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Etiologic Spectrum of Optic Disc Edema in Mongolia: Clinical Insights from a Resource Limited Setting

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Objective: Optic disc edema (ODE) is a critical clinical sign that often marks the initial manifestation of a vision- or life-threatening systemic disease, depending on its underlying etiology. This first Mongolian observational study defines the demographic characteristics, etiologies, and clinical features of ODE, assessing diagnostic reassessment as a challenge for multiple specialties. **Methods:** We conducted a 5-year retrospective–prospective observational study with reassessment to evaluate etiologies, clinical features, and visual outcomes. **Results:** Seventy patients (90 eyes) with optic disc edema were included in the analysis. Demyelinating optic neuritis (36.4%) predominated in younger females, ischemic cases (44.4%) in older patients, and infectious causes (26.9%, syphilis) in males. Inflammatory etiologies were mostly uveitis. Baseline vision was poor, with 20% presenting with profound impairment (Visual acuity (VA)≤20/400). Severe loss (<6/120) was most frequent in demyelinating cases (72.2%). Visual recovery was most significant in demyelinating and inflammatory cases, and minimal in ischemic cases, with initial BCVA being the strongest prognostic factor (p value < 0.05). **Conclusion:** We conclude that optic disc edema shows a diverse age-related etiologic spectrum, with demyelinating and ischemic causes predominating, with differing prognoses and a high burden of infectious (mainly syphilis) and uveitis cases. Initial visual acuity was the key prognostic factor, emphasizing the importance of systemic screening and multidisciplinary care.

Keywords: Optic Disc, Papilledema, Optic Neuropathy, Optic neuritis, Visual acuity

Introduction

The optic nerve, divided into intraocular, intraorbital, intracanalicular, and intracranial segments, connects the retina to the brain. The optic disc, the only portion visible with ophthalmoscopy, represents the convergence of neural and vascular elements. Disturbance at the optic nerve head can result in swelling of the disc, clinically recognized as disc

edema.[1] Because of its unique anatomical configuration, the optic disc is selectively vulnerable, and disc edema represents a non-specific manifestation of various pathological insults, most often related to axoplasmic stasis, ischemia, inflammation, or compression.[2,3] Optic disc edema is a frequent finding in both general ophthalmology and urgent vision loss referrals, with multiple possible etiologies. It typically lasts 4–6 weeks and, if untreated, can progress to optic pallor, irreversible vision loss, or indicate an underlying life-threatening disorder.[4]

It is not a diagnosis by itself but rather a clinical sign of various systemic causes that go beyond ophthalmology, often reflecting neurological, ischemic vascular, infectious, or ocular inflammatory diseases.[5] For neurologists, it may indicate increased intracranial pressure, also known as papilledema or demyelinating optic neuritis (ON); for internists, it can be associated with infectious, autoimmune, or metabolic disorders; and for vascular specialists, it may suggest ischemic optic neuropathies with poor prognosis.[6-8] Even in primary care, recognizing optic disc edema (ODE) is essential, as it might be the first sign of a life-threatening condition requiring urgent referral. In some cases, the initial diagnosis may change after further investigations or subsequent neurological events, making reassessment crucial for an accurate diagnosis. Fundus examination is a quick and practical way to evaluate the optic nerve, but the cause of disc swelling usually cannot be determined from appearance alone. Optical coherence tomography (OCT) provides a non-invasive method for detecting axonal loss and monitoring disc edema. Additionally, orbital CT, MRI (including MRI of the orbits with or without contrast and fat-suppression techniques), ultrasonography, and multimodal imaging can be used as complementary diagnostic tools.[9,10] Early recognition and prompt referral are vital for preserving vision and life, especially in resource-limited settings where delays can lead to irreversible blindness or missed systemic illnesses.

The etiologic classification of optic disc edema is mainly based on consensus, derived from decades of clinical experience and documented in major neuro-ophthalmology textbooks and reviews, rather than from a single official guideline. It is both pathophysiological and clinical. Pathophysiological, it involves mechanisms such as axoplasmic stasis, ischemia, inflammation, infection, compression, or toxic demyelination. Clinically, it represents a final common pathway of optic nerve head swelling, where differentiating features on presentation and fundus examination help determine the underlying cause.[2,12-14]

In Central Asia—especially in sparsely populated countries like Mongolia—the etiologies and clinical patterns of ODE remain poorly understood, and no national-level data currently exist to characterize its burden. Therefore, this study aims to define the demographic characteristics, etiological spectrum, clinical features, and visual outcome of ODE in Mongolia, while also examining diagnostic reassessment during follow-up. These findings underscore ODE as a complex, multispecialty diagnostic challenge.

Materials and Methods

This retrospective–prospective observational study was performed at the First Central Hospital of Mongolia, a national referral hospital, and involved patients with optic disc edema. The retrospective group included cases admitted from 2020 to 2024, while the prospective group consisted of patients newly diagnosed in 2025 under the care of the lead investigator.

Data extracted included demographics, laterality, baseline and follow-up best-corrected visual acuity (BCVA), fundus findings, and systemic or neurological associations. Visual acuity was converted to logMAR units for analysis. For cases with incomplete records, patients were recalled for follow-up evaluation, which included further imaging, such as optical coherence tomography and magnetic resonance imaging, fundus photography, serologic testing, and assessment of treatment response, to refine the etiologic classification. The findings from ocular imaging are provided as an example (Figure 1).

As etiologies were not always established at presentation, diagnostic re-examination was performed using clinical data, imaging, laboratory investigations, and follow-up findings to refine diagnostic accuracy. Cases were categorized by presumed etiology into the following groups: demyelinating, infectious, inflammatory, ischemic, papilledema, or idiopathic. The diagnosis depended on the following criteria: for demyelinating optic neuritis — Optic Neuritis Treatment Trial (ONTT), ischemic — Ischemic Optic Neuropathy Decompression Trial (IONDT) criteria, papilledema — Dandy criteria of idiopathic intracranial hypertension (IIH).[10,15,16] Infectious, idiopathic (autoimmune), and diabetic optic neuropathies are defined as conditions that confirm the presence of disease based on laboratory findings, to rule out other substantial conditions.

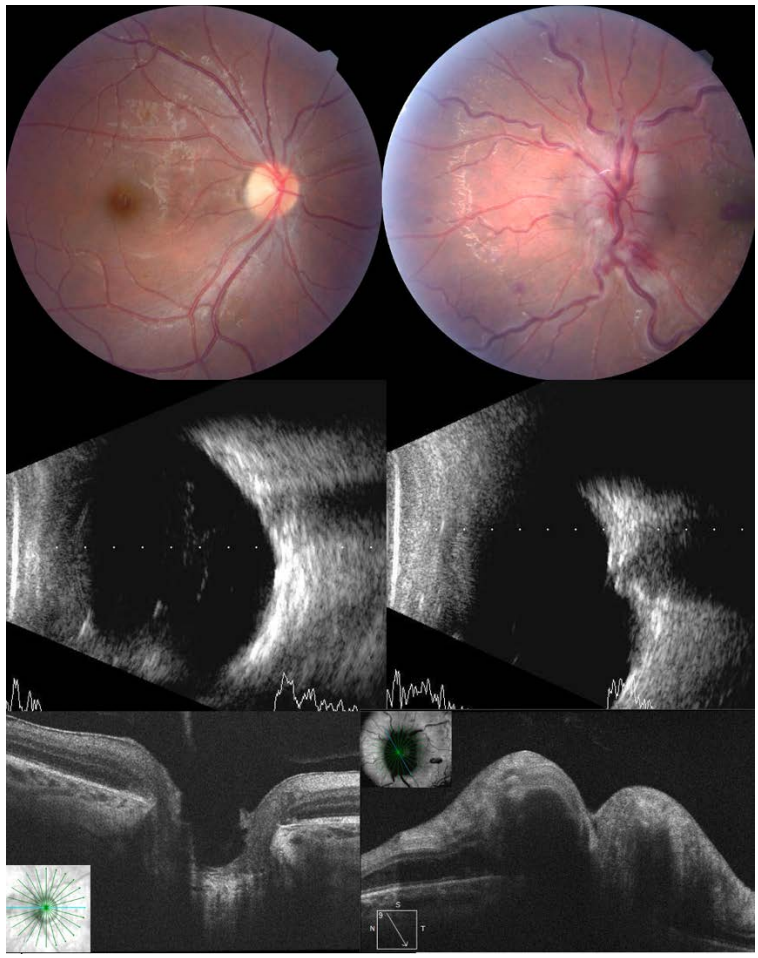

Figure 1. Multimodal imaging of the normal optic disc anatomy versus optic disc edema of one patient's two eyes. A). Color fundus photograph of both eyes of a patient with leukemic optic nerve infiltration with optic nerve dysfunction. Right eye showing standard optic disc with normal margin and normal vascular structure, B). left eye showing marked disk edema characterized with optic disc elevation, peripapillary flame-shaped hemorrhages, congestion, and dilation of the retinal vein's tortuosity, consistent with leukemic optic disc infiltration. (A 28-year-old Mongolian male with acute myeloid leukemia (AML) diagnosed 7 months ago initially, s/p after fourth chemotherapy, was referred to our emergency clinic due to acute loss of vision in the left eye.) C). Ultrasonography of the right eye revealed a standard optic disc with a hypoechogenic contour and no edema. The left eye showed bulging of the optic disc into the intraocular cavity and increased papilledema. E). The OCT (Optical Coherent Tomography) radial HD image of the optic nerve showed normal findings in both the right and left eyes. Markedly elevated optic nerve head on the left side.

This combined design enabled a thorough review of past cases and ongoing observation of new instances, allowing for more accurate etiologic classification and outcome evaluation through ongoing reassessment, rather than relying solely on admission data. All ON patients were included regardless of age; additionally, patients treated with steroid pulse therapy were also included. Visual outcomes were measured by comparing initial and final best-corrected visual acuity (BCVA). Profound visual impairment was defined as BCVA ≤ 0.05 (Snellen equivalent ≤ 20/400) in at least one eye.

Statistical analyses included one-way ANOVA for age distribution, chi-square testing for gender and laterality distribution, and parametric or non-parametric tests for clinical features and visual acuity improvement, depending on data distribution.

Results

Patient Demographics

Age and gender variation: Seventy patients (90 eyes) with ODE were included (26 males, 36.7%; 44 females, 63.3%), with bilateral involvement in 20 (28.6%). The mean age varied significantly across etiologies (ANOVA, $p < 0.001$). Non-arteritic ischemic optic neuropathy (NAION) and arteritic anterior Ischemic Optic Neuropathy (AION) were associated with older age (60–70 years), whereas demyelinating optic neuritis (ON) and papilledema cases clustered in younger individuals (<45 years). In contrast, demyelinating optic neuritis (ON) (50%), infectious (70%), and inflammatory (54.5%) etiologies predominated in younger adults (30–50 years). Patients <30 years were most frequently affected by papilledema (60%). A significant association was observed between gender and etiology ($\chi^2 = 14.936$, $p < 0.001$). Demyelinating optic neuritis (ON) and NAION were more common in females, whereas infectious and traumatic etiologies predominated in males. In this study, unilateral ocular involvement was most commonly observed in 50 patients (71.4%), particularly in the demyelinating, idiopathic, and ischemic groups. There is a significant difference in eye laterality based on etiology groups ($\chi^2 = 13.4839$, $p < 0.001$) (Table 1).

Table 1. Demographic and Clinical Characteristics of Optic Disc Edema by Etiology

Variables	Demyelinating	Infectious	Inflammatory	Idiopathic	Ischemic	Papilledema	Orbital	Diabetic	*p* value
Mean age (years)	42.7 ± 12.6	39.4 ± 9.8	42 ± 10.5	43 ± 13.3	63.4 ± 10.3	32.2 ± 14.7	41.6 ± 15.1	45.3 ± 22.8	<0.001[a]
>70	-	-	-	-	4 (44.4%)	-	-	-	
50-70	7 (38.9%)	1 (10%)	2 (18.2%)	2 (22.2%)	4 (44.4%)	1 (20%)	3 (60%)	2 (66.7%)	
30-50	9 (50%)	7 (70%)	6 (54.5%)	6 (66.7%)	1 (11.1%)	1 (20%)	1 (20%)	-	
<30	2 (11.1%)	2 (20%)	3 (27.3%)	1 (11.1%)	-	3 (60%)	1 (20%)	1 (33.3%)	
Gender									<0.001[b]
Male	2 (7.7%)	7 (26.9%)	4 (15.4%)	6 (23.1%)	0 (0%)	2 (7.7%)	1 (3.8%)	4 (15.4%)	
Female	16 (36.4%)	3 (6.8%)	7 (15.9%)	3 (6.8%)	9 (20.5%)	3 (6.8%)	2 (4.5%)	1 (2.3%)	
Laterality									<0.001[b]
Unilateral	16 (32%)	4 (8%)	6 (12%)	8 (16%)	9 (18%)	4 (8%)	2 (4%)	1 (2%)	
Bilateral	2 (10%)	6 (30%)	5 (25%)	1 (5%)	0 (0%)	1 (5%)	1 (5%)	4 (20%)	

a – One factor ANOVA test, b – Chi-square test

Etiologic Spectrum

Demyelinating optic neuritis was the leading cause, mainly multiple sclerosis (44.4%), Neuromyelitis Optica Spectrum Disorder (NMOSD) (27.8%), and Myelin Oligodendrocyte Glycoprotein Antibody–Associated Disease (MOGAD) (16.7%). Infectious etiologies were largely syphilis (80%), and inflammatory cases were uveitis primarily (72.7%). Ischemic causes were predominantly attributed to NAION (58.3%). Orbital cases were chiefly traumatic (60%), while idiopathic/autoimmune etiologies were overwhelmingly autoimmune (88.9%). Less frequent causes included diabetic papillopathy (classified within ischemic optic neuropathies), Neuro-Bechet's disease, Leber Hereditary Optic Neuropathy (LHON), methanol intoxication, leukemic infiltration, and retrobulbar hemorrhage (Table 2).

Table 2. Etiologic Spectrum of Optic Disc Edema and Its Subtypes in a Mongolia

Categories	Causes of Optic Disc Edema				
	n	(%)	Causes	n	(%)
Demyelinating	18	25.7%	NMOSD	5	7.1%
			MOGAD	3	4.3%
			MS	8	11.4%
			Other	2	2.9%
Infectious	10	14.3%	Syphilitic	8	11.4%
			Viral	2	2.9%
Inflammatory	11	15.7%	Neuro-Behçet's disease	1	1.4%
			LHON	1	1.4%
			Methanol toxication	1	1.4%
			Uveitic optic neuropathy	8	11.4%
Ischemic	12	17.1%	NAION	7	10.0%
			AION	2	2.9%
			Diabetic papilledema	3	4.3%
Orbital	5	7.14%	Leukemic infiltration	1	1.4%
			Traumatic	3	4.3%
			Retrobulbar hemorrhage	1	1.4%
Autoimmune/Idiopathic	9	12.9%	GPA	1	1.4%
			Autoimmune	8	11.4%
Raised Intracranial pressure	5	7.14%	Papilledema	5	7.1%
Total	70	100%	Total	70	100%

NMOSD - Neuromyelitis Optica Spectrum Disorder; MOGAD - Myelin Oligodendrocyte Glycoprotein Antibody–Associated Disease; MS - Multiple Sclerosis; LHON - Leber Hereditary Optic Neuropathy; NAION - Non-Arteritis Anterior Ischemic Optic Neuropathy; AION - Arteritis Anterior Ischemic Optic Neuropathy; GPA - Granulomatosis with Polyangiitis

Clinical Features

The most frequent features were ocular pain (45.6%), relative afferent pupillary defect (45.6%), and color vision deficit (44.4%). Inflammatory and demyelinating cases frequently presented with pain, and marked disc swelling, while ischemic cases were typically painless with pallid swelling (Figure 2).

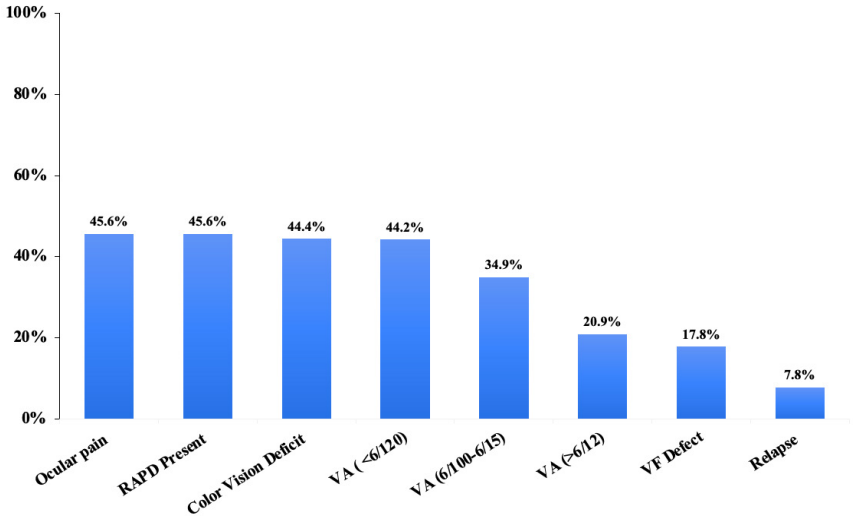

Figure 2. Clinical Features of Optic Disc Edema

Visual Outcomes and Prognostic Factors

The initial mean vision was very poor (around 20/320), with no significant difference among different causes (*p* value = 0.06). Visual recovery was highest in demyelinating, inflammatory, and diabetic-related cases, while ischemic causes (NAION, AION) showed minimal improvement. Outcomes in infectious cases depended on how quickly treatment was provided. At the start, 20% of patients (13/65) had severe visual impairment (BCVA ≤ 20/400). Severe vision loss (<6/120) was most common in demyelinating (72.2%), inflammatory (46.7%), and orbital (50%) cases, while papilledema more frequently presented with mild loss (50%) (Table 3). Final improvement in visual acuity did not significantly differ between causes (*p* value = 0.437). However, initial best-corrected visual acuity (BCVA) was the strongest predictor of outcome (*p* value< 0.05).

Most patients with ODE in this study were managed with intravenous (IV) steroids (72.9%), reflecting the predominance of demyelinating and inflammatory etiologies. A smaller proportion received antibiotics such as intravenous (IV) benzathine penicillin G (11.4%) for neurosyphilis or other targeted therapies for infectious causes. Overall, nearly three-quarters of patients showed visual improvement, with 59.7% achieving meaningful recovery and 13.4% showing slight improvement, underscoring the importance of timely treatment initiation in determining outcomes (Table 4).

Table 3. Baseline Visual Acuity by Etiology of Optic Disc Edema

Visual acuity	Initial BCVA			Final BCVA		*p* value
	<6/120	6/100-6/15	<6/12	Improved	Worsen / stable	
Demyelinating	13 (72.2%)	5 (27.8%)	-	14 (77.8%)	4 (22.2%)	0.01*
Infectious	6 (37.5%)	4 (25%)	6 (37.5%)	10 (62.5%)	6 (37.5%)	0.15
Inflammatory	7 (46.7%)	6 (40%)	2 (13.3%)	6 (54.5%)	5 (45.5%)	0.54
Idiopathic	3 (30%)	7 (70%)	-	4 (44.4%)	5 (55.6%)	0.03*
Ischemic	4 (44.4%)	3 (33.4%)	2 (22.2%)	7 (77.7%)	2 (22.3%)	0.63
Papilledema	1 (12.5%)	3 (37.5%)	4 (50%)	1 (12.5%)	7 (87.5%)	0.53
Orbital	3 (50%)	1 (16.7%)	2 (33.3%)	3 (50%)	3 (50%)	0.71
Diabetic	1 (25%)	1 (25%)	2 (25%)	2 (50%)	2 (50%)	0.82

*BCVA - Best Corrected Visual Acuity, *statistical significance,*
NOTE: p value was defined by ANOVA test depends on initial and final BCVA

Table 4. Management and Treatment Outcomes of Optic Disc Edema

Management	n	%
Intravenous pulse steroid	54	77.1%
Oral steroid	3	4.2%
Additional Immunosuppressant	3	4.2%
Mannitol	1	1.4%
Intravenous benzathine penicillin	8	11.4%
Antiviral	1	1.4%
No treatment	8	11.4%
Treatment response		
Improved	40	59.7%
Slight improvement	9	13.4%
No change	17	25.4%
Worsened	1	1.5%

Discussion

Our findings confirm that optic disc edema (ODE) is a systemic warning sign that requires multidisciplinary care, particularly in resource-limited settings. In this study, ischemic etiologies predominated in older patients, while demyelinating and papilledema cases were more frequent in younger individuals. Female predominance in demyelinating ON aligned with the epidemiology of multiple sclerosis, whereas male predominance in infectious cases likely reflected sociocultural and occupational exposures. The study findings that demyelinating papilledema is more common in young adults and female patients were shown in previous literature. [17-19]

The etiologic spectrum in our study was primarily composed of demyelinating ON (36.4%) and ischemic optic neuropathies (44.4%), consistent with patterns reported globally. However, we observed a notably higher burden of infectious etiologies, particularly syphilitic optic neuropathy (80%), and uveitic inflammatory causes, which are relatively less common in high-income countries.[20,21] Similar distributions have been reported in tertiary centers in India, Turkey, and Saudi Arabia, where demyelinating ON accounts for over 30% of cases.[6,22,23] These regional variations likely reflect differences in disease prevalence, access to diagnostic tools, and systemic comorbidity profiles.

Internationally, Idiopathic Intracranial Hypertension (IIH) predominates in North America, optic neuritis (ON) and NAION are most common in patients with profound visual loss. NAAION/ON are leading causes in Korea.[13,25] Despite geographic variations, prognostic patterns were consistent: optic neuritis generally demonstrated better recovery, ischemic causes had poor outcomes paralleling Western cohorts, while favorable recovery in demyelinating and inflammatory cases aligned with Asian reports.[26,27] Across all settings, initial BCVA emerged as the strongest predictor of visual outcome, underscoring the importance of systemic evaluation, vascular risk management, and timely intervention.[28-30] Visual prognosis in ODE depends on the etiology, with 20% of our study participants presenting with profound visual loss, underscoring the urgency of early diagnosis, systemic evaluation, and access to treatment to prevent irreversible blindness in resource-limited settings. This is the first observational study analysis of ODE from Mongolia. Our findings demonstrate that ODE is not merely an ophthalmic concern, but a multisystemic warning sign that spans neurology, infectious diseases, rheumatology, and vascular medicine. Early recognition and interdisciplinary management are crucial for preventing irreversible blindness and identifying underlying systemic diseases.

This study's limitations include its single-center design, a

small sample size of 70 participants, reliance on BCVA, and inconsistent follow-up, all of which limit its generalizability and long-term accuracy. Moreover, hospital-based data are not sufficient for reliably estimating population incidence. Future research should involve multi-center prospective studies with standardized imaging, thorough serology, uniform treatment protocols, and a wider range of outcome measures to improve diagnostic accuracy and broader applicability.

Conclusions

This first study of optic disc edema (ODE) in Mongolia revealed that demyelinating and ischemic etiologies were predominant, each with distinct prognostic profiles. The notably high burden of infectious and uveitis cases underscores the importance of routine screening for infectious and autoimmune diseases and the need for early referral. Among all clinical factors, initial visual acuity emerged as the strongest predictor of visual outcome, highlighting the critical role of early recognition and multidisciplinary management. Comprehensive demographic and clinical profiling remain essential for effective triage, systemic evaluation, and the prevention of irreversible vision loss.

Conflict of Interest

The authors declare that they have no funding or conflicts of interest to disclose.

Authors Contribution

Anarsaikhan Narmandakh: conceptualization, funding acquisition, project administration, methodology, supervision, validation, investigation, writing—original draft preparation, writing-review editing, visualization
Altanzul Tumurpurev: formal analysis, resources, investigation, software
Bat-Erdene Bataa: conceptualization
Khulgun Enkhjargal: conceptualization
Dorjpagma Rentsendorj: data curation
Sunderya Yostoikhuu: data curation
Undarmaa Tumurbaatar: visualization, resources
Enkhtuul Sedbazar: conceptualization, funding acquisition
Enkhzul Damdin: conceptualization, funding acquisition
Tuvshintugs Baljir: visualization, resources
Sainbilig Disan: validation, visualization, resources
All authors have read and agreed to the published version of the manuscript.

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