



Original Article

Clinical Assessment and Etiological Evaluation of Optic Nerve Atrophy

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Abstract

Introduction: Optic atrophy results from the disease process that cause irreversible damage to the ganglion cells and the anterior visual pathway, but may also result from posterior visual pathway involvement. The etiology causing this condition is vast and regardless of underlying cause it carries bad visual prognosis and at times may be life threatening. The study aims to assess patients with optic nerve atrophy presenting to B.P. Koirala lions centre for ophthalmic studies and identify the underlying etiology.

Materials and methods: This is a descriptive study conducted at B.P. Koirala Lions Centre for Ophthalmic studies. All cases of optic atrophy who presented to our outpatient department from March 2016 to March 2017 were included in the study. In addition to detailed evaluation, assessment of visual acuity, color vision, contrast sensitivity and visual field were done if feasible. Other relevant investigations were conducted to establish the underlying etiological cause.

Results: A total of 62 patients were included in the study, with 35 patients having bilateral disease and 27 having unilateral disease accounting for 97 eyes with optic atrophy. The mean age of the affected was 40.63 ± 17.36 years with male to female ratio of 1.2:1. The most common etiology for optic atrophy was traumatic neuropathy (n=16, 25.8%). Majority of eyes had pale disc (n=70, 72.2%) and the rest had temporal pallor (n=27, 27.8%).

Conclusion: Traumatic optic neuropathy was the most common etiological cause of optic nerve atrophy.

Key words: Optic Atrophy, Anterior visual pathway, Traumatic, Pale disc, Temporal pallor

Introduction

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Optic atrophy is a pathologic generalization of the appearance due to degeneration of axons in the anterior visual system that is clinically discernible and may result from injury to the optic nerve head, or injury to retinal ganglion cell due to anterograde degeneration or due to injury to the posterior optic nerve, optic chiasm or optic tract because of retrograde degeneration (Albert et al., 2008; Miller et al., 2005).

On ophthalmoscopic evaluation, there may be diffuse or sectoral pallor of the optic disc. In addition there is a reduction of the number of small vessels in the margin of the optic disc in cases of diffuse optic atrophy (Kestenbaum, 1946). Thinning of the nerve fiber layer around the optic disc is difficult to recognize in early stages while in advanced stages there is decreased opacity of the arcuate fiber bundles with diffuse thinning of the nerve fiber layer and the appearance becomes evident when large numbers of axon undergoes degeneration (Miller et al., 2005).

There are four types of optic nerve atrophy; primary optic atrophy results from the injury of the axon of the optic nerve (Albert et al., 2008). The common causes are optic neuritis and compressive lesions of the optic nerve including orbital, intracranial or chiasmal tumors (Walsh, 1969). Secondary optic atrophy occurs as a consequence of severe disc oedema. It occurs commonly following severe papilledema, long standing severe orbital inflammation, severe uveitis (Albert et al., 2008). Optic atrophy caused by the disease of the inner retina or its blood supply is termed as consecutive optic atrophy. Glaucomatous Optic Atrophy is generally seen in cases of advanced glaucoma.

Any etiology resulting in any form of optic neuropathy may eventually lead to optic nerve atrophy. The etiological causes of optic atrophy are different in different age groups. The causes of optic atrophy in adults could result from

different forms of optic neuropathies (Golnik, 2010). Therefore the causes of optic nerve atrophy can be classified as shown in Table 1.

Optic nerve atrophy, rather than being a disease in itself, is manifestation of another potentially vision and/ or life threatening condition, which demands prompt intervention, but underlying etiology is often overlooked. Therefore, this study has attempted to find out the underlying etiology of optic nerve atrophy presenting to our center. In addition we also aimed to compare the various clinical characteristics among the patients with optic atrophy.

Material and methods

The ethical approval was taken from the Institutional Review Board (IRB) at Institute of Medicine, Tribhuvan University and adhering to the tenets of Helsinki a hospital based cross sectional study was conducted from 5th March 2016 to 4th March 2017 in neuro ophthalmology clinic of B.P. Koirala Lions Centre for Ophthalmic Studies (BPKLCOS), Department of Ophthalmology and Inpatient Department of Tribhuvan University Teaching Hospital (TUTH). Informed written consent was obtained from all the patients. Those patients below 16 years of age, glaucomatous optic atrophy, whose optic disc could not be visualized due to media opacity and not willing to give consent; were excluded.

Optic atrophy was diagnosed based on the appearance of diffuse or sectoral pallor of the optic disc associated with reduction of the number of small vessel in the margin of the optic disc (Kestenbaum, 1946), narrowed or attenuated retinal arterioles (Papastathopoulos and Jonas, 1995; Rader et al., 1994) and significant loss of visual acuity, colour vision and visual field with little or mild cupping (Trobe et al., 1980).

A detailed history was obtained from all the diagnosed cases with special emphasis on past illness, history of trauma, associated risk

factors, neurological or systemic complaints, use of medication, personal habits and family history. Ophthalmic evaluation included best corrected visual acuity (BCVA), extraocular motility assessment, examination of direct and consensual pupillary light reflex, anterior segment evaluation under slit lamp bio microscope and fundus evaluation after pupillary dilatation with direct ophthalmoscope, slit lamp with +90D lens and binocular indirect ophthalmoscope with +20D lens. Examination was supplemented with investigations like color vision test (Farnsworth Munsell Dichotomous D-15 test), contrast sensitivity (Pelli-Robson contrast sensitivity chart), Visual Field (Takagi Goldmann Perimeter). Fundus fluorescein angiography (FFA) and Visual evoked potential (VEP) were performed if required.

All the patients underwent complete neurological examination by consultant neurologist and were referred to other subspecialty where necessary. In addition further investigation like hematology that included but not limited to complete blood count, Erythrocyte sedimentation rate, serum electrolytes, serum calcium, and vitamin B12 and radiological was done when necessary. Finally clinical photography of fundus and proforma were filled up for record keeping. The data was finally analyzed using SPSS software version 23 with appropriate statistical tools like percentage, mean, median, standard deviation.

Results

A total of 97 eyes of 62 patients with 54.8% male and 45.2% female were included in the study. The mean age was 40.63 ± 17.36 years (16 to 86 year). Table 2 shows the etiological diagnosis of optic nerve atrophy in our study, where the most common etiology was found to be traumatic, either as traumatic optic neuropathy or surgical trauma in cases who had undergone surgery for mass occupying lesion (n=16, 25.8%). Table 3 shows the

gender distribution, age group, laterality and complaints of the patients, BCVA, pupillary reaction, color vision defect (CVD) and visual field finding in eyes with optic atrophy in accordance with their etiology.

The etiology of trauma was found mainly in the age group of 16 to 30 years and 31 to 45 years of age (n=6). Post inflammatory etiology was most prevalent among the age group 31 to 45 years with ischemic etiology being prevalent in age group 61 to 75 years. Demyelinating (n=2) and compressive (n=3) etiology were found in equal numbers among age group of 16 to 30 years and 31 to 45 years.

Nonspecific defect (n=24, 24.7%) was the most common color vision defect seen among the examined eyes while Proton, Deutan and Tritan defect was found in 3 (3.1%), 3 (3.1%) and 4 (4.1%) eyes respectively. The mean contrast sensitivity in eyes with pale disc was 0.6586 log units (0.5082 – 0.8089, 95% C.I) and in eyes with temporal pallor was 1.1741 log units (1.1991 – 1.3849, 95% C.I).

Among the 16 patients whose etiology was traumatic; they had a history of trauma of varying severity prior to the onset of the diminution of vision and on the routine investigation no other detectable cause could be identified. Among the 10 patients with optic atrophy resulting from post inflammatory cause, 6 patients had suffered from optic neuritis receiving trial of pulse steroid therapy, 2 patients had a history of chorio-retinal disease and 2 patients had meningitis.

Four out of 6 patients with compressive etiology had intracranial space occupying lesions while among the remaining two, 1 had hydrocephalus and 1 had suffered orbital compartmental syndrome. Among 5 patients with demyelinating cause, 2 cases were diagnosed as cases of neuromyelitis optica, 2 cases were diagnosed as cases of multiple sclerosis while the remaining 1 case showed

features of demyelination in MRI with no established specific cause. All 5 patients with ischemic cause had small vessel disease and microvasculopathy.

Among 4 patients with metabolic cause of optic atrophy, two of them were vitamin B12 deficiency with 1 patient on Anti Tubercular

Therapy. While Ethambutol induced toxicity and diabetic papillopathy was the cause in one patient each. Finally 2 patients with hereditary cause, were one case each of Wolfram Syndrome and Leber's hereditary optic neuropathy (LHON).

Discussion

Table 1: Etiological causes of optic nerve atrophy with its key points

Etiology	Keynotes
Hereditary	Congenital and infantile optic atrophy; recessive or dominant; associated with other neurological and systemic signs.
Ischemic	Anterior or posterior ischemic optic neuropathy; arteritic or non arteritic
Metabolic	Nutritional and toxic causes; agents are methanol (Naeser, 1988), tobacco (Bern et al., 1995), Ethylene glycol (Jacobsen and McMartin, 1986), Ethambutol (Carr and Henkind, 1962), Hydroxyquinolones (Oakley, 1973), disulfiram (Acheson and Howard, 1988) etc.
Demyelinating	Optic neuritis occurs as an isolated occurrence; patients who have or will develop multiple sclerosis, neuromyelitis optica (Miller et al., 2005).
Compressive	Compression of anterior visual pathway; with or without disc oedema; life threatening causes (Marcus et al., 1991)
Post inflammatory	After inflammation of the optic disc subsides without treatment (Neetens and Smets, 1989); optic neuritis (non-infectious, infectious and para-infectious causes) and papilledema
Traumatic	Direct and indirect injuries; high momentum deceleration injuries, especially with midface trauma and results from the frontal impact by falling debris, assault (Seiff, 1990), stab wound, gunshot wound (Spoor et al., 1990), following trivial injuries (Sullivan and Helveston, 1969), and endoscopic sinus surgery (Dunya et al., 1996). Optic nerve sheath hemorrhage (Crompton, 1970); force applied to the frontal bone is transferred and concentrated in the area of the optic canal (Gross et al., 1981).

Table 2: Etiology of Optic nerve Atrophy

Etiology	Number	Percent
Traumatic	16	25.8
Idiopathic	14	22.6
Post Inflammatory	10	16.1
Compressive	6	9.7
Ischemic	5	8.1
Demyelinating	5	8.1
Metabolic	4	6.5
Hereditary	2	3.2
Total	62	100

Table 3: Clinical Characteristics of the patients with optic atrophy in accordance with their etiology

	Hereditary	Ischemia	Metabolic	Demyelinating	Compressive	Post Inflammatory	Traumatic	Idiopathic	Total
	N=2(%)	N=5(%)	N=4(%)	N=5(%)	N=6(%)	N=10(%)	N=16(%)	N=14(%)	N=62(%)
Gender Distribution									
Male	1(2.9)	2(5.9)	3(8.8)	1(2.9)	4(11.8)	4(11.8)	14(41.2)	5(14.7)	34(54.8)
Female	1(3.6)	3(10.7)	1(3.6)	4(14.3)	2(7.1)	6(21.4)	2(7.1)	9(32.1)	28(45.2)
Age Group									
16 - 30	1(5.6)	0(0)	1(5.6)	2(11.1)	3(16.7)	3(16.7)	6(33.3)	2(11.1)	18(29)
31 - 45	0(0)	1(4)	2(8)	2(8)	3(12)	6(24)	6(24)	5(20)	25(40.3)
46 - 60	1(5.6)	1(11.1)	1(11.1)	1(11.1)	0(0)	1(11.1)	2(22.2)	2(22.2)	9(14.5)
61 - 75	0(0)	3(42.9)	0(0)	0(0)	0(0)	0(0)	2(28.6)	2(28.6)	7(11.3)
more than 75	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	3(100)	3(4.8)
Laterality									
Both Eyes	2(5.7)	4(11.4)	4(11.4)	2(5.7)	3(8.6)	6(17.1)	4(11.4)	10(28.6)	35(56.4)
Right Eye	0(0)	1(10)	0(0)	1(10)	2(20)	1(10)	5(50)	0(0)	10(16.1)
Left Eye	0(0)	0(0)	0(0)	2(11.8)	1(5.9)	3(17.6)	7(41.2)	4(23.5)	17(27.4)
Presenting Complaint									
Diminution of Vision	2(3.8)	5(9.4)	3(5.7)	5(9.4)	6(11.3)	8(15.1)	14(26.4)	10(18.9)	53(85.5)
Decrease Field of Vision	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	1(100)	1(1.6)
Headache	0(0)	0(0)	0(0)	0(0)	0(0)	1(100)	0(0)	0(0)	1(1.6)
Routine Evaluation	0(0)	0(0)	1(25)	0(0)	0(0)	0(0)	1(25)	2(50)	4(6.4)
Diplopia	0(0)	0(0)	0(0)	0(0)	0(0)	1(100)	0(0)	0(0)	1(1.6)
Deviation of Eye	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	1(100)	0(0)	1(1.6)
Others	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	1(100)	1(1.6)

	Hereditary	Ischemia	Metabolic	Demyelinating	Compressive	Post Inflammatory	Traumatic	Idiopathic	Total
	N=4(%)	N=9(%)	N=8(%)	N=7(%)	N=9(%)	N=16(%)	N=20(%)	N=24(%)	N=97(%)
Best Corrected Visual Acuity									
6/6 - 6/18	0(0)	3(12.5)	2(8.3)	0(0)	2(8.3)	5(20.8)	4(16.7)	8(33.1)	24(24.7)
6/24 - 6/60	4(12.5)	4(12.5)	6(18.8)	1(3.1)	0(0)	3(9.4)	5(15.6)	9(28.1)	32(33)
5/60 - 3/60	0(0)	0(0)	0(0)	0(0)	0(0)	1(25)	2(50)	1(25)	4(4.1)
<3/60 - PL	0(0)	0(0)	0(0)	5(21.7)	3(13)	6(26.1)	3(13)	6(26.1)	23(23.7)
No Light Perception	0(0)	2(14.3)	0(0)	1(7.1)	4(28.6)	1(7.1)	6(42.9)	0(0)	14(14.4)
Pupillary Reaction									

Round/ Regular/ Reactive	4(5.5)	8(11)	8(11)	4(5.5)	5(6.8)	12(16.4)	10(13.7)	22(30.1)	73(75.2)
RAPD	0(0)	0(0)	0(0)	2(11.8)	2(11.8)	4(23.5)	7(41.2)	2(11.8)	17(17.5)
APD	0(0)	0(0)	0(0)	1(16.7)	2(33.3)	0(0)	3(50)	0(0)	6(6.2)
Irregular	0(0)	1(100)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	1(1)
Colour Vision									
Could not be done	2(4.8)	3(7.1)	2(4.8)	5(11.9)	7(16.7)	5(11.9)	9(21.4)	9(21.4)	42(43.3)
Normal	0(0)	2(9.5)	4(19)	1(4.8)	2(9.5)	4(19)	1(4.8)	7(33.3)	21(21.6)
Non Specific defect	2(8.3)	2(8.3)	2(8.3)	1(4.2)	0(0)	4(16.7)	6(25)	7(29.2)	24(24.7)
Proton	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	3(100)	0(0)	3(3.1)
Deutan	0(0)	0(0)	0(0)	0(0)	0(0)	2(66.7)	1(33.3)	0(0)	3(3.1)
Titran	0(0)	2(50)	0(0)	0(0)	0(0)	1(25)	0(0)	1(25)	4(4.1)
Goldmann Visual Field									
Normal	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	4(100)	4(4.1)
Enlarged Blind Spot	0(0)	4(22.2)	4(22.2)	0(0)	0(0)	2(11.1)	3(16.7)	5(27.8)	18(18.5)
Relative Scotoma inferior to fovea	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	1(100)	1(1)
Constriction of sup. field and central region with enlarged blind spot	2(15.4)	0(0)	2(15.4)	0(0)	0(0)	1(7.7)	5(38.5)	3(23.1)	13(13.4)
Constriction of all isopters	2(11.1)	2(11.1)	0(0)	1(5.6)	2(11.1)	4(22.2)	2(11.1)	5(27.8)	18(18.5)
Inferior Altitudinal Defect	0(0)	0(0)	0(0)	0(0)	0(0)	0(0)	1(100)	0(0)	1(1)
Constriction of isopters at centre	0(0)	0(0)	0(0)	0(0)	0(0)	1(100)	0(0)	0(0)	1(1)

In our study, the most common etiology for optic nerve atrophy was traumatic, similar to study by Bajracharya K et al(2016) while in the study by Osaguona and Okeigbemen(2015) traumatic was the second common cause. It was seen from our study, unilateral optic atrophy resulting from trauma was 3 fold

greater than bilateral. This finding of traumatic as the commonest cause could be interpreted as a result of referral; being, a tertiary center, cases of RTA and other trauma are referred. The majority of patients with trauma were in the young productive age group of 16-45 years, leading to blindness.

In our study, the number of patient who had history of optic neuritis or had ischemic optic neuropathy were high compared to other literature (Bajracharya et al., 2016) but the reported chorioretinal disease was significantly lower in our study than that reported by Osaguona and Okeigbemen(2015). However 30 to 33% of the cases of optic neuritis has been reported to eventually result in optic atrophy (Trip et al., 2006). The cases with compressive etiology were similar to the comparing literature (Bajracharya et al., 2016; Osaguona and Okeigbemen, 2015) but incidence as high as 29.5% has been reported by Menon V et al(1992). The number of cases with metabolic and toxic causes in our study was similar to that found by Osaguona and Okeigbemen(2015) but significantly higher than that reported by Bajracharya K et al(2016). The hereditary causes we reported were different and this may have resulted due to exclusion of pediatric age group.

The mean age in this study was similar to that reported by Osaguona and Okeigbemen(2015) but lower than that reported by Bajracharya K et al(2016). There was male preponderance similar to other studies (Menon et al., 1992), but it is noteworthy that all 3 cases of optic atrophy above 75 years that presented were all female.

Diminution of vision was the most common presenting complaint, even though our cases had better visual acuity compared to other literature. The commonest BCVA in this study ranged from 6/24 to 6/60. Color vision defect that is present in this study is similar to the reported in other studies (Katz, 1995) also this study obelizes with reduction of contrast sensitivity as reported by other literature (Marshall, 1950; Värinen et al., 1983)

We found enlarged blind spot and constriction of all isopter as the commonest visual field seen in almost all of our etiologies although central

scotoma is reported as the most common visual field defect among toxic neuropathies with other field defect like peripheral isopter constriction, altitudinal defect and bitemporal field defect being less common in cases of LHON with cecentral scotomas as classical presentation (Kedar et al., 2011; Marshall, 1950).

The main limitation of our study was, it was a hospital based cross sectional study with a short duration and small sample size from a specific clinic. Therefore the study results could not be generalized. In addition, we lacked availability of the advanced laboratory at the genetic level for the confirmatory diagnosis of hereditary disease. Also, we were unable to include the pediatric population due to ethical consideration, inclusion of whom may have given us different results.

Conclusion

It might be inferred that unilateral optic atrophy tends to present more often with etiology such as trauma and demyelinating disease, whereas bilateral optic atrophy presented with etiology such as hereditary, ischemia, metabolic and post inflammation. Nevertheless, detailed evaluation should be conducted in all patients with optic nerve atrophy to establish the underlying etiology.

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