EVALUATION OF OPTIC DISC OEDEMA

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In our routine day to day practice, if we get a case of optic disc edema, by our self or by referral from our other colleagues of different discipline (Neurologist, internist, pediatrician or obstetrician) to opine as expert, to establish whether the case is a true or a pseudo papilledema, it always raises the forehead of both patient & the physician.

To clinch the diagnosis & establish the etiology is after all quite challenging. This can be made simple by step approach. What we are following in our clinic, summarized as follows:-

History Taking

A good history taken from the patient or from his/ her relative/accompanying person is one of the corner stone for establishing the diagnosis:-

a. Symptoms-

Enquire about the refractive status, any type of visual loss with respect to its laterality, transient obscuration of vision.

b. History of diplopia & its association intermittent/ constant with squinting, floater and clouding of visual field, any type of headache, vertigo & giddiness, vomiting (whether more in morning & projectile in nature, nature of vomitus).

c. Loss of Consciousness.

d. Note about whether there was premature birth (Bergmeister's Papilla)

d. Know about the history of diabetes (NIDDM/ IDDM), endocrine disorder (Thyroid & Pituitary), infectious diseases (TB & Syphilis), Auto immune disease like SLE, Blood dyscrarsia i.e. leukemia.

e. Any history of drug intake - amino glycoside (Tetracycline & Nalidixic Acid), Vitamins (especially Vit -A overdose), steroids, oral contraceptives.

Clinical Examination

- Note visual Acuity
- Do the refraction & note BCVA
- Examine the pupil before dilatation
- Do the red pin test.
- Check the ocular movement in all gazes.
- Do confrontation test with red hair/ hat pin for rough assessment of field
- Do ophthalmoscopy.

- High hypermetropic optic disc confuses with papillodema
- Red pin test gives evidence of decrease color contrast & saturation as in optic neuritis or optic N. compression.
- Abnormal ocular movement gives clue for palsy of 3rd, 4th or 6th CN and some typical nystagmoid movement give clue for lesion at specific site of brain.
- Pupil examination should always be done before dilated fundus examination. Check for anisocoria, abnormal pigmentation, RAPD, reaction to light, near & accommodation.

The fundoscopy is the most important part of examination of the Eye.

Disc ÕAlways evaluate the disc with +90 or +78 D lens, as it give binocular stereoscopic view.

1) Size - Normal disc is 1.5mm in diameter. Small disc causes crowding of axons of ganglion cell at its exit through lamina cribrosa. In nanophthalmus & in high hypermetropia, where the eye is relatively small but the number of axons remain same as the normal eye. These axons come out through a comparative small exit, gives false appearance of disc swelling.

2) shapeÕWhether it is vertically oval (Astigmatism causes blurring of the margin in either meridian of disc & alter the shape of the disc)

3) ColorÕ whether the disc is hyperemic, normal or pale. Count the capillaries on disc (Kastenbaum's sign- normally 10, Optic atrophy < 6, Hyperemic disc > 12).

4) MarginÕ Blurring is also seen in refractive error & media opacities.

5) CupÕ whether the central physiological cup is filled up or not. If so, it is due to odema or glial tissue proliferation. In case of poor cup margin, note the bending of the out coming vessels from disc.

   In case of small disc with blurred margin & poor cup contour, always examine the Sibling & parents, as the disc nature is inherited.

6) Vessels exiting from discÕ note the pulsation. 20% of normal population shows no pulsation. When present in blurred disc, it excludes papilloedema.

7) In chronic cases the disc may show optico-cilliary shunts and the appearance takes the shape of champagne cork.

Peri-palillary areaÕ

1. Flame shaped hemorrhages in retinal nerve fiber layers.
2. Vessels are buried in edematous nerve fiber layers, so are ill visible.
3. Cotton wool spots (Infarction of axon).
4. Paton's line (vertically oriented lines temporal to disc).
5. Radiating fold in ILM
6. Macular star

Fundus finding in a case of established papillodemaÕ

DiscÕ

Margin- Looks Blurred. Edematous blurring starts first at inferiorly then superiorly then nasally & finally temporally. (ISNT)

ColorÕ Hyperemic. This is due to dilatations of the capillaries on the disc.

CupÕfilled up, sometimes so severe that mushrooming of the disc occurs. Edema > 2 DD. Even > 6 DD sometimes.

VesselsÕStopped pulsating, dilated & engorged, may hemorrhages over the disc & paton's line are noticed.

Peri-papillary areaÕ

Hiding of vessels in edematous tissue

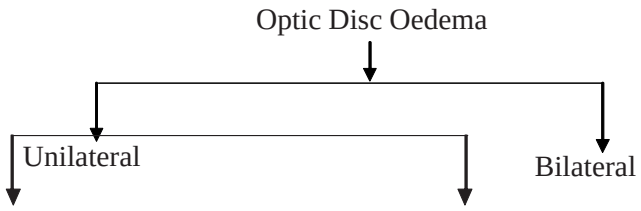
Flame shaped hemorrhages

Folding of ILM

Cotton Wool Spots

Fan shaped macular star (may be incomplete)

Causes of Papillodema



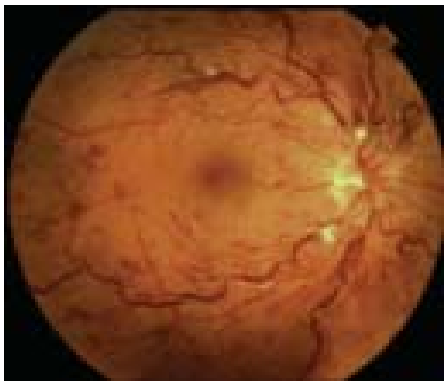
Orbital	Intraocular	Malignant Hypertension
Mass in muscle cone	Uveitis	SLE
Capillary/Cavernous haemangioma	Disseminated choroiditis	Drug toxicity
	Neuro-retinitis	ICSOL
Cysticercosis	VKH syndrome	Idiopathic
		Bilat compressive thyroid ophthalmopathy
Compression of ON-	Post-op-hypotony	Meningitis, Extra & sub dural haemorrage
Glioma of ON	Posterior scleritis	Brain Abscess
	CRVO	Benign intracranial hypertension
Thyroid ophthalmopathy	AION	
Orbital cellulites	Papillophlebitis in young	
Lesion of nearby sinuses e.g- Maxillary CA, Orbital invasion of Nasopharyngeal CA		
Pseudotumour e.g. orbital apex syndrome		

Unilateral disc edema with optic atrophy of other side seen in Frontal lobe tumor (Foster Kennedy Syndrome)

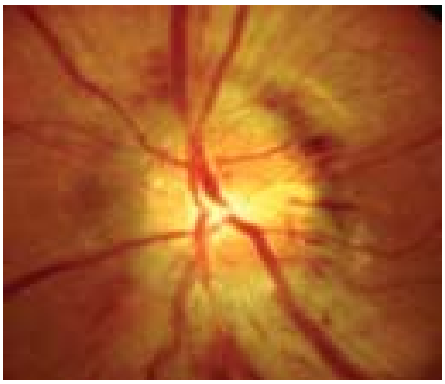
Two conditions mimic bilateral papilloedema without increased intracranial tension

.Leber's hereditary optic neuropathy

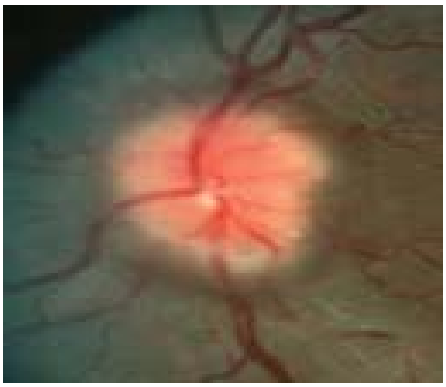
.Non arteritic ischemic optic neuropathy



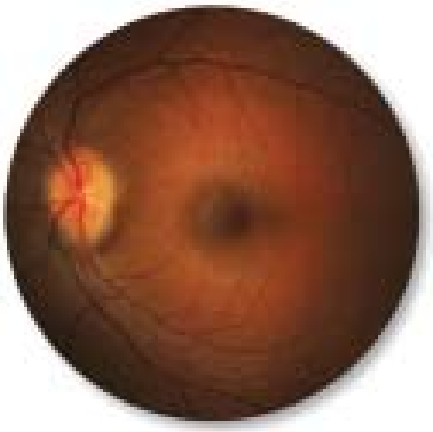
Papillodema in CRVO



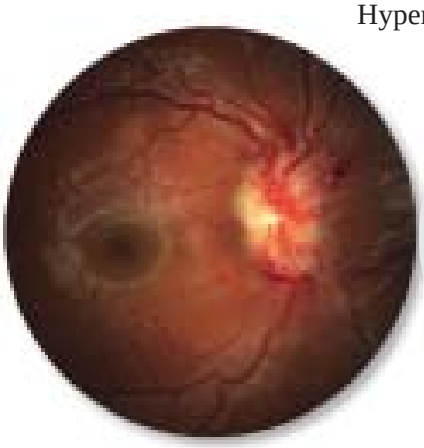
Diabetic Papillopathy



Papilloedema in Benign Intracranial Hypertension



Paton's Line



Papillodema in ICSOL

Investigation

Aim is to establish the diagnosis and to localize the site of lesion and thereby to tailor the approach for surgical or medicinal treatment.

1) Always do the systemic & neurological examination. Take the help & refer the case to other discipline personals like internist, neuro-physician/surgeon. Don't forget to measure BP (Malignant hypertension) & blood sugar level (Diabetic papillopathy)

2) Assess Visual fieldÕ

   a. Confrontation field test with red hair/hat pin

   b. Automated field test with Humphrey/ octopus (30 degree only)

   c. Goldman perimeter is better for field in neuro-ophthalmic cases.

   w Note for blind spot enlargement

   w Note homonymous hemianopic or quadrantonopic defects with congruousity of field. Whether these defects are respecting the vertical line or horizontal line!

If no automated perimeter available, take help of Bjerrum's screen.

3) Stereo photograph for documentation.

4) Fundus fluorescence AngiographyÕ It rules out pseudo papillodema. Here there is no capillary dilatation on disc and no hyper fluorescence as no leakage of dye. Drusen of disc (Auto-fluorescence & blurring of margin), high hypermetropia simulate papilloedema.

5) B-Scan-

   Posterior scleritis

   Burried disc drusen

6) CAT ScanÕ It has replaced the conventional radiography. Accurately localizes the lesion in respect of size, site and shape. This helps in tailoring the surgical approach.

7) MRIÕSpecially for Posterior fossa & brain stem lesions.

8) Other hematological & biochemical & immunological investigation should be planned according to suspicion-

e.g. - Gumma - for syphilis
Granulloma - for TB

ESR & C-reactive protein for Giant cell Arteritis
TWBC & peripheral smear for Leukemia and so on…

After above investigations and by ruling out the possibility of ICSOL, do Lumbar Puncture which is for both diagnostic & therapeutic for benign intracranial hypertension.

w(LP in ICSOL may cause herniation of brainstem leading to death)

Follow upÕ

After diagnosis & curative measures, the optic disc odema starts to regress. It takes 6-10 weeks for resolution. The result may be complete resolution of papillodema or the disc may become atrophic. So the close follow up is essential for any recurrence.

ReferencesÕ

1. The Wills Eye Manual: office &Emergency room diagnosis & treatment of eye Diseases. 5th edition.
2. Walsh & Hoyt's clinical Neuro-ophthalmlology.
3. Yanoff M. Duker J.S., Ophthalmology.