Janaki Medical College Journal of Medical Sciences (2023); Vol.11 (2):18-27

JMCJMS

Original Article

Clinical Etiology of Optic Neuropathy patients visiting BPKLCOS, Institute of Medicine, Nepal

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ABSTRACT

Background & Objective: The clinical etiology of Optic neuropathy is vast and may be associated with life threatening conditions which might demand initiation of treatment. Therefore, the aim of the study was to evaluate clinical etiology among patients visiting ophthalmology OPD of Institute of medicine, Nepal.

Material and Methods: This is a descriptive study conducted at B.P. Koirala Lions Centre for Ophthalmic Studies, Institute of Medicine, Nepal. All cases of optic neuropathy who presented to outpatient department (OPD), indoor patients from 7th July 2017-6th July 2018 were included in the study. A detailed clinical history was obtained which was followed by ophthalmic examination and relevant investigation. In addition, assessment of visual acuity, color vision, contrast sensitivity, visual field were done. Data was analyzed with SPSS version 24.

Results: A total of 86 eyes of 66 patients with optic neuropathy meeting all the inclusion criteria during the study period were enrolled with 20 patients having bilateral eye involvement and 46 having unilateral eye involvement accounting for a total of 86 eyes with optic neuropathy. The mean age of the affected population was 39.12±13.57 years with male to female ratio of 1:1.1. The most common etiology for optic neuropathy was optic neuritis (n=40 patients, 60.6%). Diminution of vision was the most common presenting complaints. Best corrected visual acuity was 6/24 –6/60 in 39.5% cases (n=34). Majority of the eyes had presence of RAPD (n=53 eyes, 61.7%) The most common color vision defect was nonspecific defect (n=41eyes, 47.7%).There was reduced contrast in 55.8% of the affected eyes in cases of optic neuropathy. Majority of affected eyes had blurred disc margin (n=39 eyes, 45.3%) followed by hyperemic disc and pale disc. Majority of the patients could not perform visual field due to low vision but those who could perform had enlarged blind spot.

Conclusion: The most frequent etiological factor for optic neuropathy was optic neuritis. In cases of optic neuropathy, the main complaint of the majority of patients was a dimunition in vision. The majority of cases of optic neuropathy

involved abnormalities in both colour vision and contrast sensitivity. The most typical visual field pattern in eyes with optic neuropathy was an enlarged blind spot. Optic neuritis is the most common cause of optic neuropathy, followed by traumatic optic neuropathy, and toxic metabolic optic neuropathy.

Keywords: Blurred disc margin, Optic neuropathy, Optic neuritis, Traumatic optic neuropathy, Temporal pallor

INTRODUCTION

Optic neuropathy is one of the most common causes of visual loss that ophthalmologists face. Inflammatory or non-inflammatory causes of optic nerve injury are mentioned [1]. Some of the most prevalent conditions that result in vision loss are ocular neuropathies, which can manifest on their own or in conjunction with neurological or systemic symptoms and indications. Neurologists frequently identify and treat various visual neuropathies, particularly inflammatory optic neuropathies, because they are linked to neurological illnesses. The different mechanisms underlying optic neuropathies often appear in the diseased eye with diminished visual acuity, altered colour vision, and an aberrant visual field. The mode of start of visual loss, the presence of pain with eye movements, the visual acuity, and the retention of colour vision are all crucial factors in the diagnosis, which is based on the clinical history and clinical examination [2].

Visual loss in the affected eye is a sign of optic neuropathy, which is validated by aspects of the history and examination that set it apart from retinal and ocular pathology. When central vision is impacted, the patient will often notice the onset of visual loss early. Patients with more subtle peripheral sight loss may not be aware of the impairment and delay in seeking treatment. Furthermore, because binocular acuity, which is more important practically, is unaffected, visual loss in 1 eye could go unreported. Important hints regarding the likely aetiology of an optic neuropathy can be found in the rate of visual loss. An optic neuropathy is confirmed by key findings of an in-depth examination, which also separate it from other causes of vision loss. An optic neuropathy is characterized by aberrant optic disc appearance, relative afferent pupillary deficit (RAPD), and impaired colour vision. When these signs of monocular vision loss are lacking, an optic neuropathy diagnosis should be regarded as doubtful and other possible causes should be investigated. Central visual acuity is frequently decreased in ocular neuropathies, although this test is insensitive and does not accurately reflect the degree of optic nerve damage [3,4].

By measuring acuity with the patient wearing corrective glasses or using a 2-mm pinhole that significantly lessens optical distortion, refractive errors should be ruled out as the source of acuity loss. The Snellen eye chart is frequently used for visual acuity testing. The patient's minimal angle of resolution determines whether they can correctly distinguish a letter. The size of the 20/20 Snellen "E" is based on the average human range for high-contrast central vision, which is between 30 and 1 minute of arc [4]. Low-contrast acuity, which allows for the discovery of minor defects that spare high-contrast acuity, is another sensitive indicator of optic nerve function [5,6].

Ophthalmologists frequently face optic neuropathy as the cause of visual loss. Based

on clinical evidence, the diagnosis is made. Frequently, the history suggests a potential cause for the optic neuropathy. When addressing this condition's endpoint in terms of the patient's vision, the underlying etiology is frequently disregarded.

Optic neuropathy might be identified at an early stage using all current diagnostic techniques at a tertiary eye centre, and visual function could be tracked over time. Vision loss could be avoided with prompt treatment of the underlying problem. We can better understand how the effects of optic neuropathy on the patient's remaining function by using optical coherence as a research instrument to measure the loss of nerve fibre layer thickness. Reliable data are not consistently available. Therefore, this study has attempted to find out the underlying etiology and clinical profile of optic neuropathy presenting to BPKLCOS, IOM, Nepal.

MATERIAL AND METHODS

Study Site and Design

A hospital based, descriptive study conducted from July 2017- July 2018 among patients in outpatient department at B. P. Koirala Lions Centre for Ophthalmic Studies (BPKLCOS), Institute of Medicine (IOM), Tribhuvan University (TU), Kathmandu, Nepal and in patients at Tribhuvan University Teaching Hospital (TUTH), IOM, TU, Kathmandu, Nepal.

Study Population

A total of 86 eyes of 66 patients with optic neuropathy meeting all the inclusion criteria during the study period were enrolled.

Inclusion and Exclusion Criteria

All cases with Optic neuropathy presenting to BPKLCOS and TUTH with age above 15 years were included. Glaucomatous optic atrophy, Retinal detachment, massive vitreous hemorrhage, Open Globe injury, Dense Cataract, Intraocular surgery and conditions in which optic disc could not be visualized due to media opacity. Pediatrics age group below 15 years and patients not meeting the study protocol were excluded.

Data Collection, Patient examination and Investigations

A semi-structure questionnaire was developed for the data collection procedure. After taking formal verbal and written consent, all the patients diagnosed with optic neuropathy underwent detailed evaluation. Detail history was taken from the patient and /or patient's relatives for the Profile of the patients including age and gender, presenting symptoms, past illness, associated risk factors, any form of treatment received, personal habits like smoking and drinking and family history were taken.

Ocular examination

Visual acuity was assessed by internally illuminated Snellen vision box with multiple optotype and E chart (for illiterates) at a distance of 6 meters, visual acuity with best refractive correction, extraocular motility, lids and adnexa examination with a torch light examination of pupillary light reflex in dark room for better assessment of RAPD.

Anterior segment examination with the help of Haag-Streit 900 slit lamp bio microscope in appropriate magnification and illumination, fundus examination after pupillary dilatation, with Heine Beta 200 direct ophthalmoscopy, Haag-Streit 900 slit lamp with Volk +90 diopter lens and binocular indirect ophthalmoscope with Volk +20 diopter lens. Intraocular Pressure was also taken.

Complete neurological examination

Neurological examinations were carried out by consultant neurologists in neuro ophthalmology clinic at BPKLCOS, IOM, Kathmandu, Nepal. After completion of neurological examination patient were referred to other sub specialty clinics where necessary.

Investigations

Color vision was tested with Farnsworth Munsell Dichotomous D-15 test. Contrast sensitivity was recorded using Pelli-Robson contrast sensitivity chart at 1m distance. Visual field test was done using Goldman Perimeter (Takagi Company). Visual evoked potential (VEP) were also performed. For hematological investigations, Complete blood count, random blood sugar, Erythrocyte sedimentation rate (ESR), VDRL test, TPHA, serum calcium, Serum ACE, Vitamin B12 in relevant cases were done. All the above mentioned data were recorded in a proforma.

Statistical Analysis

The data were computed in MS Excel and Statistical analysis was performed on SPSS 24.

Ethical Consideration

The study protocol was approved from the Institutional Review Board (IRB) at Institute of Medicine (Ref: 470(6-11-E)^2/073/074 and consent was given for the study. After obtaining informed consent from the patients, all patients meeting the inclusion criteria were included in the study.

RESULTS

The total of 66 patients from the eye OPD and those admitted in eye ward at BPKLOS who met the inclusion criteria were included in the study. The included patients were subjected to all the relevant investigation for the diagnosis of the underlying cause. The mean age for the cases with optic neuropathy was 39.12±13.57 years with minimum age of 17 years and maximum age of 81 years.

Majority (33.3%) of cases belonged to the age group of 26-35 years (n=22) followed by 30.3% in age group of 36-45 years (n=20). There was only 1 case above 75 years of age. Among 66 patient, 51.5% were male (n=32) and females accounted for 51.4% of cases (n=34). The male to female ratio was found to be 1:1.1. Majority of cases (68.2%, n=45) were patient from hilly region while (28.7%, n=19) were from Terai region and (3%, n=3) of cases were from mountain region as depicted in table 1.

Table 1: Age and gender Distribution of patients with optic neuropathy (n=66 patients)

Age Group (In Years)	**No (%)**
15-25	8(12.1)
26-35	22(33.3)
36-45	20(30.3)
46-55	7(10.6)
56-65	5(7.6)
66-75	3(4.5)
76-85	1(1.5)
Gender	
Male	32(48.5)
Female	34(51.5)
Geographical distribution	
Mountain region	2(3.0)
Hilly region	45(68.2)
Terai region	19(28.7)

Majority of the patients had unilateral ocular involvement (69.7%, n=46 eyes).Among those cases left eye involvement was (39.4%, n=26) and right eye involvement was (30.3%,

n=20).Bilateral eye involvement was found to be (30.3%, n=20).

Table 2: Laterality of eyes of patient with optic neuropathy (n=66 patients)

Affected Eye	No (%)
Both eyes	20(30.3)
Left eyes	26(39.4)
Right eyes	20(30.3)
Total	66(100)

Table 3 presents the most common presenting complaint of the patients was isolated DOV only, (50%, n=33 patients). Ocular pain only was the complaint in (1.5%,n= 1). Likewise DOV along with headache was present in (21.3%, n=14) patients, DOV and ocular pain in (16.7%,n=11) patients, DOV, headache and ocular pain in (7.5%,n=5) patients and headache with ocular pain in (1.5%,n=1) patients. DOV, ocular pain, headache and diplopia was found to be in (1.5%, n=1)

A total of 86 eyes of 66 patients with optic neuropathy were evaluated. Majority (39.5%, n=34) of patient's eyes had best corrected visual acuity between 6/24-6/60.While visual acuity between <3/60-perception of light was found in (23.2%, n=20), (19.8%, n=17) had visual acuity between 5/60-3/60, (14%, n=12) between 6/6 and 6/18, and (3.5%, n=3) had no light perception. Among the 86 eyes of 66 patients that were diagnosed as having optic neuropathy, the pupillary reaction in majority (61.7%, n=53) of eyes had RAPD while (38.3%, n=33) eyes had no RAPD. The majority of the affected eyes had non-specific color vision defect (47.7%, n=41).Color vision test could not be performed in (32.5 %, n=28), while (19.8 %, n=17) had normal color vision. The contrast sensitivity was reduced in majority (55.8%) of the affected eyes (n=48 eyes).It was normal in 11.7 % of the affected eyes (n=10), It could not be done in 32.5% of the affected eyes (n=28). Among the 86 eyes with optic neuropathy, (55 eyes, 63.9%) had disc edema. Pale disc and temporal pallor were found in (n=14 eyes, 16.3%), (n=7 eyes, 8.1%) respectively. Normal disc finding was there in (n=10 eyes, 11.6%). Prolongation of P100 latencies as most common VEP finding among optic neuropathy patients (47.7%, n=41) of the diseased eyes followed by reduced amplitude and prolonged p100 latencies (31.4%, n=27) as depicted in Table 4.

Table 3: Ocular complains of the patients of optic neuropathy (n=66 patients)

Ocular Complains	No (%)
Diminution of vision(DOV) only	33(50)
Ocular Pain only	1(1.5)
Headache only	0
Diplopia only	0
DOV and headache	14(21.3)
DOV and ocular pain	11(16.7)
DOV and diplopia	0
Headache and pain	1(1.5)
DOV and pain and headache	5(7.5)
DOV and pain and headache and diplopia	1(1.5)
Total	66(100)

Table 4: Ocular Characteristics in Optical neuropathy (n=86 eyes)

Best Corrected Visual Acuity	**N (%)**
6/6 – 6/18	12(14)
6/24 – 6/60	34(39.5)
5/60 – 3/60	17(19.8)
<3/60 – PL	20(23.2)
No Light Perception	3(3.5)
Total	86(100)
Pupillary Reaction	
RAPD absent	33(38.3)
RAPD present	53(61.7)
Total	86(100)
Color Vision test	
Could not be done	28(32.5)
Non Specific Defect	41(47.7)
Normal	17(19.8)
Total	86(100)
Contrast sensitivity test	
Normal	10(11.7)
Reduced	48(55.8)
Could not be done	28(32.5)
Total	86(100)
Disc Findings	
Disc edema	55(63.9)
Pale disc	14(16.3)
Temporal Pallor	7(8.1)
Normal	10(11.7)
Total	86(100)
Goldman Visual Field Test	
Blind spot enlargement	14(16.2)
Normal	8(9.3)
Central scotoma	12(14.2)
Could not perform	29(33.6)
Generalized constriction of all isopters	10(11.5)
Centrocecal scotoma	3(3.5)
Paracentral scotoma	6(7.0)
Inferotemporal visual field defect	1(1.2)
Inferior altitudinal defect	1(1.2)
Superior altitudinal defect	2(2.3)
Total	86(100)
Visual evoked potential (VEP)	
Normal	18(20.9)
Prolonged P100 latencies	41(47.7)
Reduced amplitude and prolonged p100 latencies	27(31.4)
Total	86(100)

Among the 66 patient included in the study, the most common etiology of optic neuropathy was found to be Optic Neuritis (n=42, 63.6%) among which multiple sclerosis was found in (n=2, 3%) patients which was followed by traumatic optic neuropathy (n=12, 18.2%).Toxic and Metabolic optic neuropathy was found to be in 18.2% (n=12) as shown in Table 5.

Table 5: Etiology of optic neuropathy (n=66)

Etiology	N (%)
Optic Neuritis	42(63.6)
Traumatic Optic neuropathy	12(18.2)
Toxic and Metabolic optic neuropathy	12(18.2)
Total	66(100)

DISCUSSION

Our study revealed that the mean age of the cases with optic neuropathy was 39.12±13.57 in line with study carried out by Verma et al. [7]. This study showed that optic neuropathy was most common in the age group 26-35 years (33.3%) followed by 36-45 years (30.3%). Sixty-three percent of the patients were in age group 26-45 years which was similar to study done by Bihari and Pandey [8] where most of patients (65%) belonged to age group 20-50 years which is almost parallel to our findings. The mean age of our patients (38.07±1.37) was similar to that reported in Wang et al [9], and whereas in Taiwanese study patients were younger than those reported from oriental countries [9-11].This might due similarity in the geographical region in Nepal and India. In the present study out of 66 patients 51.5 %(n=34) were female and 48.5 %(n=32) were male. The male to female ratio was 1:1.1 which is similar to study done in previous studies [7, 8]. In this study majority of cases (68.2%, n=45) were from hilly region and mostly from Kathmandu, Bhaktpur and Lalitpur as the hospital is located in Kathmandu valley. Our study had majority of the patients with unilateral ocular involvement (69.7%, n=46) while bilateral eye involvement was found to be in 30.3% cases (n=20) which contrasts the findings in the study done by Verma et al. [7], where only 36% patients had unilateral involvement.

Isolated DOV alone was the patients' most frequent presenting complaint (50%, n=33 patients). Only ocular pain (1.5%, n=1) was the complaint. Similarly, DOV and headache were both present in (21.3%, n=14) patients, as were (16.7%, n=11) patients with DOV and ocular pain, (7.5%, n=5) patients with DOV, headache, and ocular pain, and (1.5%, n=1) patients with DOV and ocular pain. 1.5% of the patients had DOV, ocular pain, headaches, and diplopia. But, in contrast to our study conducted by Rajkarnikar et al. [12] 46.6% of patients reported painful ocular movement, which is almost identical to the results of studies conducted in China by Zhang et al. [11] (42.9%) and in the far- and mid-western regions of Nepal by Thapa et al. (40%) [13] respectively. In another study, Godar et al. [14] from Nepal and Saxena et al. [15] from India both discovered higher (58%) and elevated (66%) occurrences of painful ocular movement. However, significantly fewer cases of painful ocular movement were reported in studies conducted in Eastern Nepal [16], Chandigarh [17], and Japan [18].

The majority of patient eyes in our study (39.5%, n=34) exhibited greatest corrected visual acuity between 6/24-6/60.While (23.2%, n=20) of the population had visual acuity between 3/60 and 3/60, (19.8%, n=17) had visual acuity between 5/60 and 3/60, (14%, n=12) between 6/6 and 6/18, and (3.5%, n=3) had no perception of light. In a comparable study, Rajkarniker et al. [12] found that although in the studies by Thapa et al. [13] and Das et al. [16] (23%) and Godar et al. [14] (8%) individuals presented with no perception of light, 20% of patients did so in Rajkarniker's study [12].

The majority of the affected eyes had no specific color vision defect (47.7%, n=41).

While (19.8%, n=17) had normal colour vision, the colour vision test was unable to be completed by (32.5%, n=28). The majority (55.8%) of the afflicted eyes (n=48 eyes) had decreased contrast sensitivity. It could not be done in 32.5% of the affected eyes (n=28), although it was normal in 11.7% of the affected eyes (n=10). 94% of the eyes in a study conducted by Rajkarniker et al.,[12] had total colour blindness, and the remaining 6% had red-green deficiency. Total colour blindness was more prevalent, according to Shrestha et al. [19], although Godar et al. [14] study revealed that non-specific colour vision defects were more prevalent.

The term "optic disc edema" refers to all types of optic disc swelling; "papillitis" refers to inflammation of the optic disc; "papilledema" refers to disc swelling brought on by elevated intracranial pressure; and "pseudo papilledema" refers to a physiological variation of the optic disc that mimics some of the symptoms of papilledema. When the posterior ciliary arteries are blocked, the optic nerve head suffers an infarction, which causes ischemic optic neuropathy [20]. The majority (61.7%, n=53) of the 86 eyes of the 66 patients whose optic neuropathy was diagnosed had RAPD, while only (38.3%, n=33) of the eyes did not. 55 eyes (63.9%) of the 86 eyes with optic neuropathy showed disc edema. The prevalence of pale disc and temporal pallor was (n=14 eyes, 16.3%), and (n=7 eyes, 8.1%), respectively. There were normal disc findings in (n=10 eyes, 11.6%). However, the neuro-ophthalmology department at the Tilganga Institute of Ophthalmology reported patients with disc edema, with papilledema identified in 35 of these patients as the most frequent cause of disc swelling. [21].

VEP is a well-known non-invasive tool for investigating the function of the visual system.11 Decreased amplitude and prolonged latency of VEP recording is believed to reflect axonal damage and demyelination in the optic nerve [22]. The most frequent VEP observation in individuals with optic neuropathy was extended P100 latencies (47.7%, n=41), which was followed by reduced amplitude and prolonged p100 latencies (31.4%, n=27). In contrast to the findings of the study conducted by Verma et al. [7], where only 36% of patients had unilateral involvement, the majority of the patients in our study (69.7%, n=46) had bilateral eye involvement in 30.3% of instances (n=20). Similar to the research from Nepal [19], China [11], and Japan [18], unilateral optic neuritis was more common than bilateral. While another centre in Kathmandu revealed an equal number of unilateral and bilateral cases, a study from Eastern Nepal [19].

Among the 66 patients included in this study, optic neuritis (n=42, 63.6%) ad traumatic optic neuropathy (n=12, 18.2%) were shown to be the most common etiologies of optic neuropathy. 18.2% of cases (n=12) of toxic and metabolic optic neuropathy were identified. So far to our knowledge, research and scientific literatures on optic neuropathy are still scarce in Nepal. Since this study was conducted at a hospital, the actual magnitude of optic neuropathy all over the Nepal might be understated. Finding the patients with optic neuropathy requires large scale studies, a longer and more thorough follow-up.

CONCLUSION

The study concludes that females were more likely than males to have unilateral involvement in optic neuropathy. The most

frequent complaint of the majority of patients with optic neuropathy was a reduction in vision, a colour vision defect, and a decreased sensitivity to contrast. Optic neuritis was reported to be the most frequent cause of optic neuropathy, followed by traumatic optic neuropathy and toxic and metabolic optic neuropathy.

ACKNOWLEDGEMENT

We are grateful to all the participants and BPKLCOS, IOM, Kathmandu for their support during this study.

Conflict of interest

None declared

Funding

None

Author's Contribution: *Research Concept and design, Literature review, Data collection, statistical analysis and interpretation-**AKY;** Supervision and revision of the 1st draft of manuscript, decisive approval of article of final draft-**SS;** Literature review; revision of 2nd draft of manuscript; reference management and final revision –**SNJ, AKS,AKY.** All the authors read the final draft of manuscript and agreed to publish.*

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