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OPTIC DISC OEDEMA: A RETROSPECTIVE STUDY IN A TERTIARY CARE HOSPITAL

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ABSTRACT **BACKGROUND:** Optic disc oedema in many cases is an important ocular manifestation of some systemic pathology. The underlying pathology of optic disc oedema can be a potentially life-threatening condition and can lead to severe visual loss. **AIM:** To study the epidemiologic and clinical characteristics of patients with disc oedema and diagnose the underlying aetiology.**METHOD:** This retrospective observational study was conducted in patients with disc oedema attending eye OPD or referred from other departments from March 2019 to August 2019 in a tertiary care hospital. Detailed ocular examination including visual acuity, slit-lamp examination, fundus evaluation by +90D lens or indirect ophthalmoscopy, and visual field analysis was done. Blood investigations and neuroimaging were done as and when required.**RESULTS:** A total of 58 consecutive cases with optic disc oedema were studied with a mean age of 36 years. Among them 51.72% were females and 48.28% were males. Out of the 58 cases, the most common cause was optic neuritis (41.38%), followed by papilloedema (24.14%), compressive optic neuropathy (8.62%), retinal venous occlusion (8.62%), anterior ischemic optic neuropathy (8.62%), inflammatory (6.9%), and diabetic papillopathy (1.7%).**CONCLUSION:** Proper diagnostic approach to a patient with optic disc oedema is necessary as the treatment strategy is based upon underlying aetiology.

KEYWORDS : Optic disc oedema, papilloedema, optic neuritis

INTRODUCTION

Optic disc oedema can be due to varied aetiologies. It can be unilateral or bilateral. The commonest cause of optic disc oedema in the Indian population is optic neuritis.[1]

One of the common causes of bilateral optic disc swelling is raised intracranial tension which can be potentially life-threatening.[2] Disc oedema if left untreated, can lead to irreversible vision loss and secondary optic atrophy.[3] Unilateral disc oedema can be due to optic neuritis, anterior ischemic optic neuropathy, etc.

This study is aimed at describing the epidemiological and clinical characteristics of patients with disc oedema and diagnosing the underlying aetiology.

MATERIALS AND METHOD

This was a retrospective observational institutional study conducted in the department of ophthalmology of a tertiary care hospital in Eastern India from 1st March 2019 to 31st August 2019. Data was collected from OPD and indoor records. Consecutive 58 patients with disc oedema were retrospectively analyzed. Ethical clearance was taken from the Institutional Ethical Committee.

All patients had undergone a detailed history taking, general, and ophthalmic examination including visual acuity by Snellen's chart, pupillary reaction, colour vision assessment by Ishihara's colour vision chart, detailed slit lamp examination, fundus evaluation by +90D lens or indirect ophthalmoscope with +20D lens, optical coherence tomography, digital fluorescein angiography. Visual field test by confrontation and/or automated perimetry using Humphrey Field Analyzer 24-2 was done wherever possible. Visual Evoked Potential (VEP) was done as indicated. Blood investigations and neuroimaging including Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) (contrast/non-contrast) were done as and when required.

The collected data were compiled in Microsoft Excel and the analyses were mostly descriptive.

RESULTS

A total of 58 cases were studied. Among them, 51.72% (n = 30) were females and 48.28% (n = 28) were males. Age distribution of the study population showed 24.14% (n = 14) between 21-30 years, followed by 20.7% (n = 12) between 41-50 years, 17.24% (n = 10) between 31-40 years, 15.5% (n = 9) between 51-60 years, 8.6% (n = 5) between each od 0-10 sand 11-20 years and 5.17% (n = 3) between 61-70 years. Unilateral cases were 53.4% (n = 31) and bilateral cases were 46.6% (n = 27).

The mean time of presentation was 13.6 days with 89.6% of the cases presenting with acute onset of symptoms. Dimness of vision was the most common presenting symptom involving 70.69% (n = 41) of the study population, followed by headache in 22.41% (n = 13), ocular pain in 20.69% (n = 12) and proptosis in 8.62% (n = 5). Few of the patients also had accompanying neurological symptoms like seizure, lower limb paresis, etc.

The diagnosis of the study population has been shown in Figure 1.

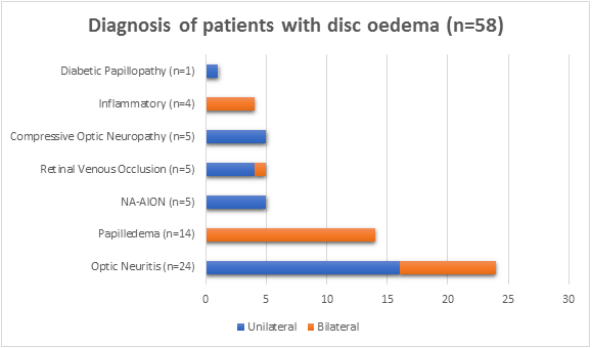

Figure 1 showing diagnosis of optic disc oedema in study population (n = 58)

Papilloedema, inflammatory causes of disc oedema were bilateral, whereas Non-arteritic Anterior Ischemic Optic Neuropathy (NA-AION), Compressive Optic Neuropathy (CON) were unilateral. Among the optic neuritis patients, 66.7% (n = 16) were unilateral and 33.33% (n = 8) were bilateral, 80% (n=4) cases of Retinal Venous Occlusion (RVO) were unilateral and 20% (n=1) were bilateral. Diabetic papillopathy was found to be unilateral.

Visual acuity on presentation was less than 20/200 in 55.2% (n=32) and greater than 20/40 in 10.3% (n=6). Twenty patients could respond to colour vision testing with 85% (n=17) of them having a red-green defect. Visual field testing by Automated Perimetry was done in patients with visual acuity greater than 20/40 (n=6). Four patients with papilloedema showed an enlarged blind spot, one patient with NA-

AION had an inferior altitudinal field defect and one patient with optic neuritis showed central scotoma.

Table 1 shows the aetiologies of disc oedema. Among the three cases of papilloedema due to hypertensive retinopathy, one was secondary to Pregnancy Induced Hypertension.

Table I showing aetiological causes of optic disc oedema (N=58)

Diagnosis	Causes	n (%)
Papilloedema (N = 14)	Idiopathic Intracranial Hypertension	3 (22)
	Hypertensive Retinopathy	3 (22)
	Intracranial space-occupying lesion	1 (7)
	Cerebral venous sinus thrombosis	1 (7)
	Hydrocephalus	1 (7)
	Cerebral malaria	1 (7)
	Others (Encephalomalacia, Apert syndrome, unknown)	4 (29)
Optic Neuritis (N = 24)	Tuberculosis	4 (29)
	Infective	7 (29)
	Multiple sclerosis	3 (13)
	Neuromyelitis optica	2 (8)
	Japanese encephalitis	1 (4)
	Undiagnosed	7 (29)
CON (N = 5)	Orbital cellulitis	3 (60)
	Orbital tumour	1 (20)
	Ethmoidal mass	1 (20)
RVO (N = 5)	Central retinal vein occlusion	4 (80)
	Superotemporal branch retinal vein occlusion	1 (20)
Inflammatory (N = 4)	Tubercular chorioretinitis	2 (50)
	Vogt-Koyanagi-Harada Syndrome	1 (25)
	Exudative retinal detachment	1 (25)

DISCUSSION

The swelling of the optic disc is due to the basic underlying pathogenesis of axoplasmic flow stasis which may be due to vascular or mechanical causes.[4] It can be a true swelling or a pseudo-optic disc swelling. It is important to differentiate between true optic disc oedema and pseudo-optic disc oedema in the first place to avoid unnecessary tests and initiate proper management without delay.

In our study, we have tried to find the various aetiologies of true optic disc oedema based upon their clinical presentations and necessary diagnostic tests and excluded cases of pseudo-optic disc oedema.

Optic disc swelling was found to be more common in the age group of 21-30 years with a mean age of 35.66 years, a finding similar to that of Meena V, et al and Solanki, et al.[2,3] Papilloedema can affect all age groups as observed by Rigi M, et al 5 and a similar finding has been seen in our study.

We observed more cases of unilateral disc oedema (53.4%) than bilateral (46.6%), a finding contradictory to that of Solanki, et al.[2]

Most of our patients presented to the OPD with the complaint of acute onset dimness of vision. This observation was consistent with the findings of Ijeri R, et al.[1] Among the symptomatology, decreased vision was followed by headache and ocular pain.

Dimness of vision was seen in patients with optic neuritis, RVO, papilloedema, diabetic papillopathy, hypertensive retinopathy, NA-AION, tubercular chorioretinitis, Vogt-Koyanagi-Harada (VKH) syndrome, exudative retinal detachment and CON. Headache was commonly reported in patients with papilloedema, similar to findings of previous studies.[3,6,7] Ocular pain was a common feature of patients with optic neuritis. Proptosis was present in patients with CON.

Ijeri R, et al. observed optic neuritis to be the most common aetiology of optic disc oedema in the Indian population, and a similar finding was reported in our study.[1] Among the optic neuritis patients, 62.5% presented with dimness of vision, 45.8% with ocular pain, and 8.3% with a headache. Optic neuritis secondary to viral infection was common in the young age group. Two patients of Neuromyelitis Optica and one patient of Japanese encephalitis were referred to us from the Department of Medicine and on ocular examination, they were diagnosed to have optic neuritis. Four cases of optic neuritis had pulmonary tuberculosis undergoing anti-tubercular therapy. Three cases of optic neuritis were diagnosed to have multiple sclerosis after an MRI report and consultation with a neurologist. But the majority of cases of optic neuritis were idiopathic, which coincides with the findings of Solanki et al.[2]

Papilloedema was the second most common cause of disc oedema found in our study. It is commonly bilateral, although rare cases of unilateral papilloedema have been reported.[8,9] Our study reported bilateral cases. It is of utmost importance to diagnose papilloedema and differentiate it from pseudo papilloedema as sometimes, papilloedema is the only objective sign to clinically confirm raised ICP. Three cases of papilloedema in our study were due to Idiopathic Intracranial Hypertension (IIH), out of which two were reported in young females. This finding was consistent with the findings of Meena, et al.[3] Four cases reported transient blurring of vision along with headache before presentation, which was a common finding reported in patients with papilloedema in previous studies. [10,11] One case of papilloedema secondary to cerebral venous sinus thrombosis was found. Underlying systemic pathologies leading to papilloedema as recorded from our study include cerebral malaria, frontoparietal tumour, encephalomalacia, malignant hypertension, Apert syndrome. Similar reports have been found by Solanki, et al.[2]

Relative afferent pupillary defect (RAPD) was appreciated in cases of unilateral optic neuritis and compressive optic neuropathy. A red-green colour defect was noted in patients with optic neuritis, intracranial space-occupying lesion (ICSOL), grade IV hypertensive retinopathy, etc. Likewise has been reported in a study by Parajuli A, et al.[12]

Fundus evaluation helped to diagnose the aetiology of disc oedema in cases associated with vascular changes like central retinal vein occlusion, diabetic papillopathy, hypertensive retinopathy, and cases with associated exudative retinal detachment, features of posterior uveitis as in VKH syndrome. (Figure 2)

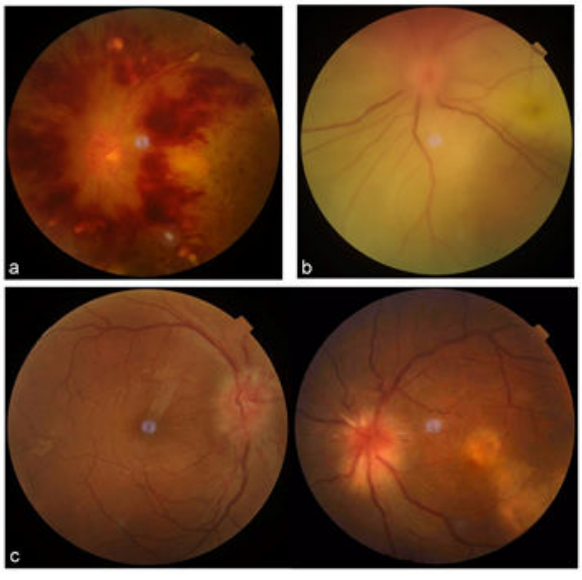

Figure 2 showing optic disc oedema in (a) LE CRVO, (b) LE Compressive Optic Neuropathy and (c) BE Tuberculous Chorioretinits

Visual field testing helped in the diagnosis of NA-AION which showed characteristic inferior altitudinal defect. Reported cases of NA-AION in our study were in the age group of 55-65 years and 75% of them had underlying co-morbidities like diabetes and hypertension. This observation was consistent with the findings of Vaidya K, et al.[13]

Neuroimaging techniques like CT scan and MRI of the brain, orbit as well as paranasal sinus play an important role in diagnosing the underlying aetiology of disc oedema. Radiologic examination showed demyelination in multiple sclerosis and neuromyelitis optica, optic nerve signal changes in optic neuritis, and empty sella in IIH. Other conditions like hydrocephalus, frontoparietal tumour, cerebral venous sinus thrombosis and encephalomalacia were also diagnosed from neuroimaging.

In our study, VEP was done in patients with optic neuritis which showed a prolonged latency and reduced amplitude of P100. The delay in latency suggested a demyelinating cause, which was supported by MRI findings. In a comparative study[14] by Jayaraman M, et al. optic nerve diseases showed reduced amplitude in VEP but the extent of delay in latency was found to be more in optic neuritis compared to ischemic optic neuropathy. However, we did not conduct VEP in our patients with NA-AION.

Causes of CON can be identified with the help of neuroimaging. Our study reported four cases of CON, out of which one was due to ethmoidal mass extending up to cavernous sinus with bony erosion, three cases were due to orbital cellulitis and subperiosteal abscess of orbit secondary to pansinusitis and one was due to compression from an orbital tumour. This finding was similar to a study by Williams BJ, et al.[15]

Thus, both local and systemic causes have been delineated as underlying pathology leading to unilateral and bilateral disc oedema. Local causes reported in our study include optic neuritis, CRVO, NA-AION, orbital tumour, ethmoidal mass, subperiosteal abscess of orbit secondary to pansinusitis. Systemic causes include malignant hypertension, pregnancy-induced hypertension, diabetes, multiple sclerosis, tuberculosis, NMO, Japanese encephalitis, cerebral malaria, SLE, ICSOL, meningitis.

CONCLUSION

Optic nerve head swelling although not commonly encountered in the OPD is important to be approached in a tailored manner as it includes both local as well as systemic causes. The aetiological diagnosis of optic disc oedema is often complex and based upon the association of both clinical findings and ancillary tests like visual field testing, neuroimaging, blood investigations, VEP. A guided clinical approach is necessary to pinpoint the underlying pathology as management is aimed at individual diagnosis which can require a multidisciplinary approach.

REFERENCES

1. Ijeri R, R.c J. Optic Disc Oedema: Presentation and Causes at a Tertiary Centre in North Karnataka. Off Sci J Delhi Ophthalmol Soc. 2018; 29(1):31–4.
2. Solanki D, Meena V, Sharma U, Agrawal S. Optic disc oedema/papilloedema: a clinical profile. J Evol Med Dent Sci. 2016;5(16):795–801.
3. Meena V, Sharma U. To evaluate the profile of patients with disc oedema/ papilloedema and their presenting pattern. Int J Med Res Rev. 2016;4(3):301–8.
4. Hayreh SS. Pathogenesis of optic disc oedema in raised intracranial pressure. Prog Retin Eye Res. 2016;50:108–44.
5. Rigi M, Almarzouqi SJ, Morgan ML, Lee AG. Papilloedema: epidemiology, etiology, and clinical management. Eye Brain. 2015;7:47–57.
6. Digre KB, Corbett JJ. Idiopathic intracranial hypertension (pseudotumor cerebri): a reappraisal. The Neurologist. 2001;7(1):2–68.
7. Wang SJ, Silberstein SD, Patterson S, Young WB. Idiopathic intracranial hypertension without papilloedema: a case-control study in a headache center. Neurology. 1991;51(1):245–9.
8. Brosh K, Strassman I. Unilateral papilloedema in pseudotumor cerebri. Semin Ophthalmol. 2013;28(4):242–3.
9. Wattamwar PR, Baheti NN, Radhakrishnan A. Idiopathic intracranial hypertension presenting as unilateral papilloedema. Neurol India. 2010;58(5):818.
10. Chow S, Draman N, Teh W, Azhany Y. Recurrent transient visual loss in a middle aged woman. Malays Fam Physician Off J Acad Fam Physicians Malays. 2017;12(3):42–6.
11. Feroze KB, O'Rourke MC. Transient Loss Of Vision. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2020 [cited Jul 24 2020]. Available from: http://www. ncbi. nlm. nih. gov/ books/ NBK 430845/
12. Parajuli A, Sharma A, Sitaula S. A Clinical Study of Optic Disc Oedema in a Tertiary Eye Center of Nepal. Nepal J Ophthalmol. 2019;11:122–9.
13. V K, Bhandari S, Gurung R. Etiologies of Optic Disc Oedema in Tertiary Eye Care Centre in Nepal. Nepal J Ophthalmol Biannu Peer-Rev Acad J Nepal Ophthalmic Soc NEPJOPH. 2018;10(20):139–42.
14. Jayaraman M, Gandhi RA, Ravi P, Sen P. Multifocal Visual Evoked Potential in Optic Neuritis, Ischemic Optic Neuropathy and Compressive Optic Neuropathy. Indian J Ophthalmol 2014; 62:299-304
15. Williams BJ, Harrison HC. Subperiosteal abscesses of the orbit due to sinusitis in childhood. Aust N Z J Ophthalmol. 1991;19(1):29–36.