ISSN: 2584-0355
DOI: doi.org/10.51219/MCCRJ/Ian-Caldeira-Ruppen/236

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Medical & Clinical Case Reports Journal

https://urfpublishers.com/journal/case-reports

Vol: 3 & Iss: 2

Review

Optic Neuropathy: Etiologies and Advances in Treatment - An Updated Review

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Citation: Pugliese FR, Ruppen IC, Otani LH, de Araujo JD, de Oliveira RR, Otani FSI. Optic Neuropathy: Etiologies and Advances in Treatment - An Updated Review. *Medi Clin Case Rep J* 2025;3(2):914-916. DOI: doi.org/10.51219/MCCRJ/Ian-Caldeira-Ruppen/236

Received: 11 May, 2025; **Accepted:** 16 May, 2025; **Published:** 19 May, 2025

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ABSTRACT

Optic neuropathy comprises a spectrum of diseases that damage the optic nerve and impair the transmission of visual impulses, resulting in varying degrees of permanent vision loss. The most prevalent etiologies include ischemic, inflammatory, hereditary, compressive and traumatic mechanisms, which require specific diagnostic approaches integrating clinical examinations, imaging studies and genetic testing. Advances in optical coherence tomography (OCT) techniques orbital magnetic resonance imaging and serum biomarkers have enhanced our ability to detect axonal lesions early and guide targeted interventions. In the therapeutic realm, conventional strategies-such as systemic corticosteroids and immunomodulators-remain central to controlling inflammatory neuropathies, while antiplatelet therapy and strict management of cardiovascular risk factors underpin the treatment of ischemic forms. Over the past decade, notable progress has been made in gene therapies for Leber hereditary optic neuropathy, antioxidant neuroprotectors, transorbital electrical stimulation and mesenchymal stem cell applications, all in various phases of clinical trials. Despite their promise, these approaches still lack robust long-term efficacy and safety data, underscoring the need for large scale randomized studies. This review synthesizes diagnostic and therapeutic advances, discusses challenges such as pathophysiological heterogeneity, the difficulty of defining sensitive clinical endpoints and the high cost of cutting-edge techniques and outlines future trends rooted in precision medicine and multimodal protocols. The integration of emerging technologies, multidisciplinary collaboration and public policies facilitating access to innovative treatments will be crucial to improving outcomes for patients with optic neuropathy in the coming years.

Keywords: Optic neuropathy; Visual loss; Neuroprotection; Gene therapy; Regeneration

Introduction

Optic neuropathies are a major cause of irreversible visual impairment worldwide, affecting individuals across all age groups and socioeconomic backgrounds. It is estimated that over two million people suffer some degree of vision loss due to optic nerve damage[1]. The pathophysiology involves injury to the axons of retinal ganglion cells and secondary demyelination, leading to failure of signal transmission to the visual cortex. This process can be triggered by multiple etiologies, including

ischemic, inflammatory, hereditary, compressive, toxic and traumatic factors[2].

Clinically, presentations range from sudden to progressive loss of visual acuity, visual field defects and dyschromatopsia. Diagnostic evaluation demands integration of traditional ophthalmic exams-such as fundoscopy-with modern modalities like OCT, which quantifies retinal nerve fiber layer thickness, a sensitive marker of axonal damage[3]. Cranio orbital MRI is essential for detecting compressive lesions and characterizing demyelinating inflammatory processes[4].

Historically, treatment was limited to supportive measures and empirical corticosteroid use in inflammatory cases. However, significant advances have emerged recently, including gene therapies targeting specific mutations in Leber hereditary optic neuropathy[5], neuroprotective agents modulating axonal apoptosis[6] and visual rehabilitation via transorbital electrical stimulation[7]. These developments reflect the convergence of molecular biology, bioengineering and clinical ophthalmology. Nevertheless, important gaps remain-such as heterogeneity in clinical trials, lack of biomarkers predictive of therapeutic response and high costs of emerging Technologies and the complexity of axonal regeneration in the central nervous system poses further challenges to translating experimental findings into practice[8].

Objectives

This article aims to provide a comprehensive review of the main etiologies of optic neuropathy and discuss the most recent diagnostic and therapeutic advances from an evidence based medicine perspective.

Materials and Methods

A literature review was conducted using the PubMed, SciELO, Google Scholar and ScienceDirect databases.

Discussion

Therapeutic interventions for optic neuropathy have evolved substantially, encompassing pharmacological, genetic and technological strategies. In inflammatory neuropathies, high dose intravenous corticosteroids followed by oral taper remain the gold standard, shortening recovery time and reducing severity of vision loss[4]. Investigational biological therapies such as monoclonal antibodies against the interleukin 6 receptor are being explored, particularly in refractory autoimmune optic neuritis[9].

For ischemic neuropathies, secondary prevention hinges on stringent control of cardiovascular risk factors and antiplatelet therapy, though evidence for visual recovery remains limited[10]. Neuroprotective agents, including calcium channel antagonists and antioxidants, aim to mitigate oxidative stress and glutamatergic excitotoxicity that perpetuate axonal injury[6]. While in vitro and animal-model results are encouraging, clinical effectiveness is yet to be conclusively demonstrated.

Gene therapy stands out as the most dynamic field: AAV based vectors delivering the ND4 subunit cDNA have shown sustained visual acuity improvement in Leber hereditary optic neuropathy patients[5], inaugurating a new paradigm for mitochondrial disease treatment but also raising ethical, regulatory and cost accessibility concerns. Cellular therapies targeting axonal regeneration such as intravitreal mesenchymal stem cell infusions have demonstrated safety and neurotrophic effects, albeit with variable visual outcomes[11]. Transorbital electrical stimulation has been tested to enhance cortical plasticity and synaptic reconnection, but results remain inconsistent[7]. Collectively, these advances suggest an integrated care model combining early intervention, ongoing neuroprotection and tailored visual rehabilitation. Precision medicine leveraging machine learning to analyze large scale OCT and visual evoked potential data emerges as a promising approach for patient stratification and response prediction[12]. However, implementation will require substantial infrastructure, workforce training and sustainable economic frameworks.

Conclusions

Optic neuropathy represents a complex clinical and scientific challenge, demanding deep understanding of pathogenic mechanisms and multimodal therapeutic approaches. While conventional treatments like corticosteroids and vascular risk factor control remain foundational, the therapeutic landscape is rapidly broadening with gene therapies, neuroprotective agents and electrical and cellular stimulation techniques[13,14]. Gene therapy for specific mitochondrial mutations offers the most concrete advances, with sustained visual gains demonstrated in phase III trials; yet, generalizability, cost and long-term safety monitoring must be addressed. Cellular and electrical stimulation therapies show acceptable safety profiles but require methodological standardization and randomized trials to confirm functional benefits. Integrated strategies early intervention, detailed OCT monitoring, continuous neuroprotection and personalized visual rehabilitation are recommended and should be supported by multicenter research networks to enable large cohort inclusion and protocol comparability.

Public policies fostering the incorporation of innovative technologies into health systems, alongside professional training programs, will be vital to democratize access to these advances. In summary, the future of optic neuropathy management lies in precision medicine, interdisciplinary integration and combination therapies[15]. Investments in translational research and international collaboration are essential to turn promising discoveries into clinically effective interventions, thereby improving the quality of life for millions at risk of vision loss.

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