Study of radiological findings in papilledema

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Abstract

Background: Early recognition of papilledema and elevated ICP is of paramount importance for ensuring restoration of vision. Papilledema, frequently occurs in the setting of increased ICP and in a variety of medical conditions, including pseudotumorz, sinus thrombosis, intracerebral hemorrhage, frontal lobe neoplasms, and Chiari malformation. The primary role of imaging in the diagnosis of idiopathic intracranial hypertension (IIH) has been to exclude other conditions that can cause increased intracranial pressure (ICP) and papilledema. Material & Methods: The present study is a non randomized prospective case series being conducted in the 50 patients with disc edema/papilledema attending OPD and referred from other departments to DEPARTMENT OF OPHTHALMOLOGY, Gandhi Medical College and associated Hamidia Hospital, Bhopal from January 2013- December 2014. All patients underwent a complete medical evaluation including careful history taking, ophthalmic examination. Investigations includes MRI Scan was done in Radiology as well neurology reference was taken from Neurosurgery department, Hamidia Hospital, G.M.C. Bhopal. Result: In radiological study ICSOL is observed in 70.58% cases followed by 11.76% cases of sinusitis followed by 5.88% case each venous thrombosis,demyelination and infarction. Conclusion: Early recognition of papilledema and elevated ICP is of paramount importance for ensuring restoration of vision. Newer advanced MR imaging techniques such as fMRI and DTI may prove useful in the future to assess the potential effects of papilledema on retinal and visual pathway integrity.

Keywords: Idiopathic Intracranial Hypertension, Cerebral Venous Thrombosis, Venous Hypertension, MRI.

Introduction

The term papilledema should be strictly reserved for optic disc edema as a result of increased cerebrospinal fluid (CSF), which bears specific etiologic implications. The most important entity to consider in cases of increased intracranial pressure is a space occupying lesion of the brain. This is often done with diagnostic tools such as Magnetic Resonance Imaging (MRI) and/or Computerized Tomography (CT) scans in conjunction with a lumbar puncture (LP). Computerized Tomography has traditionally been the imaging study of choice because of its availability and lower cost per patient than MRI. However, MRI has emerged as the technically optimal imaging modality [1]. Over the past decade, numerous publications have emphasized the MRI findings seen in patients with increased intracranial pressure (ICP) [2-4].

Empty sellaturcica has been described as a classic sign of chronically elevated ICP [5-9]. Additionally, transverse sinus stenosis (TSS) [10,11], optic disc protrusion [12], flattening of the posterior globes [3,7], and prominence of the perioptic nerve CSF spaces [3,13-16], are also commonly reported in patients with increased ICP, particularly those with idiopathic intracranial hypertension (IIH). However, none of these signs are pathognomonic of IIH [17,18], and all have even been described in presumed normal subjects [10].

Papilledema has gained increasing interest in recent years among neuro-ophthalmologists as the result of several clinical studies demonstrating that it may have not only diagnostic potential as a measure of increased ICP but also therapeutic potential as a measure of disease severity and response to treatment[13]. Pathogenesis of Papilledema. Because all CSF spaces communicate freely, the pressure and composition of the
CSF is thought to be the same throughout the CNS. Consequently, researchers have based their theories and experiments on the assumption that the pressure in the SAS surrounding the ON is the same as that in the cerebral and spinal SAS. However, this assumption has not been proved, mainly due to the difficulty in obtaining accurate pressure measurements in the SAS of the ON in vivo.

In the SAS of the ON, CSF flows from the chiasmatic cistern through the canalicular portion and into the intraorbital portion of the ON, a space that becomes a cul de sac at the back of the globe [19]. In attempting to detect and diagnose papilledema as early as possible in patients, MR imaging is becoming a useful noninvasive method. Because MR imaging can provide gross visualization of the optic globe, ON, orbits, and optic tract [20] it is an ideal tool to study the details of papilledema. This review provides a brief outline of the common MR imaging findings of papilledema and its pathologic mechanisms.

**Material and Methods**

The present study is a non randomized prospective case series being conducted in the patients with disc edema/papilledema attending OPD and referred from other departments, to, DEPARTMENT OF OPHTHALMOLOGY, Gandhi Medical College and associated Hamidia Hospital, Bhopal from January 2013- December 2014.

**Inclusion Criteria:**
- Proven case of disc edema
- Proven case of papilledema

**Exclusion Criteria:**
- Cases of pseudopapilledema

All patients underwent a complete medical evaluation including careful history taking, ophthalmic examination, complete blood count, MRI scan, and CSF analysis (including opening pressure). Ocular examination consists of visual acuity measurement with Snellen’s chart, anterior segment examination using slit lamp biomicroscopy, applanation tonometry, stereoscopic fundus photography and visual fields evaluation using automated perimetry with the Humphrey 30-2 program. The degree of papilledema was graded using Frisen’s scheme [3,4]. Visual acuity, optic disc changes, and visual field defects were checked in all the patients during follow-up. Fundus evaluation with indirect ophthalmoscope and +90 D slit lamp examination.

In early papilledema we may get following fundus findings.

- Hyperemia of the disc
- Blurring of the disc margin
- Apparent forward protrusion of disc
- Blurring of the physiological cup
- Overfilling of the vein

In some early case of papilledema, haemorrhage and exudates may be present at some distance from the disc. Fully developed and late papilledema The physiological cup becomes partially or completely obliterated, the margin of the disc becomes definitely blurred, the surrounding retina may have grayish tinge and the vessels are seen to climb to attain the disc, the veins become engorged. Haemorrhage may appear as a linear streak on the disc or around it.

**Persistent papilledema:** The arteries are not any time narrowed because when the arteries exhibit narrowing atrophic changes in the disc invariably follow.

In all patients showing papilledema in fundus examination, Investigations include MRI and CT Scan were done in Radiology department as well neurology reference was taken from Neurosurgery department, Hamidia Hospital, G.M.C. Bhopal.

All patients were evaluated of papilloedemarequired a patient to undergone urgent neuroimaging to rule out an intracranial mass or dural sinus thrombosis. Although computerised axial tomography is certainly adequate in most instances, magnetic resonance imaging is quite effective in ruling out both a mass lesion as well as a potential dural sinus thrombosis. MR angiography is done in selected cases to investigate the possibility of a dural venous sinus thrombosis, infarction, haemorrhages.

**Statistical Analyses:** The analyses were largely descriptive, with means, standard deviations, and ranges reported for continuous variables and counts and percentages reported for categorical variables. Associations between continuous variables are described using either Pearson correlation coefficients or Spearman rank correlation coefficients, as appropriate.
Results

Table No.- 1: Relation between causes and age group

| Cause                      | 0-10 |  | 11-20 |  | 21-30 |  | 31-40 |  | 41-50 |  | 51-60 |  | Total |
|----------------------------|------|------|-------|------|-------|------|-------|------|-------|------|-------|------|-------|
|                            | No.  | %   | No.   | %   | No.   | %   | No.   | %   | No.   | %   | No.   | %   | No.   |
| Optic neuropathy           | 1    | 0   | 4     | 0   | 2     | 0   | 3     | 1   | 0     | 0   | 0     | 0   | 11    |
| Aion                       | 0    | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 3     | 0   | 3     |
| Brao                       | 0    | 0   | 0     | 0   | 0     | 0   | 0     | 1   | 0     | 0   | 0     | 0   | 1     |
| Icsol                      | 0    | 0   | 4     | 0   | 4     | 0   | 4     | 0   | 0     | 0   | 0     | 0   | 12    |
| Meningitis                 | 2    | 0   | 0     | 0   | 4     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 6     |
| Malignant hypertension     | 0    | 0   | 0     | 0   | 5     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 5     |
| Diabetes                   | 0    | 0   | 0     | 0   | 0     | 0   | 0     | 1   | 0     | 0   | 0     | 0   | 1     |
| Pseudo tumor cerebri       | 0    | 0   | 0     | 0   | 1     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 1     |
| Drug history               | 1    | 0   | 1     | 0   | 0     | 0   | 0     | 2   | 0     | 0   | 0     | 0   | 4     |
| Malaria                    | 1    | 0   | 2     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 3     |
| Anaemia                    | 0    | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 1     |
| Encephalopathy             | 0    | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 0     | 0   | 1     |
| Head injury                | 0    | 0   | 0     | 0   | 0     | 0   | 1     | 0   | 0     | 0   | 0     | 0   | 1     |
| Total                      | 6    | 12  | 12    | 24  | 16    | 32  | 8     | 16  | 5     | 10  | 3     | 6   | 50    |

In this study most common age group affected was 21-30 years in which 32% are observed and least common age group were 51-60yr in which only 6% case are observed. In this study out of 50 patient 30% patient were of local cause in which 22% cases were of optic neuropathy followed by 6% cases of AION in age group of 51-60 year followed by 2% case of BRAO in age group of 41-40year. Remaining 70 % patients had systemic cause in which, 24% cases of ICSOL, followed by 12% cases of meningitis, 10% cases of malignant hypertension, 8% cases of drug history, 6% cases of malaria and 2% case each of diabetes, pseudotumor cerebri, anaemia, encephalopathy and head injury.

Table 2: Radiological Finding

| Radiologic finding        | No of cases | % of cases |
|---------------------------|-------------|------------|
| Icsol                     | 11          | 64.70      |
| Venous thrombosis         | 2           | 11.76      |
| Demyelination             | 1           | 5.88       |
| Sinusitis                 | 2           | 11.78      |
| Infarction                | 1           | 5.88       |
| Total                     | 17          | 100        |

All patients OF papilledema underwent MRI SCAN, in which abnormalities were found in 34% of pateints.in which , ICSOL were observed in 70.58% cases followed by 11.78% cases of sinusitis,11.76% cases of venous thrombosis, 5.88% cases of venous thrombosis, and 1 % case of demyelination and infarction each.

Discussion

The primary role of brain imaging in papilledema is to exclude other pathologies causing intracranial hypertension. However, subtle radiologic findings suggestive of IIH have emerged with modern neuroimaging. This review provides a detailed of the imaging findings reported and discusses their possible roles in the pathophysiology and the diagnosis of papilledema. Specific neuroimaging findings may suggest long-standing IIH, including empty sella, flattening of the posterior globes, optic nerve head protrusion, distention of the optic nerve sheaths,
tortuosity of the optic nerve, cerebellar tonsillar herniation, meningoceles, CSF leaks, and transverse venous sinus stenosis [21]. A number of studies have used imaging techniques to investigate the anatomic changes of the optic nerve [22,23,24] or the many imaging techniques, MR imaging has been of particular interest because of its ability to provide gross visualization of the optic globe, ON, orbits, and optic tract. Additionally, MR imaging provides higher soft-tissue contrast and free section orientation capabilities compared with CT and appears to be more accurate in assessing the ON than sonography [25].

Despite these advantages, the ON has been technically difficult to image because of its small size: It is 0.4–0.6 cm in diameter within the orbit. T2-weighted FSE sequences with fat-suppression have been found to be optimal for visualizing the ONs and periorbital CSF [26,27]. Coronal image acquisition is optimal for visualizing the true dimensions of the ON and periorbital CSF relative to the surrounding sheath. The most commonly reported macroscopic findings in MR images of patients diagnosed with papilledema are the following: 1) enlargement of the ONs, 2) flattening of the posterior sclera, 3) protrusion of the optic papilla into the globe, and 4) tortuosity of the ON [28]. While researchers have investigated the relationship between elevated ICP and papilledema, they have also used ocular and ON abnormalities to diagnose elevated ICP. The ophthalmoscopic appearance of IIH is most often characterized by variable. Furthermore, papilledema may be asymmetric or unilateral, and the degree of ON head swelling is poorly correlated with ICP [29].

In the recent William F. Hoyt Lecture of the American Academy of Ophthalmology, Dr Jonathan Trobe posited that papilledema is only a reliable indicator of chronically high ICP because the development of papilledema tends to lag behind the rise in ICP. Trobe noted that fewer than 20% of patients examined within a few days of head trauma or ruptured aneurysm have papilledema and only 6% of patients with chronically high ICP lack papilledema [30]. On the other hand, intracranial hypertension can occur without the presence of papilledema [30]. Possibly, MR imaging could assist in the development of intracranial hypertension before the development of papilledema [31]. According to Hansen and Helmke [32], there is a correlation between the width of the ONs and increased ICP. A width of 0.5 mm in this location is considered abnormal [20]. This may occur because the ON is not as rigid as other intracranial meningeal structures and can thus react without volume changes of intracranial CSF spaces [31]. This may occur because the ON is not as rigid as other intracranial meningeal structures and can thus react without volume changes of intracranial CSF spaces [31]. Once the diagnosis of elevated ICP is established, the appearance of the discs and the severity of papilledema are commonly used as measures of disease severity and response to therapy. However, the degree of papilledema does not predict the severity of symptoms. Increased CSF pressure might produce different disc abnormalities depending on the normal size of the ON. Further studies [13] should lead to a better understanding of the mechanisms and augment our ability to detect papilledema on imaging and allow early intervention to maintain or restore vision. Similar study done by Passi et al [33] they studied: Noninvasive imaging of the ON is possible by using MR imaging, with a variety of findings occurring in the setting of papilledema, including flattening of the posterior sclera, protrusion of the optic disc, widening of the ONs, and tortuosity of the ON. Early recognition of papilledema and elevated ICP is of paramount importance for ensuring restoration of vision.

Maya et al [34] studied MRI findings of elevated intracranial pressure in cerebral venous thrombosis versus idiopathic intracranial hypertension with transverse sinus stenosis. Found that on 29 IIH patients (28 women, 19 black, median-age 28, median-body mass index, 34) had bilateral TSS. 31 CVT patients (19 women, 13 black, median-age 46, median-BMI 29) had thrombosis of the sagittal (3), sigmoid (3), cavernous (1), unilateral transverse (7), or multiple (16) sinuses or cortical veins (1). Empty/partially-empty sellae were more common in IIH (3/29 and 24/29) than in CVT patients (1/31 and 19/31) (p<0.001). Flattening of the globes and dilatation/tortuosity of the optic nerve sheaths were more common in IIH (20/29 and 18/29) than in CVT patients (13/31 and 5/31) (p<0.04). Papilledema simply means oedema of the optic disc, without reference to its underlying cause. It may be due to different pathological states, of which the most important ones are mentioned in Table I [35]. This article mainly concentrates on papilloedema due to raised intracranial pressure. In our All patients of papilledema underwent MRI SCAN, in which abnormalities were found in 34% of patieints, in which ICSOL were observed in 70.58% cases followed by 11.78% cases of sinusitis, 11.76% cases of venous thrombosis, 5.88% cases of venous thrombosis, and 1%
case of demyelination and infarction each. Rohr AC et al [17] Cranial venous outflow obstruction and ONS hydrops were the most valid signs indicating IH with a sensitivity of 94% and 92% and a specificity of 100% and 89%, respectively. Sensitivities and specificities were 56% and 97% for reduced pituitary height, 64% and 78% for flattening of the posterior sclera, 31% and 97% for widening of the superior ophthalmic veins, 33% and 100% for optic disc protrusion, 14% and 100% for optic nerve edema, and 6% and 100% for elongation of the optic nerve. At least 2 MR imaging findings could be demonstrated in each patient but in none of the controls. The number of positive MR imaging findings correlated with CSF pressure ($r = 0.62, P = .01$). The combination of cranial and orbital MR imaging and MRV can be highly sensitive and specific in the diagnosis of patients with IH. Causes of Optic Disc Oedema and Papilloedema [35]-

1. Increased intracranial pressure (tumour, haemorrhage, infarction, abscess, oedema, benign intracranial hypertension).

2. Inflammatory optic neuropathy (optic or retrobulbar neuritis)

3. Infiltrative optic neuropathy (sarcoidosis, leukaemia and other malignancies)

4. Optic nerve tumours (angioma, meningioma, childhood optic nerve glioma, malignant optic nerve glioma, metastatic carcinoma).

5. Compressive optic neuropathy (Grave’s disease, sphenoid wing meningioma).

6. Vasculopathies (anterior ischaemic optic neuropathy, central retinal vein occlusion, malignant hypertension).

7. Intra-ocular disease (posterior uveitis, posterior scleritis).

8. Venous obstruction (due to space occupying lesions in the orbit, cortico-cavernous fistula, intrathoracic venous obstruction as by neoplasms, aneurysm of aorta).

9. Conditions associated with a massive increase in the protein content of CSF (e.g., some cases of GuillainBarre syndrome and spinal tumours).

10. Pseudopapilloedema (optic disc drusen, hyperopia and other anomalies).

Neuroimaging is helpful in excluding other causes of raised intracranial tension like space-occupying lesions. Venous sinus thrombosis can present with severe headache from intracranial hypertension secondary to impairment of CSF reabsorption, which can clinically mimic IIH symptoms. Magnetic resonance imaging (MRI) of the head and orbit with intravenous contrast and magnetic resonance venography (MRV) are the modalities of choice to exclude any structural lesions prior to IIH diagnosis. Several radiologic signs are suggestive of IIH; however, none of them are pathognomonic for this condition [36]. Similar to our study bidot S et al [21] found that Specific neuroimaging findings may suggest long-standing IIH, including empty sella, flattening of the posterior globes, optic nerve head protrusion, distention of the optic nerve sheaths, tortuosities of the optic nerve, cerebellar tonsillar herniation, meningoceles, CSF leaks, and transverse venous sinus stenosis [21]. All pateints of papelledema underwent MRI SCAN, in which abnormalities were found in 34% of pateints, in which ICSOL were observed in 70.58% cases followed by 11.78% cases of sinusitis, 11.76% cases of venous thrombosis, 5.88% cases of venous thrombosis, and 1 % case of demyelination and infarction each. Similarly Agrawal et al [35] found systemic causes are more prominent than local causes, in which ICSOL are most common among all causes.

**Conclusion**

Papilledema represents a serious warning sign for elevated ICP and potential vision loss in a variety of clinical settings. MR imaging may facilitate its detection and demonstrate changes of elevated ICP well before the appearance of papilledema on fundoscopic examination. Although the mechanisms causing papilledema and its associated signs are not entirely clear, the role for noninvasive imaging in this clinical condition is evident. Future advances in DTI, fMRI of the retina, and high-resolution MR imaging hold the promise of demonstrating the effects of papilledema on the visual pathway in patients.

**Funding:** Nil

**Conflict of interest:** None.

**Permission of IRB:** Yes

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