

ORIGINAL RESEARCH PAPER		Neurology
 AETIOLOGICAL SPECTRUM AND SHORT-TERM CLINICAL OUTCOME OF NEUROGENIC VISION LOSS: A CASE SERIES FROM A TERTIARY CARE NEUROLOGY CENTRE IN SOUTH INDIA		KEY WORDS: Neurogenic vision loss; optic neuritis; neuromyelitis optica spectrum disorder; ischaemic optic neuropathy; posterior reversible encephalopathy syndrome; case series
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ABSTRACT	Background: Vision loss of neurological origin ("neurogenic vision loss") arises from disorders anywhere along the visual pathway — retina to occipital cortex — or from non-organic causes, and is aetiologically far more heterogeneous than primary ophthalmic disease. Data characterising this spectrum from Indian tertiary neurology practice are limited. Objective: To describe the clinical and aetiological spectrum of neurogenic vision loss presenting to a tertiary care neurology department in one year period and to assess short-term visual outcome at three months. Methods: This descriptive case series included 43 consecutive patients with neurogenic vision loss evaluated in the Department of Neurology, Madurai Medical College and Government Rajaji Hospital, Madurai. Patients underwent detailed clinical, neuro-ophthalmological, neuroimaging and laboratory evaluation. Visual disability was graded using the Kurtzke Visual Functional System Score (FS-Visual, 0–6) at baseline and at 3-month follow-up. Categorical variables were expressed as frequencies/percentages and continuous variables as mean ± SD or median (IQR). The paired baseline and 3-month FS-Visual scores were compared using the Wilcoxon signed-rank test; $p < 0.05$ was considered significant. Results: The mean age was 41.2 ± 17.6 years (range 12–75) with a near-equal sex distribution (female 53.5%). Optic neuritis (20.9%; NMOSD 4, MOGAD 3, multiple sclerosis 2) and orbital apex syndrome (16.3%; rhino-orbital-cerebral mucormycosis in 4) were the commonest diagnoses. Broad aetiological groups were inflammatory/demyelinating (23.3%), ischaemic-vascular (20.9%), infective (14.0%), hypertensive/PRES-related (9.3%), functional (7.0%), traumatic (4.7%), toxic-nutritional (4.7%), hereditary (4.7%), granulomatous/orbital inflammatory (4.7%), neoplastic (2.3%) and raised intracranial pressure (2.3%). Laterality was unilateral in 51.2% and bilateral in 48.8%. Twelve patients (27.9%) had a recurrent course, predominantly antibody-mediated optic neuritis. The median FS-Visual score improved from 5 (IQR 4–5) at baseline to 2 (IQR 1–3) at 3 months (mean 4.53 ± 0.83 vs 2.33 ± 1.43 ; Wilcoxon signed-rank test, $p < 0.0001$), with improvement in 40/43 (93.0%), no change in 2/43 (4.7%) and worsening in 1/43 (2.3%, hereditary optic atrophy). Conclusion: Neurogenic vision loss encompasses a wide range of inflammatory, vascular, infective, toxic-metabolic, hereditary and functional disorders. Prompt aetiology-directed treatment was associated with significant short-term improvement in visual disability in the majority of patients, although outcome varied with the underlying cause, underscoring the importance of early and accurate aetiological diagnosis.	
	INTRODUCTION Vision loss is a common and disabling neurological presentation that may result from a lesion anywhere along the visual pathway, extending from the retina and optic nerve through the chiasm, optic radiations and occipital cortex, or from higher-order visual processing and non- organic (functional) mechanisms.[1] Unlike primary ophthalmic disease, in which the causative lesion is usually confined to the eye, neurogenic vision loss is aetiologically heterogeneous and may be the presenting feature of demyelinating and antibody-mediated disease, cerebrovascular disease, central nervous system infection, raised intracranial pressure, toxic-metabolic derangement, hereditary mitochondrial or neurodegenerative disorders, orbital or cavernous sinus pathology, and psychiatric or functional neurological disorders.[1,2] An accurate and timely aetiological diagnosis is critical, since several of these conditions — antibody-mediated optic neuritis in neuromyelitis optica spectrum disorder (NMOSD) and myelin oligodendrocyte glycoprotein antibody-associated disease (MOGAD), giant cell arteritis- related arteritic anterior ischaemic optic neuropathy (AAION), rhino-orbital-cerebral mucormycosis, and posterior reversible encephalopathy syndrome (PRES) — are treatable or preventable causes of blindness in which delay in diagnosis carries a high risk of irreversible visual loss or, in some instances, death.[3–7] Conversely, other causes such as hereditary optic neuropathies and neurodegenerative disorders carry a guarded visual prognosis despite treatment.[8,9] The Expanded Disability Status Scale (EDSS) Visual Functional System Score (FS-Visual), originally described by Kurtzke for grading disability in multiple sclerosis, provides a simple, widely used, ordinal 0–6 scale for grading the severity of visual impairment and has been extended in clinical practice to grade visual disability from causes other than demyelinating disease.[10] Published Indian data specifically characterising the aetiological spectrum of neurogenic vision loss evaluated by neurologists, as opposed to ophthalmologists, remain limited. We present a case series of 43 patients with neurogenic vision loss evaluated at a tertiary care neurology department in South India, describing the clinical and aetiological spectrum and the short-term (3-month) visual outcome following aetiology-directed treatment. Materials and Methods Study design and setting This was a hospital-based, observational case series conducted in the Department of Neurology, Madurai Medical College and Government Rajaji Hospital, Madurai, Tamil Nadu, a tertiary care referral centre, over a period of one year from July 2025 to June 2026. Consecutive patients presenting with vision loss attributed to a neurological cause, evaluated jointly with the Department of Ophthalmology where indicated, were enrolled after obtaining informed written consent. The study was conducted after approval from the	

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1

Institutional Ethics Committee, Madurai Medical College.

Inclusion criteria

1. Patients of either sex and any age presenting with acute, subacute or chronic vision loss (unilateral or bilateral) attributed to a lesion of the optic nerve, chiasm, retro-chiasmal visual pathway, occipital cortex, or to a functional/non-organic neurological cause.

2. Vision loss confirmed, by comprehensive neuro-ophthalmological assessment (visual acuity, colour vision, pupillary reactions, fundus examination, visual field assessment where feasible), to be neurogenic rather than attributable to a primary anterior-segment or refractive ophthalmic disorder.

3. Availability of relevant neuroimaging (contrast-enhanced MRI brain and/or orbit) and supportive laboratory or serological workup (e.g., aquaporin-4 and MOG antibody testing, vasculitic and metabolic workup) as clinically indicated to establish an aetiological diagnosis.

4. Patients with complete baseline clinical data and available follow-up assessment at 3 months from presentation.

5. Patients (or their legal guardians, in the case of minors) who provided informed written consent for inclusion in the study.

Exclusion criteria

1. Vision loss attributable primarily to an ophthalmic disorder not of neurological origin (e.g., cataract, primary glaucoma, refractive error, retinal detachment, age-related macular degeneration, corneal opacity, uveitis without neurological involvement).

2. Pre-existing congenital or long-standing childhood blindness unrelated to the presenting illness.

3. Patients with incomplete case records, or those lost to follow-up before the scheduled 3-month assessment.

4. Patients or guardians not consenting to participate in the study.

5. Patients in whom a reliable neuro-ophthalmological or Functional System scoring assessment could not be performed owing to severe concomitant cognitive impairment, sedation, or critical illness precluding cooperation.

Data collection

For each patient, demographic details, presenting complaints, laterality of vision loss, associated systemic risk factors and comorbidities, history of similar past episodes (recurrence), final aetiological diagnosis (established by clinical, radiological, serological and/or histopathological criteria appropriate to each disease category), and treatment administered were recorded on a structured proforma. Severity of vision loss was graded independently at presentation (baseline) and at 3-month follow-up using the Kurtzke EDSS Visual Functional System Score (FS-Visual), an ordinal scale ranging from 0 (normal) to 6 (bilateral visual acuity worse than 20/200, practical blindness), by the treating neurologist blinded to the previous score wherever feasible.[10]

Statistical analysis

Data were entered in Microsoft Excel and analysed using Python (SciPy v1.x) / SPSS software. Continuous variables (age, FS-Visual scores) were tested for normality and are expressed as mean $\pm$ standard deviation (SD) and median with interquartile range (IQR); categorical variables (sex, diagnosis, aetiology, laterality, recurrence, risk factors, treatment) are expressed as frequencies and percentages.

Because the FS-Visual score is an ordinal variable with a skewed, non-normal distribution, the paired baseline and 3-month scores for each patient were compared using the non-parametric Wilcoxon signed-rank test; a paired t-test was additionally performed as a sensitivity analysis. A two-tailed p-value <0.05 was considered statistically significant. No imputation was performed for missing data; all 43 patients had complete baseline and follow-up FS-Visual scores.

RESULTS

Forty-three patients with neurogenic vision loss met the inclusion criteria during the study period. The mean age was 41.2 ± 17.6 years (median 35 years; range 12–75 years). There were 23 females (53.5%) and 20 males (46.5%). Vision loss was unilateral in 22 patients (51.2%) and bilateral in 21 (48.8%). A recurrent course — either a further episode in the same eye or involvement of the contralateral eye — was recorded in 12 patients (27.9%), most of whom had antibody-mediated or demyelinating optic neuritis (NMOSD, MOGAD or multiple sclerosis) and rhino-orbital-cerebral mucormycosis. Baseline demographic and clinical characteristics are summarised in Table 1.

Table 1. Baseline demographic and clinical characteristics (n = 43)

Characteristic	Value
Age, years (mean $\pm$ SD)	41.2 $\pm$ 17.6
Age, years (median, range)	35 (12–75)
Sex, Female : Male, n (%)	23 (53.5) : 20 (46.5)
Laterality – Unilateral, n (%)	22 (51.2)
Laterality – Bilateral, n (%)	21 (48.8)
Recurrent course, n (%)	12 (27.9)

The final clinical diagnoses and their broad aetiological categorisation are shown in Table 2. Optic neuritis was the single commonest diagnosis (9/43, 20.9%), attributed to NMOSD (aquaporin-4 antibody positive) in 4, MOGAD (MOG antibody positive) in 3, and multiple sclerosis in 2 patients. Orbital apex syndrome accounted for 7 patients (16.3%), of whom 4 had rhino-orbital-cerebral mucormycosis in the setting of uncontrolled diabetes mellitus, and one each had Tolosa-Hunt syndrome, orbital sarcoidosis and orbital lymphoma. PRES accounted for 4 patients (9.3%), all with an antecedent hypertensive disorder (gestational hypertension in 3, chronic kidney disease-related hypertension in 1). Cortical blindness secondary to bilateral posterior circulation (occipital) infarction accounted for 3 patients (7.0%), and functional (non- organic/conversion) visual loss for another 3 (7.0%). When grouped by broad aetiological category, inflammatory/demyelinating causes were the most frequent (10/43, 23.3%), followed by ischaemic-vascular causes (9/43, 20.9%) and infective causes (6/43, 14.0%).

Table 2. Distribution of final diagnoses and broad aetiological categories (n = 43)

Aetiological category / Diagnosis	Underlying aetiology	n (%)
Inflammatory / demyelinating		10 (23.3)
Optic neuritis	NMOSD (AQP4-Ab+)	4 (9.3)
Optic neuritis	MOGAD (MOG-Ab+)	3 (7.0)
Optic neuritis	Multiple sclerosis	2 (4.7)
CRION	Vasculitis	1 (2.3)
Ischaemic / vascular		9 (20.9)
Cortical blindness	Posterior circulation stroke	3 (7.0)
NAION	Systemic hypertension	2 (4.7)
CVAO	Vascular occlusion	2 (4.7)
AAION	Giant cell arteritis	1 (2.3)
CRAO	Vascular occlusion	1 (2.3)
Infective		6 (14.0)
Orbital apex syndrome	Rhino-orbital-cerebral mucormycosis	4 (9.3)

Optochiasmatic arachnoiditis	Tuberculous meningitis	2 (4.7)
Hypertensive encephalopathy	PRES (gestational HTN 3, CKD 1)	4 (9.3)
Functional / psychogenic	Conversion disorder	3 (7.0)
Traumatic	Head/orbital trauma	2 (4.7)
Toxic / nutritional		2 (4.7)
Toxic optic neuropathy	Ethambutol	1 (2.3)
Nutritional optic neuropathy	Thiamine/B12 deficiency (alcohol use)	1 (2.3)
Hereditary		2 (4.7)
LHON	Mitochondrial mutation	1 (2.3)
Optic atrophy	Complicated hereditary spastic paraplegia	1 (2.3)
Granulomatous / orbital inflammatory		2 (4.7)
Orbital apex syndrome	Tolosa-Hunt syndrome	1 (2.3)
Orbital apex syndrome	Sarcoidosis	1 (2.3)
Neoplastic	Orbital lymphoma	1 (2.3)
Raised intracranial pressure	IIH (obesity)	1 (2.3)
Primary headache disorder	Migraine	1 (2.3)

Systemic hypertension (11/43, 25.6%) and diabetes mellitus (12/43, 27.9%, including uncontrolled diabetes) were the commonest identifiable systemic risk factors, followed by seropositivity for aquaporin-4 (4/43, 9.3%) or MOG (3/43, 7.0%) antibodies. Details of risk- factor distribution are shown in Table 3.

Table 3. Distribution of associated systemic risk factors / disease associations (n = 43)*

Risk factor / association	n (%)
Systemic hypertension	11 (25.6)
Diabetes mellitus (type 2 / uncontrolled)	12 (27.9)
Aquaporin-4 antibody positivity	4 (9.3)
MOG antibody positivity	3 (7.0)
Gestational hypertension	3 (7.0)
Alcohol use disorder	2 (4.7)
Hereditary / genetic disorder	2 (4.7)
Vasculitis	1 (2.3)
Obesity	1 (2.3)
Psychiatric illness	1 (2.3)
Tuberculosis	1 (2.3)
Trauma / assault	1 (2.3)
Migraine	1 (2.3)
No identifiable risk factor	5 (11.6)

*Percentages do not total 100 as some patients had more than one association (e.g., concurrent diabetes and hypertension).

Treatment was tailored to the underlying aetiology. Corticosteroids, alone or combined with a steroid-sparing immunosuppressant (mycophenolate mofetil, azathioprine or rituximab), were used in 15 patients (34.9%), chiefly those with inflammatory optic neuritis, CRION and optochiasmatic arachnoiditis. Antihypertensive therapy with or without antiplatelet/statin therapy was used in 10 patients (23.3%) with vascular or hypertensive aetiologies. Surgical debridement with systemic antifungal and antibacterial therapy was administered to all 4 patients with rhino- orbital- cerebral mucormycosis. Anti-tuberculous therapy was given in both patients with tuberculous optochiasmatic arachnoiditis. Supportive, psychiatric or counselling-based management was used in the functional visual loss group, and disease-specific therapy (mitochondrial cocktail, acetazolamide, thiamine/B12 replacement, cessation of the

offending drug, or oncological treatment) was used for the hereditary, raised intracranial pressure, nutritional/toxic and neoplastic categories respectively. The median Kurtzke FS- Visual score improved from 5 (IQR 4–5) at baseline to 2 (IQR 1–3) at the 3-month follow-up visit (mean 4.53 ± 0.83 versus 2.33 ± 1.43). This improvement was statistically significant on the Wilcoxon signed-rank test ($W = 4.5$, $p < 0.0001$), a finding corroborated by a paired t-test performed as a sensitivity analysis ($t = 11.64$, $p < 0.0001$). Overall, visual function improved in 40 of 43 patients (93.0%), remained unchanged in 2 (4.7%; both with severe orbital apex involvement from mucormycosis or advanced optic atrophy already at the ceiling of the scale), and worsened in 1 patient (2.3%) — the child with optic atrophy complicating hereditary spastic paraplegia, in keeping with the expected progressive course of this neurodegenerative disorder. Outcome by aetiological category is summarised in Table 4.

Table 4. Baseline and 3-month FS-Visual score by aetiological category

Category (n)	Baseline FS- Visual, mean	3-month FS- Visual, mean	Mean improvement
Inflammatory / demyelinating (10)	4.6	1.9	2.7
Ischaemic / vascular (9)	4.9	3.1	1.8
Infective (6)	5.3	3.8	1.5
Hypertensive / PRES (4)	4.5	1.5	3.0
Functional (3)	4.3	1.0	3.3
Traumatic (2)	4.5	2.0	2.5
Toxic / nutritional (2)	3.5	1.5	2.0
Hereditary (2)	3.5	2.5	1.0*
Granulomatous / orbital (2)	4.0	1.0	3.0
Neoplastic (1)	4.0	2.0	2.0
Raised ICP (1)	4.0	1.0	3.0
Migraine (1)	4.0	0.0	4.0

*One of the two hereditary cases (hereditary spastic paraplegia-related optic atrophy) worsened over follow-up, while the other (Leber hereditary optic neuropathy) improved; the net category mean reflects this divergence.

Overall, the greatest improvement was observed in functional, hypertensive/PRES-related, and granulomatous/orbital inflammatory categories, all of which are recognised to have a favourable prognosis with prompt treatment, whereas improvement was comparatively modest in the infective (rhino-orbital-cerebral mucormycosis, tuberculous arachnoiditis) and hereditary categories, consistent with the more guarded prognosis of these conditions.

DISCUSSION

This case series illustrates the broad aetiological diversity that underlies neurogenic vision loss encountered in neurology practice, spanning inflammatory-demyelinating, ischaemic-vascular, infective, hypertensive, functional, traumatic, toxic-nutritional, hereditary, granulomatous and neoplastic mechanisms. This heterogeneity mirrors that reported in other neuro-ophthalmological cohorts, in which optic neuropathies and chiasmal/retro-chiasmal disorders together account for a wide range of underlying systemic and central nervous system disease.[1,2]

Optic neuritis was the single largest diagnostic group in our cohort, and antibody-mediated disease (NMOSD and MOGAD) together outnumbered classical multiple sclerosis-associated optic neuritis, a pattern increasingly recognised in

Indian and other Asian cohorts where NMOSD and MOGAD are relatively more prevalent than in Western populations.[3,4] Aquaporin-4 and MOG antibody-associated optic neuritis are recognised to have a higher rate of recurrence and greater risk of severe, bilateral visual loss than typical demyelinating optic neuritis, which is consistent with the fact that antibody-mediated optic neuritis accounted for the majority of the recurrent episodes in our series.[3,4] Early high-dose corticosteroid therapy, followed by steroid-sparing immunosuppression (mycophenolate mofetil, azathioprine or rituximab) in antibody-positive disease, remains the standard of care and was associated with favourable short-term visual recovery in our patients, in keeping with published treatment recommendations.[4,11]

Orbital apex syndrome due to rhino-orbital-cerebral mucormycosis was the second commonest diagnosis, occurring exclusively in patients with poorly controlled diabetes mellitus, consistent with the well-documented association between hyperglycaemia, ketoacidosis-prone states and invasive fungal rhino-orbital-cerebral infection.[6] Despite aggressive multimodal therapy — surgical debridement combined with systemic antifungal and antibacterial agents — visual improvement in this group was the least among all categories, reflecting the recognised poor visual prognosis once orbital apex involvement has occurred, and underscoring the importance of early suspicion and treatment in diabetic patients presenting with acute orbital or sino-nasal symptoms.[6,7]

Ischaemic causes, comprising non-arteritic and arteritic anterior ischaemic optic neuropathy (NAION and AAION), cortical blindness from posterior circulation stroke, and central retinal/vein artery-vein occlusion equivalents, together formed the second largest category, reflecting the strong background prevalence of systemic hypertension, diabetes and atherosclerotic vascular disease in this cohort. The distinction between NAION and AAION is of particular importance, since AAION due to giant cell arteritis is a medical emergency, and prompt institution of high-dose corticosteroids is required to prevent involvement of the contralateral eye; our single AAION case, treated with corticosteroids and subsequently rituximab, is in keeping with current recommendations for refractory or relapsing giant cell arteritis.[8,9]

Posterior reversible encephalopathy syndrome accounted for a meaningful proportion of cases, entirely related to hypertensive disorders including gestational hypertension, and showed the most favourable outcome among all categories with prompt blood pressure control and anti-oedema measures, consistent with the classically reversible nature of this syndrome when recognised and treated early.[5]

Functional (non-organic) visual loss, seen in three patients including one child, responded well to reassurance, counselling and psychiatric management, underscoring the importance of recognising functional neurological presentations, which can closely mimic organic vision loss but typically show inconsistencies on careful bedside examination and carry a good prognosis with appropriate multidisciplinary management.[12]

Hereditary optic neuropathies — Leber hereditary optic neuropathy and optic atrophy complicating hereditary spastic paraplegia — showed the most limited and, in one case, negative visual trajectory, consistent with the recognised natural history of mitochondrial and neurodegenerative optic neuropathies, for which currently available therapies (mitochondrial cocktail with antioxidants and coenzyme Q10 analogues) offer, at best, modest benefit.[13,14] Toxic and nutritional optic neuropathies (ethambutol-induced and thiamine/B12 deficiency-related)

improved substantially following withdrawal of the offending agent and vitamin replacement respectively, reaffirming that these are largely reversible causes of vision loss if recognised early.[15,16]

Overall, the significant improvement in FS-Visual score across the cohort ($p < 0.0001$) highlights that the great majority of neurogenic vision loss, even when severe at presentation, carries a favourable short-term visual prognosis once an accurate aetiological diagnosis directs specific treatment — a message of particular relevance in resource-limited settings where diagnostic delay remains common.

Limitations

This study has the inherent limitations of a small, single-centre, descriptive case series without a comparator group, which precludes causal inference and limits generalisability. The 3-month follow-up period, while adequate to demonstrate early treatment response, does not capture longer-term visual outcome, relapse, or the development of disability, which would be particularly relevant for the relapsing antibody-mediated and neurodegenerative categories. The FS-Visual score, though simple and reproducible, is a relatively coarse ordinal measure and may not fully capture more granular changes in visual acuity, colour vision or visual field. Finally, the diversity of aetiologies, while illustrative of the overall spectrum, resulted in small numbers within individual diagnostic subgroups, limiting statistical comparison between them.

CONCLUSION

Neurogenic vision loss in this tertiary care neurology cohort encompassed a broad spectrum of inflammatory-demyelinating, ischaemic-vascular, infective, hypertensive, functional, traumatic, toxic-nutritional, hereditary and neoplastic disorders, with inflammatory and ischaemic causes predominating. Aetiology-directed treatment was associated with a statistically significant improvement in visual disability at 3 months in the large majority of patients, although the magnitude of recovery varied by underlying cause, being most favourable in hypertensive, functional and granulomatous/orbital inflammatory disease and least favourable in infective and hereditary aetiologies. These findings reinforce the need for prompt, systematic neuro-ophthalmological and neurological evaluation of patients presenting with unexplained vision loss, so that treatable causes are identified and managed without delay.

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Authors' Contributions

S.K.R.: Concept, design, manuscript preparation and final approval. R.K.: Data acquisition, literature review and manuscript drafting. P.K.M.: Data analysis, critical revision and manuscript editing. All authors reviewed and approved the final manuscript.

Ethical Approval

This study was conducted in accordance with the Declaration of Helsinki and was approved by the Institutional Ethics Committee, Madurai Medical College, Madurai. Written informed consent was obtained from all patients, or from parents/guardians in the case of minors, including consent for publication of anonymised clinical data.

Conflict of Interest

The authors declare no conflict of interest.

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REFERENCES

1. Biousse V, Newman NJ. Diagnosis and clinical features of common optic neuropathies. *Lancet Neurol*. 2016;15(13):1355-67.
2. Pula JH, Yuen CA. Eyes and stroke: the visual aspects of cerebrovascular disease. *Stroke Vasc Neurol*. 2017;2(4):210-20.
3. Wingerchuk DM, Banwell B, Bennett JL, Cabre P, Carroll W, Chitnis T, et al. International consensus diagnostic criteria for neuromyelitis optica spectrum disorders. *Neurology*. 2015;85(2):177-89.
4. Banwell B, Bennett JL, Marignier R, Kim HJ, Brilot F, Flanagan EP, et al. Diagnosis of myelin oligodendrocyte glycoprotein antibody-associated disease: International MOGAD Panel proposed criteria. *Lancet Neurol*. 2023;22(3):268-82.
5. Fugate JE, Rabinstein AA. Posterior reversible encephalopathy syndrome: clinical and radiological manifestations, pathophysiology, and outstanding questions. *Lancet Neurol*. 2015;14(9):914-25.
6. Cornely OA, Alastruey-Izquierdo A, Arenz D, Chen SCA, Dannaoui E, Hochhegger B, et al. Global guideline for the diagnosis and management of mucormycosis: an initiative of the European Confederation of Medical Mycology in cooperation with the Mycoses Study Group Education and Research Consortium. *Lancet Infect Dis*. 2019;19(12):e405-21.
7. Yeh S, Foroozan R. Orbital apex syndrome. *Curr Opin Ophthalmol*. 2004;15(6):490-8.
8. Ponte C, Grayson PC, Robson JC, Suppiah R, Gribbons KB, Judge A, et al. 2022 American College of Rheumatology/EULAR classification criteria for giant cell arteritis. *Ann Rheum Dis*. 2022;81(12):1647-53.
9. Hayreh SS. Ischemic optic neuropathy. *Prog Retin Eye Res*. 2009;28(1):34-62.
10. Kurtzke JF. Rating neurologic impairment in multiple sclerosis: an expanded disability status scale (EDSS). *Neurology*. 1983;33(11):1444-52.
11. Trebst C, Jarius S, Berthele A, Paul F, Schippling S, Wildemann B, et al. Update on the diagnosis and treatment of neuromyelitis optica: recommendations of the Neuromyelitis Optica Study Group (NEMOS). *J Neurol*. 2014;261(1):1-16.
12. Bruce BB, Newman NJ. Functional visual loss. *Neurol Clin*. 2010;28(3):789-802.
13. Yu-Wai-Man P, Chinnery PF. Leber Hereditary Optic Neuropathy. In: Adam MP, Feldman J, Mirzaa GM, et al., editors. *GeneReviews* [Internet]. Seattle (WA): University of Washington; 1993-2024 [updated 2021 Jul 29].
14. Finsterer J. Hereditary spastic paraplegias with autosomal dominant, recessive, X-linked, or maternal trait of inheritance. *J Neurol Sci*. 2009;278(1-2):5-15.
15. Chamberlain PD, Sadaka A, Berry S, Lee AG. Ethambutol optic neuropathy. *Curr Opin Ophthalmol*. 2017;28(6):545-51.
16. Sharma P, Sharma R. Toxic optic neuropathy. *Indian J Ophthalmol*. 2011;59(2):137-41.
17. Levin LA, Beck RW, Joseph MP, Seiff S, Kraker R. The treatment of traumatic optic neuropathy: the International Optic Nerve Trauma Study. *Ophthalmology*. 1999;106(7):1268-77.
18. Mac Grory B, Schrag M, Biousse V, Furie KL, Gerhard-Herman M, Lauer AK, et al. Management of central retinal artery occlusion: a scientific statement from the American Heart Association. *Stroke*. 2021;52(6):e282-94.
19. Friedman DI, Liu GT, Digre KB. Revised diagnostic criteria for the pseudotumor cerebri syndrome in adults and children. *Neurology*. 2013;81(13):1159-65.
20. Headache Classification Committee of the International Headache Society (IHS). *The International Classification of Headache Disorders*, 3rd edition. Cephalalgia. 2018;38(1):1-211.
21. Kidd D, Burton B, Plant GT, Graham EM. Chronic relapsing inflammatory optic neuropathy (CRION). *Brain*. 2003;126(Pt 2):276-84.
22. Beck RW, Cleary PA, Anderson MM Jr, Keltner JL, Shults WT, Kaufman DI, et al. A randomized, controlled trial of corticosteroids in the treatment of acute optic neuritis. *N Engl J Med*. 1992;326(9):581-8.
23. Hinchey J, Chaves C, Appignani B, Breen J, Pao L, Wang A, et al. A reversible posterior leukoencephalopathy syndrome. *N Engl J Med*. 1996;334(8):494-500.
24. Sethi HS, Naik M, Gupta VS. Orbital apex syndrome due to mucormycosis in a patient with diabetes: case report and literature review. *Indian J Ophthalmol*. 2021;69(10):2637-40.