10.4172/2324-8599.1000061

Introduction

Glaucoma is a group of chronic, progressive optic neuropathies characterized by degeneration of retinal ganglion cells and corresponding visual field loss. It is one of the leading causes of irreversible blindness worldwide. Although elevated intraocular pressure is the most significant risk factor, glaucoma is a multifactorial disease with complex pathogenesis. Understanding the mechanisms involved in glaucomatous damage is essential for early diagnosis, effective treatment, and prevention of vision loss [1,2].

Discussion

The central pathological feature of glaucoma is damage to the optic nerve head, particularly at the level of the lamina cribrosa. Elevated intraocular pressure results from impaired aqueous humor outflow through the trabecular meshwork or increased resistance in the drainage pathways. This pressure elevation leads to mechanical stress and deformation of the lamina cribrosa, causing compression and disruption of retinal ganglion cell axons. As a result, axonal transport is impaired, leading to ganglion cell apoptosis [3,4].

In addition to mechanical factors, vascular mechanisms play a significant role in glaucoma pathogenesis. Reduced blood flow to the optic nerve head due to systemic hypotension, vasospasm, or impaired autoregulation can result in chronic ischemia. This ischemic environment contributes to oxidative stress and further damages retinal ganglion cells. These vascular factors help explain the occurrence of normal-tension glaucoma, where optic nerve damage develops despite intraocular pressure within normal limits [5].

Neurodegenerative mechanisms are increasingly recognized in glaucoma. Excitotoxicity caused by excessive glutamate, mitochondrial dysfunction, and impaired neurotrophic support all contribute to progressive neuronal loss. Inflammatory processes and activation of glial cells within the optic nerve and retina also play a role in sustaining neuronal injury. These mechanisms suggest that glaucoma shares similarities with other neurodegenerative disorders of the central nervous system.

Genetic factors influence individual susceptibility to glaucoma. Mutations and polymorphisms in genes related to aqueous humor dynamics, optic nerve structure, and cellular stress responses have been implicated in disease development. Aging further increases vulnerability by reducing the resilience of retinal ganglion cells and connective tissue support at the optic nerve head.

Conclusion

Glaucoma pathogenesis involves a complex interaction of mechanical, vascular, neurodegenerative, and genetic factors leading to progressive optic nerve damage. While lowering intraocular pressure remains the primary therapeutic approach, a deeper understanding of underlying mechanisms may lead to novel neuroprotective strategies. Continued research is essential to develop treatments that not only control pressure but also preserve neuronal function and prevent vision loss.

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