

Nonglaucomatous optic neuropathies in Port Harcourt

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Aim: To review the types of nonglaucomatous optic neuropathies seen in Port Harcourt, Nigeria.

Materials and methods: Medical case records of all cases of nonglaucomatous optic neuropathy seen in the Eye Clinic of University of Port Harcourt Teaching Hospital, Port Harcourt, Nigeria over a 5-year period were reviewed. Relevant data including patient demography, presenting visual acuity and intraocular pressure, and fundus findings were extracted. Other details included central visual field analysis and color vision results. Cases of glaucomatous optic neuropathy were excluded.

Results: Ninety-nine cases of optic neuropathy were seen over this time period. There were 52 (52.5%) males and 47 females. The mean age of those with optic neuropathy was 40 ± 18.774 years. Twenty-two patients (22.2%) had bilateral involvement, while the rest were unilateral. About 40% of the patients with optic neuropathies had optic atrophy. Nutritional causes accounted for 31.3% and optic neuritis 27.3%. Over 60% of the patients presented with visual acuities $\leq 6/60$. Most patients (40%) had cecentral scotoma.

Conclusion: Nonglaucomatous optic neuropathies resulting in optic atrophy is not uncommon in our environment and several factors may be implicated but the actual cause is difficult to determine because of late presentation of most patients.

Keywords: nonglaucomatous, optic neuropathies, Port Harcourt

Introduction

The diagnosis of optic neuropathy is usually made on clinical grounds and sometimes with the help of ancillary tests such as visual field analysis and optical coherence tomography. Rarely can the etiology of optic neuropathy be made on the basis of a single clinical finding because there is considerable overlap of clinical features between even the relatively common optic neuropathies.^{1,2} It is said that in experienced hands, clinical diagnosis is reliable and sufficient, and further investigation is not required.³ The clinical diagnosis of optic neuropathies is based on the association of visual loss, altered color vision, visual field defect, and relative afferent papillary defect (if the optic neuropathy is unilateral).⁴ Pathologic cupping of the optic nerve is most commonly associated with glaucomatous optic neuropathy but other types of optic neuropathies have also been reported to cause cupping of the optic disk; conditions such as ischemic optic neuropathy,⁵ intracranial tumors,⁶ and optic neuritis.⁷ Clinically, clues such as optic nerve head pallor can be useful in distinguishing glaucomatous from nonglaucomatous optic nerve head cupping.⁸

The onset of optic neuropathy is not always clear. Inflammatory optic neuropathy tends to have a subacute onset over a number of days, frequently accompanied by

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periocular pain exacerbated by eye movements.⁹ The almost complete recovery of vision over the next few weeks is sufficiently characteristic of the vast majority of cases of demyelinating optic neuropathy to allow a clinical diagnosis, without the need for confirmatory investigation as long as there are no atypical features.^{10,11} Compressive optic neuropathy usually causes gradually progressive visual loss, although vascular lesions may cause abrupt visual decline occasionally, followed by some recovery. Nutritional or toxic optic neuropathy results in subacute or chronic symmetrical bilateral visual loss. Visual loss due to congenital anomalies rarely changes over time.

The ability to differentiate glaucomatous from nonglaucomatous cupping is usually difficult even with experience, however, evaluation of parapapillary chorioretinal atrophy has been found to be useful in differentiating both types of optic neuropathy.¹² Some researchers in the US¹³ analyzed four optic disk features in order to distinguish nonglaucomatous from glaucomatous optic neuropathy and found neuroretinal rim pallor to be 94% specific for nonglaucomatous atrophy, while focal or diffuse obliteration of the neuroretinal rim was 87% specific for glaucoma. These two signs were said to be the most useful in making the distinction between glaucomatous and nonglaucomatous atrophy. Thinning of the rim was, however, more common in glaucomatous atrophy and laminar dots were said to be present in both types of excavation.¹³ Also, glaucomatous optic neuropathy is not symptomatic until late in the disease course such that it is usually identified incidentally or by screening.¹⁴

The anterior visual pathway is susceptible to damage from toxins or nutritional deficiencies. This causes a type of optic neuropathy referred to as tobacco–alcohol amblyopia which is characterized by central or cecocentral scotoma.¹⁵ Toxic/nutritional optic neuropathy is quite common in

Table 2 Corrected visual acuity of affected eyes

Visual acuity	Right eye (%)	Left eye (%)	Total (%)
6/12	6 (3.9%)	8 (5.2%)	14 (9.0%)
6/18	9 (5.8%)	5 (3.2%)	14 (9.0%)
6/24	7 (4.5%)	7 (4.5%)	14 (9.0%)
6/36	9 (5.8%)	8 (5.2%)	17 (11.0%)
6/60	12 (7.7%)	16 (10.3%)	28 (18.0%)
CF	10 (6.5%)	12 (7.7%)	22 (14.2%)
HM	15 (9.7%)	6 (3.9%)	21 (13.6%)
LP	1 (0.6%)	3 (1.9%)	4 (2.6%)
NPL	13 (8.4%)	8 (5.2%)	21 (13.6%)
Total	82 (52.9%)	73 (47.1%)	155 (100%)

Abbreviations: CF, counting fingers; HM, hand movement; LP, light perception; NPL, no perception of light.

conditions of chronic alcohol abuse and malnutrition especially during times of war or famine as was the case in Cuba in the early 1990s.¹⁶

Materials and methods

Medical case records of all cases of nonglaucomatous optic neuropathy seen in the Eye Clinic of University of Port Harcourt Teaching Hospital (UPTH) over a 5-year period (January 2006–December 2010) were reviewed. Relevant data including age, sex, corrected visual acuity (Snellen chart), and intraocular pressure (Perkins MK 2 Hand held applanation tonometer; HS Clemens Clarke International, Essex, UK), and fundus findings (slit-lamp biomicroscopy with +78D) were extracted from the records. Other details were their central visual field analysis (Humphrey visual field analyzer, Model 740; Carl Zeiss Ophthalmic Systems Inc, Dublin, CA) and color vision results (Ishihara pseudoisochromatic color plates). Exclusion criteria included all cases of glaucomatous optic neuropathy (based on abnormal central visual field results [Humphrey perimetry], vertical cup/disk ratio >0.5, with or without elevated intra-ocular pressure). Data was analyzed using Epi Info (v 6.02; Centers for Disease Control and Prevention, Atlanta, GA) and *P* values <0.05 were considered significant.

Results

Table 1 shows the age and gender distribution of patients who were diagnosed with optic neuropathy during the period under review. A total of 99 cases of optic neuropathy

Table 1 Age and gender distribution of patients with optic neuropathy

Age group (years)	Sex		
	Male (%)	Female (%)	Total (%)
<10		3 (3.0%)	3 (3.0%)
10–19	9 (9.1%)	6 (6.1%)	15 (15.2%)
20–29	18 (18.2%)	10 (10.1%)	28 (28.3%)
30–39	6 (6.1%)	6 (6.1%)	12 (12.1%)
40–49	4 (4.0%)	4 (4.0%)	8 (8.1%)
50–59	5 (5.0%)	10 (10.1%)	15 (15.2%)
60–69	7 (7.1%)	5 (5.1%)	12 (12.1%)
70–79	3 (3.0%)	2 (2.0%)	5 (5.0%)
80–89		1 (1.0%)	1 (1.0%)
Total	52 (52.5%)	47 (47.5%)	99 (100%)

Table 3 Types of optic neuropathy

Types of optic neuropathy	Number (%)
Optic atrophy (most causes unknown)	41 (41.4%)
Optic neuritis	27 (27.3%)
Nutritional optic neuropathy/unknown	31 (31.3%)
Total	99 (100)

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Table 4 Fundus findings

Fundus picture	Number (%)
Pink/hyperemic disk	20 (20.2%)
Temporal disk pallor	31 (31.3%)
Pale neuroretinal rim	41 (41.4%)
Blurred disk margins	7 (7.1%)
Total	99 (100%)

were seen over a 5-year period. There were 52 (52.5%) males and 47(47.5%) females giving a male:female ratio of 1.1:1. The mean age of those with optic neuropathy was 40 ± 18.77 years. Over 55% of those with optic neuropathy were aged between 10 and 39 years old. Those in the age group 20–29 years were the most affected (28.3%) and in this particular group, males were about twice as affected as females (male:female = 1.8:1). The least affected were those in both extremes of ages (<10 years old: 3%; 80–89 year olds: 1%).

Table 2 shows the corrected visual acuity of the study subjects. The right eye was involved in 82 cases (52.9%) and the left eye in 73 cases (47.1%). Twenty-two patients (22.2%) had bilateral involvement while 77.8% (N = 77) had unilateral involvement. Altogether, 155 eyes of 99 patients were involved. Over 60% of the patients presented with visual acuities $\leq 6/60$, only 14 patients had $VA \geq 6/18$.

Table 3 shows the types of optic neuropathy in this study. About 40% of the patients had optic atrophy at presentation. Optic neuritis (retrobulbar neuritis and papillitis) accounted for 27.3% of cases, while the rest (31.3%) were due mainly to nutritional causes (on account of temporal disk pallor) and some unknown causes. The mean age of those with optic atrophy was 41.35 ± 18.28 years and for those with optic neuritis 26.67 ± 12.10 years.

Only 65 patients had perimetry. The remaining 34 did not because of severe optic atrophy. Table 5 shows that most patients (40%) had cecocentral scotoma followed by generalized depression of sensitivity (15.3%). Enlargement of the blindspot and central scotoma were noted in five cases each. Of the 20 patients who had pink/

hyperemic disks, fifteen (25.9%) had normal central visual field (CVF) results.

Discussion

Optic atrophy was the most common type of nonglaucomatous optic neuropathy reported in this study. This is much higher than the study by Gupta et al,⁸ however, a similarity between this study and that by Gupta et al was that most of our patients were also in their early forties. In their study, only twelve patients were studied and this could account for the small number of patients with optic atrophy. In addition, patients in our setting tended to report late for treatment and this may account for the large number of cases of optic atrophy as these patients may be seen at the terminal end of the disease process.

Cassava and cassava products are staple foods in southern Nigeria and there is good evidence that it causes optic neuropathy because of its high cyanide content.^{17,18} Could this be the case with some of our patients? This could also explain the large number of cases of nutritional optic neuropathy seen in this study. A characteristic of patients in our environment is the tendency to withhold important information about their health especially if they think it may be blamed for their illness. Despite this, there is the likelihood that chronic alcohol consumption and nutritional deficiency may play important roles in some of the cases of optic neuropathy seen because illicit gin is brewed and consumed in large quantities in this environment. Even though many cases of optic atrophy were seen, it was difficult to say for certain what the exact causes were but they were certainly not glaucomatous based on the exclusion criteria; most looked to be due to secondary causes such as resolved disk edema. All cases of optic neuropathy occurred in isolation. We did not record any cases of hereditary optic neuropathy and there was also no evidence of a compressive lesion in any of our cases. It is not entirely certain if some of the neuropathies could have been due to ischemic optic neuropathy as a few of the fundus pictures revealed slightly hyperemic disks.

Table 5 Central visual field analysis (Humphrey)

Fundus picture	Number	Normal CVF	Central scotoma	Cecocentral scotoma	Altitudinal defect	Generalized depression of sensitivity	Enlargement of blind spot
Temporal disk pallor	31	–	5 (7.7%)	26 (40%)	–	–	–
Blurred disk margins	7	–	–	–	–	2 (3.0%)	5 (7.7%)
Pink/hyperemic disk	20	15 (23.1%)	–	–	4 (6.2%)	1 (1.5%)	–
Optic atrophy	7	–	–	–	–	7 (10.8%)	–
Total	65 (100%)	15 (23.1%)	5 (7.7%)	26 (40%)	4 (6.2%)	3 (15.3%)	5 (7.7%)

Abbreviation: CVR, control visual field.

One of the major limitations of this study is the fact that none of our patients had an Optical Coherence Tomography (OCT) to properly assess the retinal nerve fiber layer which would have helped in characterizing the different types of optic neuropathies. OCT would also have assisted in categorically differentiating glaucomatous from nonglaucomatous optic neuropathy.

Conclusion

Nonglaucomatous optic neuropathies resulting in optic atrophy are not uncommon in our environment and several factors may be implicated but this is difficult to know because of the late presentation of most patients. A prospective study is proposed in future and should be able to address this issue as this may be part of the exclusion criteria.

Disclosure

The authors report no conflicts of interest in this work.

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