Reminder of important clinical lesson

Optic disc oedema: a diagnostic dilemma

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The authors describe a patient who presented with bilateral disc oedema and left eye visual loss. The patient was evaluated for intracranial space occupying lesion, however, a CT and cerebrospinal tap was normal. No definitive diagnosis could be established and patient developed left eye optic atrophy which was thought to be post papilloedema atrophy. Six months later, the patient had similar visual loss in right eye and he was diagnosed as anterior ischemic optic atrophy (AION) by the ophthalmologist. The inferior altitudinal visual field loss, delayed visual evoked response and clinical picture supported the diagnosis. The patient was diagnosed with hypertension and type 2 diabetes and managed accordingly. All the symptoms, clinical findings, investigations supported the diagnosis of AION which occurred in left eye followed by right eye 6 months later. Appropriate treatment was started and patient regained vision in right eye.

BACKGROUND

Disc oedema is a common clinical finding seen by physicians, neurologist, ophthalmologists and residents posted in emergency room (ER). Doctors of particular specialty try to rule out common aetiology seen by them and then refer the patient to other specialists depending on the need for appropriate management. The patients are commonly subjected to radiological investigations such as CT because it is a non-invasive test and rules out life-threatening intracranial disorders. Various congenital disc abnormality, refractive error of hypermetropia, disc drusen can also

cause hyperemic appearance of optic disc which needs to be differentiated from life-threatening and sight threatening diseases such as papilloedema and optic neuritis. If one does not find an aetiology for disc oedema, unconventional diagnosis should be thought about. It is imperative that a team approach is made and all systemic and local aetiologies should be ruled out. We would like to highlight the various causes of disc oedema and their diagnostic points. We would like to point out that a multi-specialty approach will prevent misdiagnosis and lead to better patient management.

Figure 1 Right eye showing pallid disc oedema.

Figure 2 Left eye showing disc pallor and atrophy.

Figure 3 Visual field of right eye with inferior altitudinal defect.

CASE PRESENTATION

A 52-year-old male patient presented to the eye clinic of People's College of Medical Sciences and Research Centre, Bhopal, India with complains of blurring of vision of right eye since 1 week. The patient had a similar history of decrease in vision of left eye 6 months back.

Six months back the patient had an episode of sudden diminution of vision in the left eye after which he consulted a practicing ophthalmologist. Vision recorded at that time was 20/20 right eye and 20/400 left eye. The ophthalmologist made a diagnosis of bilateral papilloedema and advised a CT of brain to rule any space occupying lesion (SOL). The CT scan was normal and the patient was referred to a neurologist for further evaluation of papilloedema. The neurologist noted left eye optic disc pallor and continue with the suspicion of post papilloedema atrophy of left eye. A guarded lumbar puncture was done to collect the cerebrospinal fluid (CSF) for analysis. The CSF analysis was normal. The patient was found to be hypertensive with blood pressure of 180/100 and started on antihypertensive medication. The patient was given on oral prednisolone (1 mg/kg/day) for 2 weeks. Poor visual prognosis was explained for left eye however a clear diagnosis could not be established.

Six months later, the patient presented to our hospital with complaints of similar loss of vision of right eye. On examination, best corrected visual acuity (BCVA) was 20/400 right eye and counting finger vision left eye. Near vision was <N/36 both eyes. Pupillary reactions were sluggish in both eyes. Ocular adnexa were normal. Movements of the eye were normal and painless. Anterior segment examination was normal and intraocular pressure was 14 mm Hg in right eye and 12 mm Hg in left eye.

On fundus examination, right eye revealed segmental disc oedema of the nasal and superior margin and pallor of the temporal margin (figure 1). Cup disc ratio was 0.3:1

Table 1 Differential diagnosis of patient with disc oedema

Diagnosis	Vision	V. Fields	R. Veins	Disc	R. Hmn.	R. Oedema	Symm	Comments
Early papilloedema	Normal/mild blurring	Normal/enlarged blind spot	Loss of venous pulsation	Pink, blurred margins	+/-	+/-	Early asymmetry	Headache, projectile vomiting
Advance Papilloedema	Mild decrease, blurring	Enlarged blind spot	Engorged veins	Hyperemia, blurred margins	++	++ (less)	Bilateral	Above +6 CN palsy, disc leakage on FA
Hyperopia	Normal	Normal	Normal	Normal	Normal	Normal	Normal	Check diopter power of focus with direct ophthalmoscope
Optic neuritis	Marked decrease	Central/centrocaecal scotoma	Dilated/engorged (less)	Hyperemia, pink, blurred	+/-	+++ (marked)	Unilateral	Pain on movements, sudden onset vision loss
Drusen/hyaline bodies at disc	Normal	Normal/enlarged blind spot	Normal	Normal	Normal	Normal	Bilateral	Familial, risk of CRVO later
Pseudotumour cerebri	Transient visual obscuration	Non-specific visual field defects	Mild dilated vs	Disc oedema, hyperemia, blurred	-	-	Bilateral	Young, obese, female, CT scan and CSF are normal
Hypertension	Mild decrease	Enlarged blind spot	Veins - dil. arterioles-attenuated	Disc oedema, hyperemia, blurred	+++ with cotton wool spots	+++ with macular star	Bilateral	Accelerated malignant hypertension
Diabetic papillopathy	Mild decrease	Central/centrocaecal scotoma	Marked telangiectasia of small disc vs	Hyperemia, pink disc	Flame shaped/dot blot	Diabetic macular oedema	Unilateral/bilateral	Small cup disc ratio (CDR), marked disc telangiectasia
C.R.V.O	Decrease mild - severe	++	Marked dilated veins	Hyperemia, pink	Extensive Hmn all over the retina	+++	Unilateral	Extensive retinal Hmn diagnostic
Non-arteritic ION	Decrease	Altitudinal defects (inferior)	Focal dilated vessels	Segmental disc oedema with pallor	+/-	+/-	Unilateral	Segmental/focal disc oedema, small CDR
Arteritic ION	Marked decrease	Altitudinal defects	Focal dilated vessels	Segmental disc oedema with pallor	++	++	Unilateral	Temporal headache, jaw pain, ESR, CRP elevated
Papillophlebitis	Mild decrease	Enlarged blind spot	Marked engorgement, vs sheathing	Pink, Hyperemia	Marked haemorrhage	+++	Unilateral	Central retinal vein inflammation at disc
Congenital disc abnormality	Decreased	Enlarged blind spot, varied	Pattern and distribution altered	Hyperemia, pallor, pigmentation	--	--	Bilateral, rarely unilateral	Since birth, poor vision, systemic abnormality
Compressive optic neuropathy	Decreased	+++	Engorged vs	Pink, opticociliary shunt vs	+/-	Choroidal folds, oedema +	Unilateral/bilateral	Proptosis, vision loss

CDR, cup disc ratio; CRP, C-reactive protein; CSF, cerebrospinal fluid; CRVO, central retinal vein obstruction; Dil, dilated; ESR, erythrocyte sedimentation rate; FA, fluorescein angiography; Hmn, haemorrhage; ION, ischemic optic neuropathy; R, Retinal veins; R, Hmn, retinal haemorrhage; R, Oedema, retinal oedema; Symm, symmetry; V, Fields, visual field defects; Vs, vessels.

with normal foveal reflex. Left eye revealed optic disc pallor without oedema (figure 2). Foveal reflex was normal.

INVESTIGATIONS

The patient was admitted for investigations and management. Blood pressure at presentation was controlled on medication, however blood sugars were raised. At presentation, the fasting blood glucose levels were 452 mg/dl and post prandial glucose levels were 497 mg/dl. Complete blood count was normal. Erythrocyte sedimentation rate was 12 at the end of 1 h. His renal and liver function tests were normal. CT scan done initially did not reveal any intracranial lesion and neurological examination was normal. Visual field charting done for the right eye revealed inferior altitudinal defect (figure 3). Visual field of the left eye could not be done due to poor vision.

DIFFERENTIAL DIAGNOSIS

The differential diagnosis in such a case would be bilateral papilloedema, anterior ischemic neuropathy, intracranial lesion, Foster Kennedy syndrome and hypertensive papilloedema.

Intracranial lesion and Foster Kennedy syndrome were ruled out due to a normal CT of the brain. The patient was diagnosed to have hypertension however; he also had unilateral loss of vision with no macular involvement. There was no significant arteriolar attenuation to attribute the disc oedema to high blood pressure.

Anterior ischemic optic neuropathy (AION) is the most plausible diagnosis in this case as there was sequential loss of vision of left eye followed by right eye 6 months later which is known to occur in AION. There was segmental pallid disc oedema of the left eye with predominant inferior altitudinal visual field defect which is classically seen in AION. The automated perimetry also shows few areas of reduced sensitivity in the superior half which can be due to extremely poor vision (20/400). A Goldman perimetry would have been better; however it was unavailable at our institute. Variety of field defects are known to occur in AION ranging from relative inferior altitudinal defect to total defects with preservation of central vision. The patient was a hypertensive and type 2 diabetic which are known risk factors for AION. The right eye disc oedema noted during the first episode was not confirmed by any other treating doctor and hence could be doubtful. There were no disc photographs to support the presence of simultaneous bilateral disc oedema at any point in time.

To summarise, our patient was a hypertensive and diabetic with painless loss of vision in left eye followed by painless loss of vision in right eye. There was segmental pallid optic disc oedema in right eye and optic disc pallor in left. A diagnosis of non-arteritic AION was made with left eye optic atrophy.

TREATMENT

The patient was started on insulin therapy dose (human insulin 30/70 combination 20 units before breakfast and 12 units before dinner) and tab glynase MF (glipizide 5 mg with metformin hydrochloride 500 mg) one tablet twice daily and continued on antihypertensive treatment (tab losartan and hydrochlor-thiazide). The blood sugar and blood pressure were controlled on treatment. Also combination of aspirin and clopidogrel was started.

OUTCOME AND FOLLOW-UP

After 4 weeks, the BCVA of right eye was 20/200 and left eye was counting fingers close to face.

DISCUSSION

Patients with optic disc oedema are seen by ophthalmologists, neurologists, physicians and residents doctors in ER. Thus, first we need to clarify the terminology used. Optic disc oedema is a descriptive term used to signify oedema of the optic nerve head. However papilloedema is optic disc oedema due to raised intracranial tension, thus it is a specific term indicating intracranial pathology.¹⁻³ Every optic disc oedema is not papilloedema.

Optic disc oedema due to various causes can be unilateral or bilateral. Bilateral disc oedema is frequently misdiagnosed as papilloedema and patients were subjected to various neuro-imaging modalities even though the clinical picture may be otherwise, ignoring relevant symptoms and signs. A diagnosis of papilloedema has been made for congenital abnormal optic disc, disc drusen, benign intracranial hypertension and optic neuritis.⁴⁻⁸ A condition called diabetic papillopathy seen in young diabetic patients is often misdiagnosed as papilloedema. Papilloedema should also be differentiated from pseudopapilloedema due to hypermetropia. The table included here will help us differentiate some of the common causes of optic disc oedema based on clinical reasoning (table 1).

Every case of optic disc oedema should be evaluated on its merit considering relevant symptoms, best corrected vision, clinical presentation and signs. Optic disc examination should be done by the attending doctor and findings may be confirmed by a neuro-ophthalmologist when in doubt. Disc photographs may be taken for comparison and relevant investigation should be done as deemed necessary. A team approach involving a neurologist and neuro-ophthalmologist would be best to diagnose complicated cases.

Learning points

- ▶ All cases of disc oedema are not papilloedema.
- ▶ A detailed clinical evaluation must be done before arriving at a diagnosis.
- ▶ CT is a non-invasive test to rule out intracranial SOL, however, may be normal in many diseases.
- ▶ Other systemic and local optic nerve pathologies must be kept in mind.

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