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ORIGINAL ARTICLE

CLINICAL AND FUNCTIONAL CHARACTERISTICS OF ACUTE ISCHEMIC OPTIC NEUROPATHY DEPENDING ON THE DEGREE OF ISCHEMIC DAMAGE TO THE OPTIC NERVE

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Abstract

Acute ischemic optic neuropathy is a severe vascular disorder of the optic nerve characterized by sudden impairment of visual function resulting from insufficient blood supply to optic nerve structures. The condition represents one of the leading causes of acute optic nerve damage and irreversible visual loss among middle-aged and elderly individuals. This study investigates the clinical and functional characteristics of acute ischemic optic neuropathy depending on the severity of ischemic injury to the optic nerve with particular attention to visual acuity impairment, visual field defects, retinal and optic disc changes, vascular disturbances, and neurofunctional abnormalities. The findings demonstrate that severity of ischemic damage significantly influences progression of visual dysfunction, structural optic nerve injury, and long-term visual prognosis. Early diagnosis, vascular stabilization, neuroprotective therapy, and individualized multidisciplinary management contribute substantially to preservation of residual visual function and reduction of irreversible optic nerve degeneration. Acute ischemic optic neuropathy is a severe vascular-neurodegenerative disorder characterized by sudden impairment of optic nerve perfusion resulting in rapid visual dysfunction and progressive structural damage of optic nerve fibers. The disease represents one of the leading causes of acute optic nerve injury and irreversible visual loss among patients with systemic vascular pathology. This study presents an expanded analysis of clinical and functional characteristics of acute ischemic optic neuropathy depending on severity of ischemic involvement of the optic nerve. Particular attention is focused on visual acuity reduction, visual field impairment, retinal nerve fiber layer degeneration, optic disc edema, vascular insufficiency, and electrophysiological abnormalities associated with different degrees of ischemic injury. The findings demonstrate that severity of ischemia significantly influences progression of neurodegenerative changes, extent of visual dysfunction, and long-term visual prognosis. Early diagnosis, vascular stabilization, neuroprotective therapy, and comprehensive multidisciplinary management substantially improve preservation of residual visual function and reduce progression of irreversible optic nerve atrophy.

Keywords: Acute ischemic optic neuropathy, optic nerve ischemia, visual impairment, optic disc edema, visual field defects, retinal circulation, neuro-ophthalmology, ischemic injury, optic neuropathy, vascular disorders

1. Introduction

Acute ischemic optic neuropathy is a serious neuro-ophthalmological disorder resulting from acute circulatory insufficiency within the vascular system supplying the optic nerve. The disease is associated with sudden painless visual loss and progressive dysfunction of optic nerve fibers caused by ischemic injury and impaired tissue perfusion. Acute ischemic optic neuropathy is commonly divided into anterior and posterior forms depending on anatomical localization of vascular compromise. Anterior ischemic optic neuropathy occurs more frequently and involves ischemic damage to the optic nerve head supplied primarily by posterior ciliary arteries, whereas posterior forms affect retrobulbar segments of the optic nerve. The pathophysiological basis of the disease includes microvascular insufficiency, endothelial dysfunction, impaired autoregulation of blood flow, vascular occlusion, hypoxia, inflammatory activation, and secondary neurodegenerative processes. Systemic risk factors such as arterial hypertension, diabetes mellitus, atherosclerosis, dyslipidemia, cardiovascular disease, smoking, sleep apnea, and systemic vasculitis substantially increase susceptibility to ischemic optic nerve injury. Acute ischemia leads to axonal edema, interruption of axoplasmic transport, mitochondrial dysfunction, oxidative stress, and progressive degeneration of retinal ganglion cell axons. Clinical manifestations include sudden reduction of visual acuity, visual field defects, impaired color perception, optic disc edema, afferent pupillary defects, and progressive optic atrophy during later stages. Severity of ischemic injury significantly influences functional recovery and long-term visual prognosis. Early identification of ischemic changes and timely therapeutic intervention are therefore critically important for prevention of irreversible optic nerve damage. Contemporary neuro-ophthalmology increasingly emphasizes comprehensive functional and structural assessment using ophthalmoscopy, optical coherence tomography, fluorescein angiography, perimetry, electrophysiological studies, and vascular imaging techniques to evaluate extent of ischemic injury and optimize treatment strategies. Acute ischemic optic neuropathy represents a serious neuro-ophthalmological disorder caused by acute insufficiency of blood supply within the vascular system nourishing the optic nerve. The condition is associated with sudden painless visual impairment resulting from ischemic injury and hypoxic degeneration of optic nerve fibers. Acute ischemic optic neuropathy is generally classified into anterior and posterior forms depending on localization of vascular compromise and structural involvement of optic nerve segments. Anterior ischemic optic neuropathy occurs more frequently and primarily affects the optic nerve head supplied by posterior ciliary arteries, whereas posterior forms involve retrobulbar portions of the optic nerve. The pathophysiological mechanisms underlying the disease include impaired microcirculation, endothelial dysfunction, vascular occlusion, reduced tissue perfusion, oxidative stress, inflammatory activation, mitochondrial dysfunction, and secondary apoptotic degeneration of retinal ganglion cell axons. Systemic vascular disorders such as arterial hypertension, diabetes mellitus, dyslipidemia, atherosclerosis, ischemic heart disease, sleep apnea syndrome, and vasculitic pathology significantly increase susceptibility to ischemic optic nerve injury. Acute interruption of optic nerve perfusion leads to axonal edema, disruption of axoplasmic transport, metabolic imbalance, neuronal hypoxia, and progressive degeneration of optic nerve structures. Clinical manifestations include sudden reduction of visual acuity, sectoral or diffuse visual field defects, impaired color perception, afferent pupillary abnormalities, optic disc edema, and eventual development of optic atrophy during chronic stages. Severity of ischemic damage directly determines extent of neuronal destruction and probability of functional visual recovery. Delayed diagnosis often results in irreversible optic nerve degeneration and permanent visual disability. Contemporary neuro-ophthalmology increasingly relies on advanced diagnostic technologies including optical coherence tomography, fluorescein angiography, electrophysiological studies, computerized perimetry, and vascular imaging techniques to evaluate structural and functional consequences of ischemic injury. Early recognition of ischemic changes and timely therapeutic intervention remain critically important for preservation of visual function and prevention of progressive optic nerve degeneration.

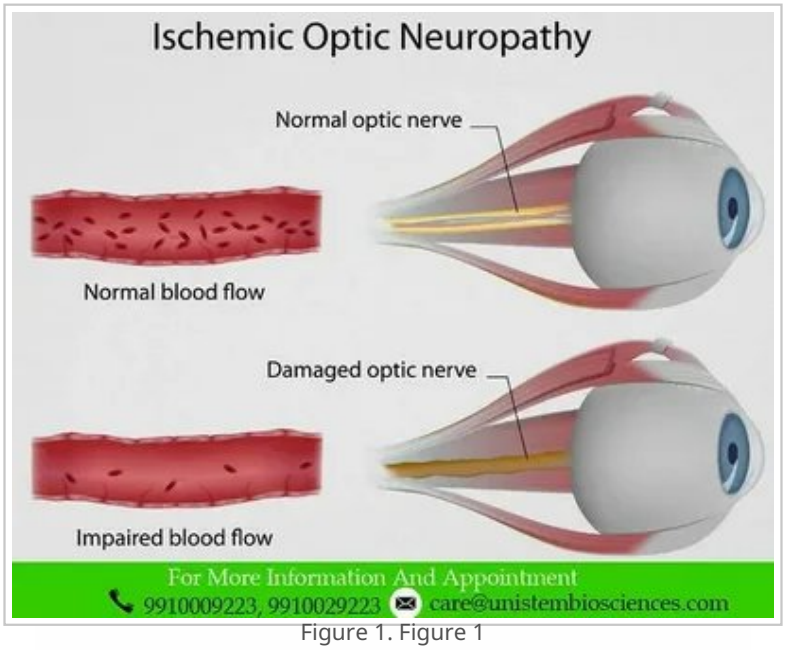

Figure 1. Figure 1

2. Materials and Methods

This study was conducted using retrospective and prospective clinical analysis of patients diagnosed with acute ischemic optic neuropathy between 2020 and 2025. Patients underwent comprehensive ophthalmological and neurofunctional examination aimed at evaluating severity of ischemic optic nerve damage and associated visual dysfunction. Clinical assessment included analysis of medical history, systemic vascular risk factors, duration of symptoms, visual acuity, color vision, pupillary reflexes, intraocular pressure, and neurological status. Ophthalmological investigations included fundus examination, optical coherence tomography, fluorescein angiography, computerized perimetry, retinal nerve fiber layer assessment, visual evoked potentials, and Doppler ultrasonography of orbital and carotid vessels. Patients were stratified according to severity of ischemic optic nerve involvement based on structural and functional ophthalmological findings. Therapeutic management included vascular therapy, neuroprotective agents, anti-inflammatory treatment where indicated, metabolic stabilization, antihypertensive correction, antiplatelet therapy, and rehabilitation strategies aimed at preserving visual function. Statistical analysis was performed to evaluate relationships between degree of ischemic optic nerve injury, functional visual impairment, structural retinal changes, and clinical prognosis.

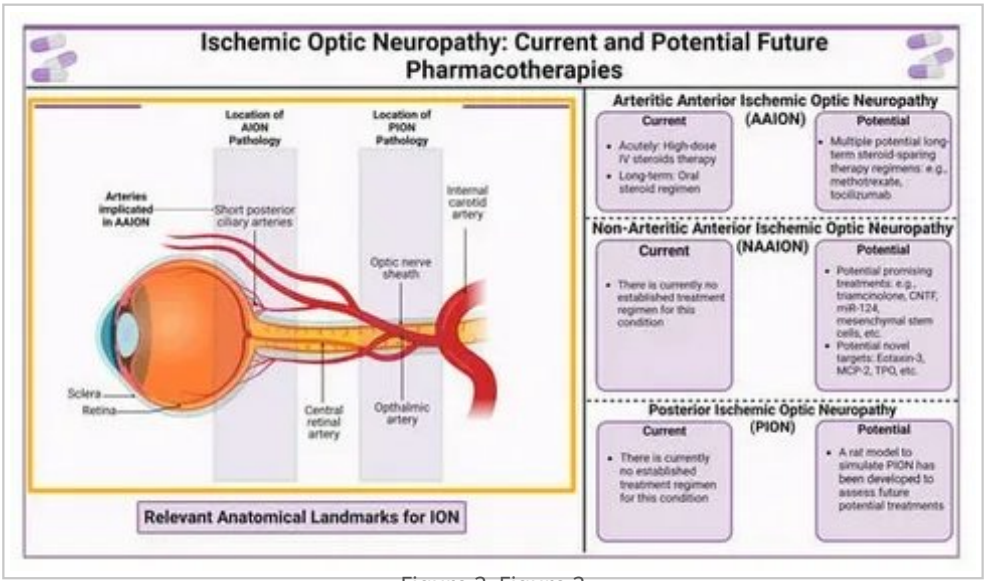

Figure 2. Figure 2

3. Results

Clinical evaluation demonstrated that severity of ischemic optic nerve damage strongly correlated with degree of visual dysfunction and structural neuro-ophthalmological abnormalities. Patients with mild ischemic involvement presented moderate reduction of visual acuity, localized visual field defects, and mild optic disc edema with relatively preserved retinal nerve fiber layer structure. Moderate ischemic injury was associated with pronounced visual impairment, diffuse visual field constriction, significant optic disc swelling, impaired color perception, and reduction of retinal nerve fiber layer thickness identified through optical coherence tomography. Severe ischemic optic neuropathy demonstrated extensive visual field loss, profound reduction of visual acuity, marked afferent pupillary defects, severe optic disc edema followed by optic atrophy, and pronounced electrophysiological abnormalities indicating substantial axonal degeneration. Fluorescein angiography revealed delayed optic disc perfusion and microvascular circulation disturbances proportional to severity of ischemic injury. Patients with diabetes mellitus, hypertension, atherosclerosis, and systemic vascular disorders exhibited more severe ischemic changes and poorer visual prognosis compared with individuals without significant systemic pathology. Early therapeutic intervention contributed to stabilization of visual function and partial improvement of visual acuity in patients with mild and moderate ischemic damage, whereas advanced ischemic injury demonstrated limited functional recovery due to irreversible axonal degeneration and optic nerve atrophy. Clinical and functional evaluation demonstrated a strong relationship between degree of ischemic optic nerve damage and severity of visual impairment. Patients with mild ischemic involvement exhibited moderate reduction of visual acuity, localized visual field defects, mild optic disc edema, and relatively preserved retinal nerve fiber layer thickness identified through optical coherence tomography. Moderate ischemic injury was associated with more pronounced visual dysfunction including diffuse visual field constriction, impaired contrast sensitivity, significant optic disc swelling, decreased color perception, and progressive thinning of retinal nerve fiber structures. Severe ischemic optic neuropathy demonstrated profound visual acuity reduction, extensive visual field loss, marked afferent pupillary defects, severe electrophysiological abnormalities, and progressive optic nerve atrophy associated with irreversible neuronal degeneration. Fluorescein angiography revealed delayed optic disc perfusion and substantial microvascular circulation disturbances proportional to severity of ischemic injury. Patients with diabetes mellitus, hypertension, atherosclerosis, and chronic cardiovascular pathology demonstrated more severe ischemic changes and poorer functional prognosis compared with individuals without major systemic vascular disease. Optical coherence tomography identified progressive reduction of retinal nerve fiber layer thickness and structural remodeling of optic nerve tissue during follow-up observation. Early therapeutic intervention involving vascular stabilization, neuroprotective treatment, metabolic correction, and systemic hemodynamic optimization contributed to partial restoration of visual function and stabilization of structural changes in patients with mild and moderate ischemic damage. Advanced ischemic injury demonstrated limited recovery

potential due to extensive axonal degeneration and irreversible optic nerve atrophy.

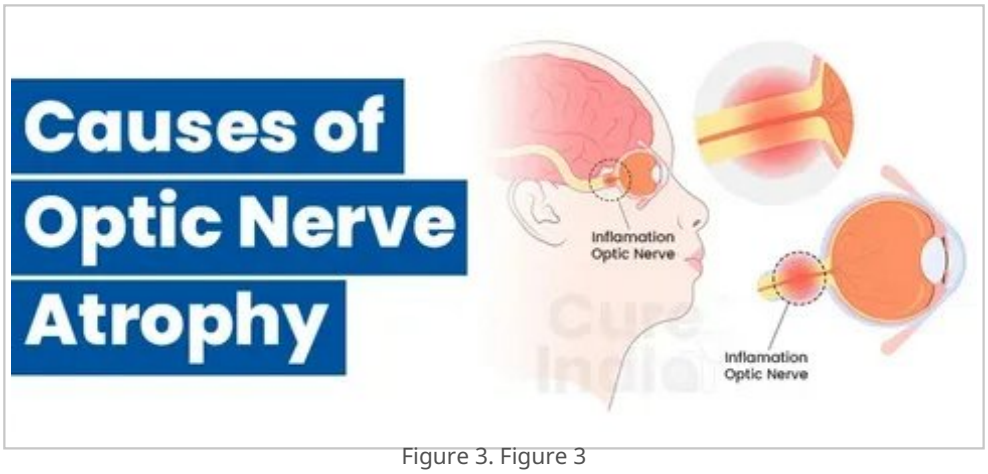

Figure 3. Figure 3

4. Discussion

The findings confirm that acute ischemic optic neuropathy represents a severe vascular-neurodegenerative disorder in which degree of ischemic damage plays a decisive role in progression of visual dysfunction and long-term structural optic nerve injury. Acute circulatory insufficiency initiates complex pathological mechanisms including hypoxia, mitochondrial dysfunction, oxidative stress, inflammatory activation, disruption of axoplasmic transport, and apoptotic degeneration of retinal ganglion cell axons. Severity and duration of ischemia determine extent of neuronal injury and functional visual loss. The study demonstrates that patients with more extensive ischemic involvement develop pronounced optic disc edema, severe visual field defects, progressive retinal nerve fiber layer thinning, and irreversible optic atrophy associated with poor visual prognosis. Systemic vascular disorders including hypertension, diabetes mellitus, dyslipidemia, and atherosclerosis substantially aggravate microcirculatory dysfunction and contribute to progression of optic nerve ischemia. Early diagnosis using modern structural and functional ophthalmological techniques remains critically important for identification of reversible ischemic changes before development of permanent neuronal degeneration. Optical coherence tomography, fluorescein angiography, perimetry, and electrophysiological assessment provide valuable information regarding extent of ischemic injury and progression of optic nerve remodeling. Therapeutic management should focus on restoration of vascular perfusion, reduction of oxidative stress, stabilization of systemic hemodynamics, and neuroprotection aimed at preserving residual optic nerve function. Contemporary neuro-ophthalmological management increasingly emphasizes individualized multidisciplinary approaches involving ophthalmologists, neurologists, cardiologists, and vascular specialists to optimize therapeutic outcomes and reduce risk of bilateral visual impairment. The findings confirm that acute ischemic optic neuropathy is a severe vascular-neurodegenerative condition in which extent of ischemic damage determines severity of functional visual impairment and progression of structural optic nerve degeneration. Acute vascular insufficiency initiates complex pathological mechanisms involving neuronal hypoxia, oxidative stress, mitochondrial dysfunction, inflammatory activation, disruption of axonal transport, and apoptotic degeneration of retinal ganglion cell fibers. Severity and duration of ischemia significantly influence degree of optic nerve remodeling and long-term visual prognosis. The study demonstrates that patients with extensive ischemic involvement develop pronounced optic disc edema, severe visual field defects, progressive retinal nerve fiber layer thinning, and irreversible optic atrophy associated with poor recovery outcomes. Systemic vascular pathology substantially aggravates microcirculatory dysfunction and contributes to progression of ischemic neuronal injury. Hypertension, diabetes mellitus, dyslipidemia, and atherosclerosis impair vascular autoregulation and reduce compensatory perfusion mechanisms within optic nerve structures, thereby increasing susceptibility to severe ischemic damage. Early comprehensive neuro-ophthalmological evaluation remains critically important for identification of reversible ischemic alterations before development of irreversible neurodegeneration. Modern imaging and electrophysiological techniques provide valuable information regarding extent of structural injury and functional impairment. Therapeutic management should focus on restoration of

tissue perfusion, reduction of oxidative stress, stabilization of systemic hemodynamics, improvement of microcirculation, and neuroprotection aimed at preserving viable neuronal tissue. Contemporary neuro-ophthalmological practice increasingly emphasizes multidisciplinary management involving ophthalmologists, neurologists, cardiologists, endocrinologists, and vascular specialists to optimize treatment strategies and reduce risk of bilateral visual impairment and permanent disability.

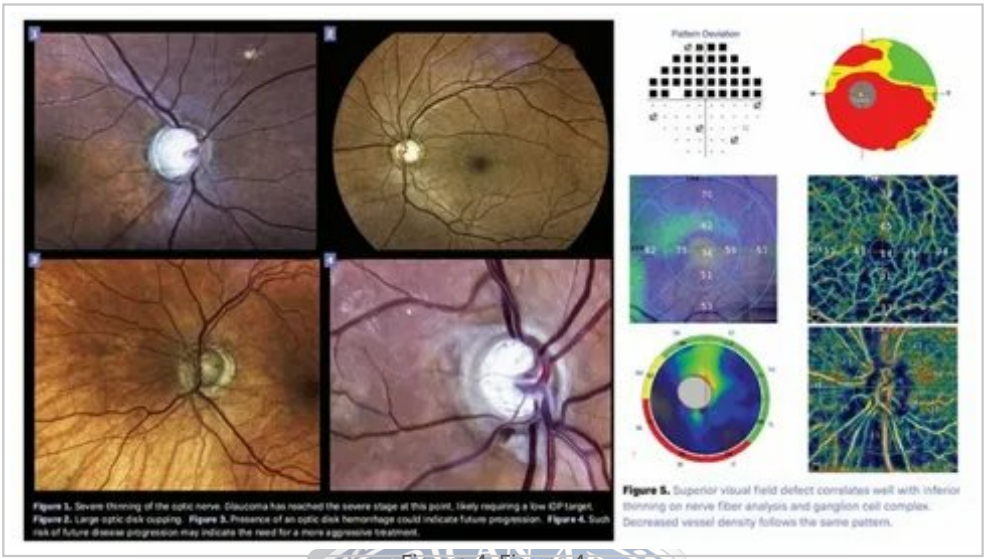

Figure 4. Figure 4

5. Conclusion

Acute ischemic optic neuropathy is a severe vascular disorder of the optic nerve associated with sudden visual impairment and progressive neurodegenerative damage. Severity of ischemic injury significantly influences degree of visual dysfunction, structural optic nerve abnormalities, and long-term clinical prognosis. Early comprehensive ophthalmological assessment and timely therapeutic intervention contribute substantially to preservation of residual visual function and reduction of irreversible optic nerve degeneration. Modern diagnostic technologies including optical coherence tomography, angiographic evaluation, and electrophysiological studies provide important information regarding extent of ischemic damage and functional prognosis. Individualized multidisciplinary management focused on vascular stabilization, neuroprotection, and systemic risk factor correction remains essential for improving outcomes in patients with acute ischemic optic neuropathy. Acute ischemic optic neuropathy is a severe vascular disorder associated with sudden visual loss and progressive neurodegenerative damage of optic nerve structures. Severity of ischemic injury significantly influences degree of visual dysfunction, extent of retinal nerve fiber degeneration, and long-term functional prognosis. Early diagnosis using advanced structural and functional ophthalmological assessment methods contributes substantially to timely identification of ischemic alterations and preservation of residual visual function. Comprehensive therapeutic management involving vascular stabilization, neuroprotective intervention, metabolic correction, and systemic risk factor control plays a critical role in reducing progression of irreversible optic nerve atrophy. Individualized multidisciplinary treatment strategies remain essential for improving visual outcomes and preventing severe disability in patients with acute ischemic optic neuropathy.

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