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A CLINICAL STUDY ON OPTIC ATROPHY IN PATIENTS ATTENDING REGIONAL EYE HOSPITAL KURNOOL.

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ABSTRACT **Aim:** to study the clinical profile of patients with optic atrophy attending regional eye hospital, Kurnool.**Methods:** Patients attending the outpatient department of Regional Eye Hospital, Kurnool, during the period from Feb 2019 to Feb 2020 were examined. The examination included distance and near visual acuity testing using the Snellen chart, refraction, color vision, slit-lamp biomicroscopy, intraocular pressure measurement using Goldmann Applanation Tonometer, fundus examination using direct and indirect ophthalmoscope, and visual field testing using Humphrey field analyzer. Patients with optic nerve involvement were exclusively included in the study. **Results:** a total of 120 patients had optic atrophy. Of these 120 patients, 25 had primary optic atrophy, 23 had secondary optic atrophy, 40 had consecutive optic atrophy, and 32 had glaucomatous optic. Among the 40 patients with consecutive optic atrophy, 28 had associated retinitis pigmentosa, 12 had associated choroiditis.

KEYWORDS : Primary Optic Atrophy, Secondary Optic Atrophy, Consecutive Optic Atrophy And Glaucomatous Optic Atrophy.

INTRODUCTION

Optic atrophy results from various lesions of the visual pathway from the ganglion cell layer to the lateral geniculate body[1]. Since the optic nerve is the conduit for information from the retina to the brain, a damaged optic nerve will result in vision loss. Slight damage might not affect visual acuity but may lead to a loss of contrast or color vision. Severe damage may lead from legal blindness to no light perception. Damage to a part of the optic nerve results in loss of vision in the corresponding visual field. Clinically, optic atrophy is diagnosed from the well-known triad of the pallor of the optic disc, diminution in the visual acuity and visual field defects. Depending upon the etiology, and ophthalmoscopic picture, different classifications of optic atrophy have been in vogue[1].

MATERIALS AND METHODS

The study was conducted on optic atrophy patients presented to the outpatient department of Regional Eye Hospital Kurnool from Feb 2019 to Feb 2020 . Besides history regarding the loss of vision, a complete ophthalmological examination was carried out. The ophthalmological examination included distance and near visual acuity, both presenting and best-corrected after refraction, for each eye separately. Objective refraction was performed with a streak retinoscope, followed by subjective acceptance with which the best-corrected acuity was measured and recorded. Colour vision was tested using ishihara chart. External eye examination was done under slitlamp biomicroscope where relative afferent pupillary defect was observed in some cases. Intra ocular pressure was measured using Goldmann applanation tonometry. Fundus examination with direct and indirect ophthalmoscopy was done after full dilatation of pupil. Automated visual fields were done with the Humphrey visual field analyzer using the central 24-2 threshold strategy in those participants suspicious of having optic nerve pathology with better vision. Those with optic nerve pathology were subjected to OCT examination to examine the peripapillary retinal nerve fibre layer.

The optic atrophy was classified according to the ophthalmoscopic picture as under[1]:

1. Primary optic atrophy : chalky white pallor of the disc is present extending upto the disc margin, the margins of the disc is well defined ; retinal vessels are of normal caliber. The physiological cup was slightly deeper than normal and lamina cribrosa were seen more clearly.
2. Secondary Optic Atrophy*:* fundus shows greyish white pallor of the disc with ill-defined margins of the disc with obliteration of the physiological cup and fibroglial tissue proliferation totally obscuring the lamina cribrosa. Sheathing of vessels predominantly arteries nearer to the disc with narrowing of the vessels noted. Such a picture may be due to papilloedema or papillitis.
3. Consecutive Optic Atrophy*:* the disc is waxy pallor with marked attenuation of the arteries and veins with normal well defined margins of the disc and physiological cup.
4. Glaucomatous optic atrophy: fundus shows disc with wide cupping, bean pot cup with nasalization of the vessels, with notching of the neuro-retinal rim, backward bowing and excavation of the lamina cribrosa.

RESULTS :

Table 1 : Age distribution of cases

Age	Primary optic atrophy	Secondary optic atrophy	Consecutive optic atrophy	Glaucomatous optic atrophy	Total
0-10	1	0	4	0	5(4.16%)
11-20	4	3	6	0	13(10.83%)
21-30	12	6	14	0	32(26.67%)
31-40	6	5	12	5	28(23.34%)
41-50	1	4	2	12	19(15.83%)
>50	1	5	2	15	23(19.16%)
Total	25(20.83%)	23(19.16%)	40(33.34%)	32(26.67%)	120(100%)

Table 2: Gender distribution of cases

Sex	Primary optic atrophy	Secondary optic atrophy	Consecutive optic atrophy	Glaucomatous optic atrophy	Total
Males	16	14	24	18	72(60%)
Females	9	9	16	14	48(40%)
Total	25 (20.83%)	23 (19.16%)	40 (33.34%)	32 (26.67%)	120 (100%)

Table 3: Distribution of cases with respect to laterality of eye involved

Laterality	Primary optic atrophy	Secondary optic atrophy	Consecutive optic atrophy	Glaucomatous optic atrophy	Total
RE	7	9	6	8	30(25%)
LE	6	6	6	14	32(26.67%)

BE	12	8	28	10	58(48.34%)
Total	25(20.83%)	23(19.16%)	40(33.34%)	32(26.67%)	120(100%)

Table 4 : Causes of optic atrophy

Cause of optic atrophy	No of cases	Percentage
Glaucomatous optic atrophy	32	26.67%
Trauma	8	6.67%
Retinitis pigmentosa	28	23.34%
Choroiditis	12	10%
Papilloedema	8	6.67%
Others	32	26.67%
Total	120	100%

DISCUSSION

Optic atrophy results from conditions that produce degeneration of ganglion cells and axons of the retina upto the lateral geniculate body. It is a descriptive term that does not imply a specific etiology or mechanism of injury. Diagnosis is based on the ophthalmoscopic appearance of a pale optic disc with defective visual function. Loss of visual acuity, visual field defects, or both may occur. A reduction in the vascularity of the optic nerve head occurs, so that ophthalmoscopically the disc becomes pale or white rather than its normal pink color.

In our study, 120 patients had optic atrophy. Of these 120 patients, 25(20.83%) had primary optic atrophy, 23 (19.16%) had secondary optic atrophy, 40(33.34%) had consecutive optic atrophy and 32(26.67%) had glaucomatous optic atrophy.

Of these 120 patients, 72 (60%) were males, and 48 (40%) were females indicating males were more affected than females. In a study done in Nigeria, 52.5% male and 47.5% female were found[2].

Among these 120 patients, Right eye involvement was seen in 30(25%) patients, left eye was involved in 32(26.67%) patients, and bilateral involvement was seen in 58(48.34%) patients.

Among 25 (20.83%) patients with primary optic atrophy, 12(48%) patients were between 21-30 years and 16 (64%) were males, and 9 (36%) were females. Among these 25 patients, 8 (32%) patients had a history of trauma, which lead to the development of traumatic optic atrophy, causing blindness. This traumatic optic atrophy manifested unilaterally. These 8 patients were categorized under primary optic atrophy, depending on the fundus picture.

Among 23(19.16%) patients with secondary optic atrophy, 6 (24%) patients were between the age group 21-30. Among these 23 patients, 14 (56%) were males, and 9 (32%) were females.

Among 40 (33.34%) patients with consecutive optic atrophy, 14(35%) patients were between the age group 21-30 and 12 (30%) were between the age group of 31-40 years. 24 (60%) were males, and 16 (40%) were females. Among these 40 patients, 28 (70%) patients had associated retinitis pigmentosa, 12 (30%) patients had associated choroiditis.

In our study, 32 (26.67%) patients had glaucomatous optic atrophy. Glaucoma was the cause in 58% of cases in a study conducted by Kabindra Bajracharya et al. [3]

In our study, 8(6.67%) patients had a history of trauma. Chaddah MR et al.[1] found trauma comprised 7% of optic atrophy, and Oluleye T.S et al. [4] found 8%. But in the study done in Singapore, it was responsible for 16.2% [5].

In our study, 28 (23.34%) patients had associated retinitis pigmentosa, 12(10%) patients had associated choroiditis. Oluleye T.S et al. found a 3% association with RP[4].

In our study, 8(6.67%) patients had a history of papilloedema. Chronic papilloedema was a causative factor in 4% of cases in a study conducted by Kabindra Bajracharya et al. [3].

In our study, other causes constitute 32 (26.67%) cases.

CONCLUSION

Patients with optic atrophy in our study typically had ophthalmoscopic findings that led to an etiological diagnosis. The optic nerve has some reserve axons before vision loss is appreciated, so early detection is vital in order to slow the progression of the disease-causing optic atrophy so that progression to complete irreversible blindness can be delayed.

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