Etiology and Clinical Profile of Non-Glaucomatous Optic Neuropathy in a Tertiary Government Hospital in the Philippines

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ABSTRACT

Objective: This study described the etiology and clinical profile of non-glaucomatous optic neuropathy (NGON) cases in a tertiary government hospital in Quezon City, Philippines.

Method: This was a retrospective, cross-sectional study of patients seen at a neuro-ophthalmology clinic of a tertiary referral center in Quezon City, Philippines from January 01, 2008 to December 31, 2020. The patients' medical records were reviewed and the following data were extracted and analyzed: demographic data, mechanism of NGON, and clinical profile.

Results: A total of 911 patient records were reviewed. Most patients were males (61.3%) within the economically productive age group (72.7%). Overall, the top three mechanisms that contributed to NGON were (1) trauma-related (25.9%), (2) ischemic (19.8%), and (3) inflammatory causes (18.0%). When patients were stratified by age, the most common causes in the pediatric group were traumatic (33.9%), inflammatory (28.6%), and papilledema-related (11.3%) optic neuropathies. In the middle age group, traumatic (29.1%), inflammatory (18.5%), and ischemic (17.8%) etiologies predominated. Among the elderly, ischemic (44.0%), drug-induced (26.1%), and compressive (17.9%) causes were most frequently identified.

Conclusion: Traumatic optic neuropathy emerged as the leading cause of vision loss from NGON in both pediatric and middle aged groups. In the absence of trauma, further investigations should focus on inflammatory and papilledema-related etiologies in the pediatric and middle aged groups. Among the elderly, ischemia and drug-induced toxic optic neuropathies were the most prevalent, with thorough history-taking being crucial for identifying medication-related causes.

Keywords: non-glaucomatous optic neuropathy, trauma, optic atrophy

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Non-glaucomatous optic neuropathies (NGON) comprise a heterogeneous group of disorders that affect the optic nerve, excluding those caused by glaucoma. These conditions vary widely in terms of patient demographics, clinical presentation, management strategies, and visual prognoses.[1] The etiologies of NGON are broadly categorized into 11 subtypes: those associated with papilledema, compressive lesions, congenital anomalies, hereditary conditions, infiltrative processes, inflammatory causes, ischemia, nutritional deficiencies, post-radiotherapy effects, post-traumatic, and toxic exposures.[1]

The differentiation of glaucomatous optic neuropathy (GON) from NGON is mainly through the application of thorough clinical history and ophthalmologic clinical examination, including intraocular pressure measurement and careful examination of the optic disc, and the use of ancillary tests such as optical coherence tomography (OCT) and automated visual field perimetry (VFP).[1-3]

The clinical presentation of NGON varies by etiology – often resulting to acute loss of vision in inflammatory or ischemic cases, or insidious in compressive or hereditary forms.[2,3] Unlike GON, NGON may present with additional ophthalmologic signs such as ptosis or ocular motility disturbances, and may be associated with neurologic signs and symptoms. On examination, color and contrast sensitivity deficits, normal intraocular pressures, and variable optic disc pallor further support an NGON diagnosis.

The disease burden of GON is estimated to be 3-5% of the global population aged 40 years and above.[4] On the other hand, overall global incidence of NGON is poorly defined. In the Philippines, the Third National Survey on Blindness identified optic atrophy, regardless of cause, as a leading contributor of bilateral blindness and low vision.[5] To date, the only published study on NGON etiologies among Filipinos was conducted in 1979 by Reyes-Noche and Fajardo, who reviewed records of 134 patients seen in a tertiary referral center and reported inflammation, compression, and trauma as the three most common etiologies of optic atrophy.[6]

There remains to be a gap in knowledge due to the scarcity and outdated data on NGON. The study aimed to address this gap by identifying and ranking the etiologic causes of NGON and describing their clinical characteristics in an urban Filipino population.

METHODS

This retrospective, cross-sectional study was conducted at the Neuro-Ophthalmology Section of the Department of Health Eye Center (DOHEC) of the East Avenue Medical Center, a tertiary government hospital in Quezon City, Philippines. The study adhered to the principles of the Declaration of Helsinki and conformed with the guidelines set forth by the International Council for Harmonization - Good Clinical Practice. Ethics approval was obtained from the East Avenue Medical Center Institutional Ethics Review Board.

The medical records of all Filipino patients, regardless of age, who were diagnosed with NGON by a qualified neuro-ophthalmologist between January 1, 2008 and December 31, 2020 were reviewed. Included cases had an identified etiology categorized as: associated with papilledema, compressive, congenital, hereditary, infiltrative, inflammatory, ischemic, nutritional deficiency, post-radiotherapy, post-traumatic, or toxic.

Charts that were illegible or had incomplete data were excluded. Patients who had concomitant ocular diseases (e.g., vitreoretinal, corneal pathology) were also excluded from the study in order to reduce their confounding effects on visual acuity data.

The following data were collected: demographic data, presenting symptom, baseline best-corrected visual acuity, baseline optic nerve status (normal, swollen, atrophic), and clinical diagnosis. The cases were stratified by age into 3 groups: pediatric (< 19 years), middle age (19 - 59 years), and elderly (≥ 60 years).

Visual acuity was converted to logarithm of the minimum angle of resolution (LogMAR) values for statistical analysis. Visual acuities recorded as counting fingers, hand movement, light perception, and no light perception were assigned a LogMAR value of 2.0, 2.4, 2.7, and 3.0, respectively.[7]

Statistical Analysis

Data was entered and analyzed in a spreadsheet (Microsoft Excel 2017©, Microsoft 365, Washington, United States). The number of cases per etiology was ranked accordingly. Measures of central tendency were used for continuous data, while frequency distribution was used for categorical data.

RESULTS

A total of 2,242 charts were reviewed. Of these, 1,331 were excluded due to various reasons (**Figure 1**). A total of 911 patient records were included in the study.

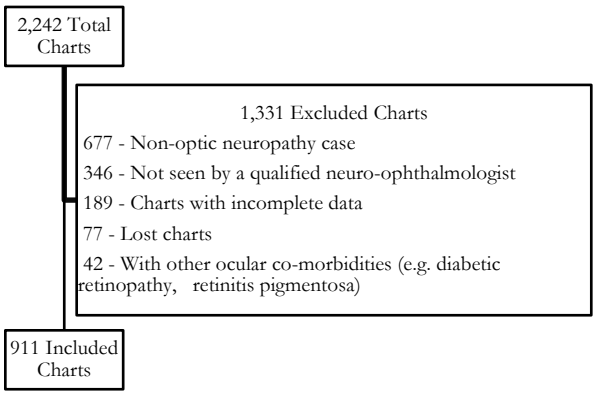

Figure 1. Excluded Charts

Table 1 shows the four most frequent etiologies of NGON identified in this study: traumatic (25.9%), ischemic (19.8%), inflammatory (18.0%), and compressive (16.4%). Patient age ranged from 2 months to 88 years, with a mean age of 40.2 $\pm$ 18.0 years. There were more males (61.2%), with a male to female ratio of 1.6:1. Unilateral optic nerve involvement was more common, present in 53.3% of cases.

The primary presenting complaint in 93.9% of cases was decreased visual acuity. The mean BCVA of the study cohort was 1.09 $\pm$ 1.10 logMAR, which translates to a presenting visual acuity that is slightly worse than 6/60.

Bilateral involvement was observed in etiologies related to drug toxicity, papilledema-related, drug toxicity, hereditary, nutritional, and post-radiation (all 100%). Compressive etiologies presented bilaterally in 60.7% of cases. Presentation was predominantly unilateral in etiologies related to trauma (95.8%), infiltrative (66.7%), ischemic (57.2%), and congenital (57.1%). Inflammatory etiology presented either unilaterally (51.2%) or bilaterally (48.8%).

Presenting BCVA was notably worst among cases of toxic optic neuropathy due to ethambutol intake. Most cases demonstrated non-remarkable optic disc findings in both eyes. Optic atrophy was present in 27.8% of all eyes on initial presentation.

Distribution of Etiologic Profile by Sex and Age Group

Males were predominant in etiologies related to trauma (86.9%), ischemia (64.4%), and hereditary (100%) optic neuropathies. The single case of nutritional deficiency-related optic neuropathy was a 52-year-old male with chronic alcohol abuse of 30 years.

Females contributed more cases in inflammation-related (67.1%) optic neuropathy. The two female cases of infiltrative optic neuropathy were related to breast cancer with central nervous system metastasis, the single case of post-radiation optic neuropathy followed whole-brain irradiation for intracranial meningioma. No sex predilection was observed in etiologies related to compression, sequelae of increased intracranial pressure, and congenital optic neuropathies.

The pediatric age group accounted for majority of optic neuropathies secondary to congenital (57.2%) causes. This age group also contributed large proportions in etiologies related to papilledema (27.1%), inflammation (20.1%), hereditary (16.7%), and post-traumatic (16.5%).

On the other hand, the middle age group accounted for majority of optic neuropathies secondary to hereditary (83.3%), post-traumatic (81.8%), compressive (76.7%), inflammatory (75.0%), papilledema-related (70.8%), ischemic (65.5%), and drug toxicity (63.4%).

The elderly age group was not the predominant group in any of the optic neuropathies, but was observed to contribute large proportions in drug toxicity (34.6%) and ischemic (32.8%) etiologies.

Table 1. Demographic Profile and Clinical Characteristics on Initial Presentation

	All	Post-Trauma	Ischemic (Non-arteritic)	Inflammatory	Compressive	Drug Toxicity (Ethambutol)	Papilledema-related	Congenital	Hereditary	Infiltrative	Nutritional Deficiency	Post-Radiation
N (%)	911	236 (25.9)	180 (19.8)	164 (18.0)	150 (16.4)	101 (11.1)	48 (5.3)	21 (2.3)	6 (0.7)	3 (0.3)	1 (0.1)	1 (0.1)
Gender												
Male, n (%)	558 (61.3)	205 (86.9)	116 (64.4)	54 (32.9)	77 (51.3)	63 (62.4)	24 (50.0)	11 (52.4)	6 (100.0)	1 (33.3)	1 (100.0)	0 (0.0)
Female, n(%)	353 (38.7)	31 (13.1)	64 (35.6)	110 (67.1)	73 (48.7)	38 (37.6)	24 (50.0)	10 (41.7)	0 (0.0)	2 (66.7)	0 (0.0)	1 (100.0)
Mean Age ± SD (yrs)	40.2 ± 18.0	30.1 ± 13.8	53.7 ± 13.0	33.0 ± 15.9	44.5 ± 16.0	53.1 ± 14.2	33.1 ± 17.1	24.3 ± 19.0	29.3 ± 13.9	37.0 ± 26.3	52.0 ± 0.0	48.0 ± 0.0
Pediatric, n(%)	115 (12.6)	39 (16.5)	3 (1.7)	33 (20.1)	11 (7.3)	2 (2.0)	13 (27.1)	12 (57.2)	1 (16.7)	1 (33.3)	0 (0.0)	0 (0.0)
Middle Age, n (%)	662 (72.7)	193 (81.8)	118 (65.5)	123 (75.0)	115 (76.7)	64 (63.4)	34 (70.8)	7 (33.3)	5 (83.3)	1 (33.3)	1 (100.0)	1 (100.0)
Elderly, n(%)	134 (14.7)	4 (1.7)	59 (32.8)	8 (4.9)	24 (16.0)	35 (34.6)	1 (2.1)	2 (9.5)	0 (0.0)	1 (33.3)	0 (0.0)	0 (0.0)
Laterality												
Unilateral, n(%)	486 (53.3)	226 (95.8)	103 (57.2)	84 (51.2)	59 (39.3)	0 (0.0)	0 (0.0)	12 (57.1)	0 (0.0)	2 (66.7)	0 (0.0)	0 (0.0)
Bilateral, n(%)	425 (46.7)	10 (4.2)	77 (42.8)	80 (48.8)	91 (60.7)	101 (100.0)	48 (100.0)	9 (42.9)	6 (100.0)	1 (33.3)	1 (100.0)	1 (100.0)
Presenting Ophthalmologic Symptom												
Decline in Visual Acuity, n (%)	855 (93.9)	233 (98.7)	155 (86.1)	161 (98.2)	130 (86.7)	101 (100.0)	46 (95.8)	18 (85.7)	6 (100.0)	3 (100.0)	1 (100.0)	1 (100.0)
Decline in Visual Field, n(%)	48 (5.3)	0 (0.0)	25 (13.9)	1 (0.6)	20 (13.3)	0 (0.0)	2 (4.2)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Decline in Color/Contrast, n(%)	5 (0.5)	3 (1.3)	0 (0.0)	2 (1.2)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Incidental Finding, n(%)	3 (0.3)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)	3 (14.3)	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Right Eye Findings												
Mean BCVA ± SD (LogMAR)	1.09 ± 1.10	1.04 ± 1.18	0.81 ± 1.02	1.27 ± 1.05	1.10 ± 1.22	1.34 ± 0.67	0.98 ± 1.08	1.38 ± 1.13	1.60 ± 0.66	1.35 ± 1.24	0.30 ± 0.0	3.00 ± 0.0
Optic Disc Finding, Right Eye												
Normal, n(%)	522 (58.7)	178 (75.4)	120 (66.7)	89 (54.3)	87 (58.0)	46 (45.5)	0 (0.0)	-	0 (0.0)	2 (66.7)	0 (0.0)	0 (0.0)
Swollen, n(%)	114 (12.8)	3 (1.3)	24 (13.3)	49 (29.9)	9 (6.0)	1 (1.0)	28 (58.3)	-	0 (0.0)	0 (0.0)	0 (0.0)	0 (0.0)
Atrophic, n(%)	254 (28.5)	55 (23.3)	36 (20.0)	26 (15.8)	54 (36.0)	54 (53.5)	20 (41.7)	-	6 (100.0)	1 (33.3)	1 (100.0)	1 (100.0)
Left Eye Findings												
Mean BCVA ± SD (LogMAR)	1.09 ± 1.09	1.03 ± 1.21	0.77 ± 0.99	1.21 ± 1.09	1.16 ± 1.13	1.46 ± 0.68	1.01 ± 1.07	1.27 ± 1.14	1.81 ± 0.68	1.10 ± 1.34	0.20 ± 0.0	2.70 ± 0.0
Optic Disc Finding, Left Eye												
Normal, n(%)	539 (60.6)	185 (78.4)	126 (70.0)	89 (54.3)	91 (60.7)	46 (45.5)	0 (0.0)	-	0 (0.0)	1 (33.3)	0 (0.0)	0 (0.0)
Swollen, n(%)	111 (12.5)	5 (2.1)	23 (12.8)	47 (28.7)	6 (4.0)	1 (1.0)	28 (58.3)	-	0 (0.0)	2 (66.7)	0 (0.0)	0 (0.0)
Atrophic, n(%)	240 (26.9)	46 (19.5)	31 (17.2)	28 (17.0)	53 (35.3)	54 (53.5)	20 (41.7)	-	6 (100.0)	0 (0.0)	1 (100.0)	1 (100.0)

SD - standard deviation; BCVA – best-corrected visual acuity

DISCUSSION

This study described the leading etiologies and clinical profile of Filipinos diagnosed with NGON. The top four etiologies of NGON identified in our cohort – trauma, ischemia, inflammation and compression – were consistent with those reported in a locally published study from 1979 (**Table 2**).[6] Both local studies employed a similar methodology – a cross-sectional retrospective chart review – which is commonly used in other regional and international studies investigating distribution and clinical characteristics of NGON.[3,6,8-17] Compared to these previous reports, our study included the largest population sample and covered the longest time period of NGON cases reviewed.

The demographic profile of our patients was also consistent with findings from both regional and international literature, which report the most commonly affected age group to be between 40 and 50 years old.[3,6, 8-17]

Our study demonstrated that trauma, ischemia, inflammation, and compression were the top etiologies for NGON in the study population. Our findings were consistent with previous studies as presented in **Table 2.**

In the pediatric age group, the top three causes of NGON were traumatic (39 cases; 33.9%), inflammatory (33 cases, 28.6%) and papilledema-related (13 cases, 11.3%). In the absence of significant head or ocular trauma, our findings underscore the importance of focused evaluation for inflammatory optic neuropathy (optic neuritis) and increased intracranial pressure, both of which often necessitate neuroimaging as part of the work-up.

Majority of our study cohort are patients in the middle age group. Excluding traumatic optic neuropathy (193 cases, 29.1%), the leading causes of NGON in this age group were inflammatory (123 cases, 18.5%), ischemic (118 cases, 17.8%), and papilledema-related (115 cases, 17.3%).

Table 2. Comparison of Etiologic and Demographic Profiles of Non-Glaucomatous Optic Neuropathy in Published Literature

Author		Reyes & Cullen[8]	Pedro-Egbe *et al.*[9]	Ogun & Adediran[10]	Chinta *et al.* (Pediatric study)[11]	Bajracharya *et al.*[12]	Dias *et al.*[3]	Mbekeani *et al.*[13]	Pandey *et al.*[14]	Ihesiulor *et al.*[15]	Jones *et al.* (Pediatric study)[16]	Shresta *et al.*[17]	Reyes-Noche & Fajardo[6]	Index Study
Year Published		2010	2011	2014	2014	2016	2017	2017	2018	2020	2020	2021	1979	2021
Site		Singapore	Nigeria	Nigeria	India	Nepal	Brazil	Saudi Arabia	Nepal	Nigeria	United States	Nepal	Philippines	Philippines
Study Design		Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study	Cross-sectional study
Time Period (months)		0.25	60	Unspecified	72	3	24	9	7	12	60	12	96	144
Sample size		19	99	159	324	44	68	244	117	45	143	62	134	911
Average Age at Presentation (years)		50	40	39	7	53.6	44.6	27	45	57	5.9	40.6	Unspecified	40.2
Top Causes of NGON Ranked from Most Frequent to Least Frequent	1	Ischemic	Idiopathic	Idiopathic	Ischemia	Trauma	Ischemic	Compressive	Ischemic	Retinopathy-related	Retinopathy-related	Trauma	Inflammation	Trauma
	2	Hereditary	Inflammation	Compressive	Idiopathic	Idiopathic	Compressive	Inflammation	Papilledema-related	Nutritional Deficiency	Hereditary		Compressive	Ischemic
	3	Inflammation	Nutritional	Inflammation	Papilledema-related	Ischemic	Hereditary	Ischemic	Trauma	Idiopathic	Ischemic		Trauma	Inflammation
	4	Idiopathic		Papilledema-related	Compressive	Compressive		Hereditary	Inflammation	Inflammation	Inflammation		Ischemic	Compressive
	5	Trauma		Ischemic	Inflammation	Papilledema-related		Idiopathic	Compressive	Ischemic	Trauma		Retinopathy-related	Toxic
	6	Toxic		Trauma	Hereditary	Inflammation		Metabolic	Hereditary		Compressive		Congenital	Papilledema-related
	7			Toxic		Retinopathy-related		Trauma	Toxic		Toxic		Papilledema-related	Hereditary
	8					Toxic		Toxic	Atrophy				Idiopathic	Congenital
	9								Pituitary apoplexy					Infiltrative
	10													Nutritional Deficiency
	11													Post-radiation

NGON - non-glaucomatous optic neuropathy

The high proportion of traumatic cases in both age groups may reflect a referral bias, as the study site is a designated trauma center located in an urban area. The occurrence of vision loss secondary to preventable trauma in the middle age group – an economically productive demographic – highlights the need for targeted public health initiatives and policy measures aimed at injury prevention and vision preservation.[18-22]

Among the elderly age group, the top two causes of NGON were ischemia (59 cases, 44.0%) and drug toxicity (35 cases, 26.1%). The predominance of ischemic etiologies aligns with the current epidemiologic trend showing increased prevalence of lifestyle-related diseases, such as hypertension, dyslipidemia, and diabetes mellitus, occurring in the older age group.[23] In addition, this finding also underscores the importance of conducting a thorough review of the patient's medication history to identify potential agents that may cause toxic optic neuropathy. Ethambutol, a first-line antibiotic for tuberculosis,[24] was identified as the causative agent in all of the cases of toxic optic neuropathy in this study.

The study found that most etiologies of NGON demonstrated a normal-appearing optic disc at presentation (58.7-60.6%). Optic atrophy was seen in 26.9-28.5% of our cases. Optic atrophy, however, is a non-specific clinical sign that is the common end result of any insult on the optic nerve.[1,2] Certain features of the optic disc, such as the presence of disc-at-risk configuration in the fellow eye in a patient suspected with non-arteritic anterior ischemic optic neuropathy,[23] or the well-documented features of the various congenital nerve anomalies can facilitate accurate diagnosis.[1,25] However, such distinct diagnostic clues are often lacking in other etiologic categories, making their identification more challenging. This emphasizes the role of thorough clinical history, laboratory examination including appropriate neuroimaging, and referral to a neuro-ophthalmologist, to reliably determine the etiology of NGON.

This study has several limitations owing to its retrospective and cross-sectional design. As a result, we were unable to report the final visual acuity or document changes in visual function over time and in response to treatment. The absence of consistent ancillary testing – such as visual fields, optical coherence tomography, neuroimaging, and laboratory testing – further limited the clinical characterization in many cases. The single-center design of the study also limited generalization of our findings. We recommend future prospective cohort studies to enable longitudinal assessment of visual outcomes and provide a more comprehensive understanding of the clinical profile of NGON.

Additionally, focused investigations on specific etiologies of NGON are encouraged to inform targeted diagnostic and management strategies.

In conclusion, the study provided an update on the current trend of non-glaucomatous optic neuropathies in an urban population in the Philippines. Traumatic optic neuropathy emerged as the leading cause of vision loss from NGON in both pediatric and middle age groups. In the absence of trauma, further investigations should focus on inflammatory and papilledema-related etiologies. Among the elderly, ischemia and drug-induced toxic optic neuropathies were the most common, with thorough history-taking being crucial for identifying medication-related causes.

REFERENCES

1. Francisco P, Lorena GV, Nestor AV, *et al.* Diagnosis and Approach of Optic Neuritis. *J Neurol Neurophysiol.* 2015;6(6):1-13.
2. Gupta PK, Asrani S, Freedman SF, *et al.* Differentiating glaucomatous from non-glaucomatous optic nerve cupping by optical coherence tomography. *Open Neurol J.* 2011;5:1-7.
3. Dias DT, Ushida M, Battistella R, *et al.* Neurophthalmological conditions mimicking glaucomatous optic neuropathy: analysis of the most common causes of misdiagnosis. *BMC Ophthalmol.* 2017;17(1):2.
4. Lin Y, Jiang B, Cai Y, *et al.* The Global Burden of Glaucoma: Findings from the Global Burden of Disease 2019 Study and Predictions by Bayesian Age-Period-Cohort Analysis. *J Clin Med.* 2023;12(5):1828.
5. Cubillan LDP, Olivar-Santos EO. Third National Survey on Blindness. *Philipp J Ophthalmol.* 2005;30(3):100-114.
6. Reyes-Noche R, Fajardo RV. Causes of optic atrophy among Filipinos. *Philipp J Ophthalmol.* 1979;11(4):109-111.
7. Schulze-Bonsel K, Feltgen N, Burau H, *et al.* Visual Acuities "Hand Motion" and "Counting Fingers" Can Be Quantified with the Freiburg Visual Acuity Test. *Invest Ophthal Vis Sci.* 2006;47(3):1236.
8. Reyes KB, Cullen JF. A week in neuro-ophthalmology: the Singapore scene. *Philipp J Ophthal.* 2010;35(2):70-72.
9. Pedro-Egbe CN, Cookey SA, Awoyesuku EA, Ani N. Nonglaucomatous optic neuropathies in Port Harcourt. *Clin Ophthalmol.* 2011;5:1447-1450.
10. Ogun OA, Adediran OA. Non-glaucomatous optic neuropathy in Ibadan: extrapolations to healthcare funding in Nigeria. *Ann Ib Postgrad Med.* 2014;12(2):103-108.
11. Chinta S, Wallang BS, Sachdeva V, *et al.* Etiology and clinical profile of childhood optic nerve atrophy at a tertiary eye care center in South India. *Indian J Ophthal.* 2014;62(10): 1003-1007.
12. Bajracharya K, Gautam P, Yadav SK, *et al.* Epidemiology and causes of optic atrophy in the general outpatient department of Lumbini Eye Institute. *Journal of Universal College of Medical Sciences.* 2016; 3: 26-29.
13. Mbekeani JN, Fattah MA, Poulsen DM, *et al.* Etiology of optic atrophy: a prospective observational study from Saudi Arabia. *Ann Saudi Med.* 2017;37(3):232-239.
14. Pandey A, Shrestha S, Malla Bhari A, *et al.* A hospital based cross-sectional study to assess various types of non-glaucomatous optic neuropathies. *New Frontiers in Ophthalmology.* 2018;4(6).
15. Ihesiulor, C, Chimchetam, J, Udo, U. Prevalence of Non-glaucomatous Optic Atrophy in Ehime Mbano, Nigeria. *Asian Journal of Research and Reports in Ophthalmology.* 2020; 3(1):1-9.
16. Jones R, Al-Hayouti H, Oladiwura D, *et al.* Optic atrophy in children: Current causes and diagnostic approach. *Eur J Ophthalmol.* 2020;30(6):1499-1505.
17. Shrestha P, Sitaula S, Sharma AK, Joshi P. Clinical Assessment and Etiological Evaluation of Optic Nerve Atrophy. *Nepal J Ophthalmol.* 2021;13(25):73-81.
18. Larona AJ, Pe-Yan MR. Visual Profile of Motorcycle-related Ocular Trauma in a Tertiary Hospital. *Philipp J Ophthalmol.* 2012;37:111-116.
19. Macenas J, Tan A. Epidemiology of Eyelid Trauma Among Filipino Patients in a Tertiary Hospital. *Philipp J Ophthalmol.* 2021;46(2):103-108.
20. Esteban D, Claudio K, Arcinue C. Prognostic Value of a Classification and Regression Tree Model in Patients with Open-Globe Injuries. *Philipp J Ophthalmol.* 2024;49(1):28-32.
21. Artiaga J, Lim Bon Siong R. Quality of Life After Ocular Trauma: A Prospective, Longitudinal, Questionnaire-Based Study in a Tertiary Hospital in the Philippines. *Philipp J Ophthalmol.* 2019;44(2):59-67.
22. Ferrer K, Santiago D. Intimate Partner Violence (IPV) - Associated Ophthalmologic Injuries among Women: A Systematic Review. *Philipp J Ophthalmol.* 50(1):50-63.
23. Hayreh SS. Ischemic optic neuropathy. *Prog Retin Eye Res.* 2009;28(1):34-62.
24. Department of Health. *National Tuberculosis Control Program Manual.* 5th ed.; 2014: https://www.philhealth.gov.ph/partners/providers/pdf/NTCP_MoP2014.pdf (accessed July 15, 2025).
25. Levin AV. Congenital eye anomalies. *Pediatr Clin North Am.* 2003;50(1):55-76.