

# Optic Nerve Diseases and its Systemic Associations

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**Purpose:** To determine optic nerve diseases and its systemic associations.

**Study Design:** Descriptive cross sectional study.

**Place and Duration of Study:** Eye departments of Lady Reading Hospital Peshawar and Khyber Teaching Hospital Peshawar from Jan 2015 to Oct 2015.

**Material and Methods:** A total of 44 patients were examined. Detailed history was taken from every patient after which complete ocular examination including recording of visual acuity, checking pupillary reactions and fundoscopy with special attention to optic nerve head appearance and recording of color vision and light brightness sensitivity was carried out. Specific ophthalmic and systemic investigations were performed.

**Results:** Seven (15.91%) patients had NAION, 6 (13.64%) had demyelinating optic neuritis, 2 (4.54%) had toxic optic neuropathies, 2(4.54%) had nutritional optic neuropathy, 3 (6.82%) had pituitary macroadenoma and 3 (6.82%) had benign intracranial hypertension, 2 (4.54%) had arteritic anterior ischemic optic neuropathy, 2 (4.54%) Pseudo-Foster Kennedy syndrome, 1 (2.27%) had paraneoplastic syndrome, 1 (2.27%) had superior saggital sinus thrombosis, 1 (2.27%) had occipital lobe infarct, 1 (2.27%) had brain metastasis, 1 (2.27%) had craniopharyngioma, 1 (2.27%) had bilateral thalamic ischemia and 1 (2.27%) had hydrocephalus.

**Conclusion:** Patients presenting with optic nerve diseases may have serious systemic associations so for accurate diagnosis and management every patient presenting with optic nerve disease must be properly evaluated.

**Key words:** Optic nerve, optic neuritis, optic neuropathy.

One of the frequent causes of visual loss encountered by ophthalmologists is optic neuropathy<sup>1</sup>. Clinically, it can appear as an isolated entity due to local pathologies of the optic nerve or associated to a variety of systemic illnesses<sup>2</sup>.

Optic neuropathy may be unilateral or bilateral and usually presents with swelling of the optic disc or atrophic optic disc. However it is not uncommon for an optic nerve disease that optic nerve head appear clinically normal but it may cause other signs of optic nerve damage such as decreased visual acuity and defective color vision or light brightness sensitivity or presence of relative afferent papillary defect<sup>3</sup>.

Optic neuropathy has a number of local and underlying systemic causes including ischaemia, demyelinating disease, multiple sclerosis, systemic lupus erythematosus (SLE), sarcoidosis, Behçet's disease, vasculitis, and several infections including lyme disease, syphilis and cat scratch fever<sup>2</sup>.

Recognition of the underlying cause can not only change the visual prognosis but also the neurological consequences. Thus ophthalmologist should therefore be familiar not only with the various entities that can cause optic neuropathy but also should have knowledge of relative systemic investigations to diagnose a systemic illness which may be the cause for

optic nerve damage<sup>4</sup>. In most cases, careful clinical evaluation and appropriate investigations, can lead to a specific diagnosis<sup>5</sup>.

Similarly as the optic nerve dysfunction may be the presenting sign of a systemic illness, knowledge of clinical evaluation of the optic nerve and appropriate and relative investigations is also important for physicians because physicians may be the first person to encounter such patient and if misdiagnosed this entity can end up in blindness and optic atrophy apart from systemic sequel<sup>4</sup>.

Purpose of our study was to find out various local as well as underlying systemic causes giving rise to optic nerve damage and to emphasize the significance of early diagnosis and hence timely management with the help of clinical signs and relative ophthalmic as well as systemic investigations which can not only prevent blindness but also help diagnosis and management of underlying systemic disease.

## MATERIAL AND METHODS

It was a descriptive cross sectional study carried out at department of ophthalmology of Lady Reading Hospital Peshawar and Khyber Teaching Hospital Peshawar from Jan 2015 to October 2015. A total of 44 patients including males and females were examined. Sample size was calculated using 95% Confidence interval and 10% margin of error, under WHO sample size estimation. All patients presenting with sudden or gradual loss of vision with optic nerve involvement evident by decrease vision, presence of afferent pupillary defect, defective color vision, reduced light brightness sensitivity and contrast sensitivity and optic nerve appearance, were included in the study. Patients with all other causes of decreased vision with or without presence of afferent pupillary defect e.g. diabetic and hypertensive retinopathy, central retinal vein occlusion, central retinal artery occlusion and retinal detachment were excluded from the study. Detailed history was taken from every patient after which complete ocular examination including recording of visual acuity, pupillary reactions, intraocular pressure, recording of color vision and light brightness sensitivity and fundus examination with special attention to optic nerve head appearance was carried out for every patient. Specific ophthalmic investigations like automated visual field analysis (Humphrey) and Optical Coherence Tomography (OCT) (Optic Nerve Protocol) were carried out where needed. Systemic investigations in collaboration with

physicians were performed for individual cases based upon their history, ophthalmic and systemic examination.

All the analysis was done in SPSS version 20.0. For categorical variables like gender, and local and systemic associations, frequencies and percentages were calculated. For numeric variables like age, mean  $\pm$  standard deviation was calculated. All the results were presented in the form of tables.

## RESULTS

A total of 44 patients were included in this study. Age distribution is shown in table 1. Mean age was 44.09 years with SD  $\pm$  17.47. Gender distribution was analyzed as n = 24 (54.54%) of patients were males and n = 20 (45.46%) were females.

**Table 1:** Age Distribution.

Age	Frequency	Percentage
10 – 20 Years	2	4.54%
21 – 30 Years	9	20.46%
31 – 40 Years	12	27.28%
41 – 50 Years	4	9.10%
51 – 60 Years	5	11.36%
61 – 70 Years	10	22.72%
71 – 80 Years	2	4.54%
<b>Total</b>	<b>44</b>	<b>100%</b>

Mean age was 44.09 years with SD  $\pm$  17.47

Out of 44 patients, 25 (56.81%) patients had local optic nerve pathology resulting in either optic disc swelling or optic disc atrophy. While 19 (43.19%) patients had underlying systemic illness resulting in either optic disc swelling, papilledema or optic disc atrophy.

Leading cause of local optic disc pathologies resulting in optic disc swelling was non arteritic anterior ischemic optic neuropathy (NAION) 7 (15.91%), followed by demyelinating optic neuritis 6 (13.64%). A complete breakdown of different local causes is given in Table 2.

Local optic disc pathologies resulting in pale disc included; 2 (4.54%) patients had anterior ischemic

**Table 2:** Local Optic Disc Pathologies Causing Swollen Disc.

Disease	Frequency	Percentage	Presentation
Non arteritic anterior ischemic optic neuropathy	7	15.91%	Swollen disc
Demyelinating optic neuritis	6	13.64%	Swollen disc
Optic neuritis	2	4.54%	Swollen disc
Arteritic anterior ischemic optic neuropathy	2	4.54%	Swollen disc
Pseudo-Foster Kennedy syndrome	2	4.54%	Rt swollen and Lt pale disc
Optic nerve drusen	1	2.27%	Swollen disc
<b>Total</b>	<b>20</b>	<b>45.46%</b>	

**Table 3:** Systemic Associations of Bilateral swollen Discs/ Papilloedema.

Disease	Frequency	Percentage	Presentation
Benign intracranial hypertension	3	6.82%	Bilateral swollen discs (Papilloedema)
Superior sagittal sinus thrombosis	1	2.27%	Bilateral swollen discs (Papilloedema)
Brain metastasis	1	2.27%	Bilateral swollen discs (Papilloedema)
Known case of chronic renal failure	1	2.27%	Bilateral swollen discs
<b>Total</b>	<b>6</b>	<b>13.64%</b>	

**Table 4:** Systemic Associations of Bilateral pale Discs.

Disease	Frequency	Percentage	Presentation
Pituitary macroadenoma	3	6.82%	Bilateral temporal disc pallor
Nutritional optic neuropathy due to Vit B12 deficiency	2	4.54%	Bilateral temporal disc pallor
Nutritional optic neuropathy due to tobacco amblyopia	1	2.27%	Bilateral temporal disc pallor
Occipital lobe infarct	1	2.27%	Bilateral temporal disc pallor
Craniopharyngioma	1	2.27%	Bilateral pale discs
Bilateral thalamic ischemia	1	2.27%	Bilateral pale discs
Hydrocephalus	1	2.27%	Bilateral pale discs
<b>Total</b>	<b>10</b>	<b>22.72%</b>	

optic neuropathy, 2 (4.54%) had traumatic optic neuropathy, while 1 (2.27%) patient with pale disc was undiagnosed.

3 (6.82%) patients with underlying systemic diseases resulted in swollen disc; 1 (2.27%) patients had toxic optic neuropathy due anti-tuberculous

drugs, 1 (2.27%) had paraneoplastic syndrome secondary to squamous cell carcinoma of lung and 1 (2.27%) had neurosarcoïd.

Leading causes for patients with underlying systemic diseases resulting in bilateral swollen discs or papilloedema included; 3 (6.82%) benign intracranial

hypertension, followed by superior saggital sinus thrombosis 1 (2.27%), and brain metastasis 1 (2.27%). Complete breakdown of all the systemic causes has been shown in Table 3.

Patients with underlying systemic diseases resulting in pale discs included; 3 (6.82%) had pituitary macroadenoma, 2 (4.54%) had nutritional optic neuropathy due to vitamin B12 deficiency. Further details of this group are given in table 4.

## DISCUSSION

A variety of intrinsic, intraorbital, intracranial, or systemic diseases can lead to optic nerve damage<sup>1</sup>. Misdiagnosis of optic nerve diseases is not uncommon and can lead to sight as well as life threatening conditions<sup>6</sup>.

Reduced blood flow to the optic nerve's ganglion cells can lead to ischemic optic neuropathy which may be either non-arteritic (NAION) or arteritic (AION). NAION may result from a variety of underlying systemic conditions while AION is caused by giant cell arteritis (GCA)<sup>7</sup>. Most common form of ischemic optic neuropathy is NAION. Risk factors include hypertension, diabetes, hyperlipidemia, atherosclerosis, nocturnal hypotension, sleep apnea, certain medications and small discs. Behbehani R in his study on ischemic optic neuropathies commented on the appearance of the optic disc<sup>4</sup>. According to him the optic disc is usually swollen in non-demyelinating optic neuritis and NAION. In addition, in NAION, disc swelling can be sectoral or diffuse and associated with peripapillary hemorrhages. A small cup-to-disc ratio is seen in the fellow eye. While patients with AION due to GCA shows diffuse "chalky white" swelling of the disc with cotton wool spots<sup>4</sup>. In addition patients with NAION have afferent pupillary defect and corresponding visual field loss<sup>8</sup>. Although any type of visual field defect can occur with any type of optic neuropathy, in ischemic optic neuropathies altitudinal visual field defects are more common<sup>9</sup>. In our study the criteria we used for diagnosing non-arteritic anterior ishchemic optic neuropathy (NAION) was relevant clinical history, decreased visual acuity, presence of relative afferent pupillary defect, diffuse or sectoral optic nerve head swelling consistent with non arteritic anterior ischemic optic neuropathy and altitudinal field defect on Humphery's visual field. All patients with non arteritic anterior ischemic optic neuropathy (NAION) in our study were uncontrolled diabetics with hyperlipidemia. Humphrey's visual

fields showed altitudinal field defect in four of these patients.

Giant cell arteritis (GCA) should strongly be considered in patients older than 60 years with features of ischemic optic neuropathy<sup>10</sup>. Patients with GCA typically experience headache, scalp tenderness, jaw claudication, weight loss, fever and malaise. Complete blood count, erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) should always be performed in all such cases. ESR and CRP when combined increases the specificity (97.0%) and sensitivity (99%) for diagnosis. Thrombocytosis is also a positive finding in patients with GCA<sup>7</sup>. Our criteria for diagnosing arteritic anterior ischemic optic neuropathy was positive clinical history with special emphasis to jaw claudication, headache, scalp tenderness and weight loss, reduced visual acuity, presence of afferent pupillary defect, edematous optic nerve head consistent with arteritic anterior ischemic optic neuropathy, raised ESR and C- reactive proteins and histological confirmation of giant cell arteritis on temporal artery biopsy. In our study both patients with arteritic anterior ischemic optic neuropathy had raised ESR and C- reactive proteins and temporal artery biopsy revealed calcification.

The only patient of Pseudo-Foster Kennedy syndrome in our study had optic atrophy on one side and swollen disc on other side with altitudinal field defect on Humphery's visual fields on the side of swollen disc with normal MRI brain and orbits. Optic neuritis refers to an infective, inflammatory, or demyelinating process affecting the optic nerve. It usually presents in the second to fourth decades of life<sup>11</sup>. Most common cause of optic neuritis is Multiple sclerosis (MS)<sup>12</sup>. It usually presents with acute unilateral vision loss, pain on eye movement, presence of afferent pupillary defect and visual field defects. Optic nerve head may be swollen in the acute stage but if the optic nerve inflammation is retrobulbar than appearance of the optic nerve head is unremarkable. Optic nerve head may show signs of pallor when the acute stage subsides<sup>11</sup>. Behbehani R showed in his study that in retrobulbar demyelinating optic neuritis, optic disc is normal in 65% of cases and even if optic disc is swollen is it is usually diffuse and of mild degree<sup>4</sup>. Presence of severe optic disc swelling with peripapillary hemorrhages and exudates should point to an alternative diagnosis such as non arteritic AION or infiltrative optic neuropathy<sup>4</sup>. Our criteria for diagnosing demyelinating optic neuritis was positive clinical history, reduced visual acuity, presence of

relative afferent papillary defect, color vision and light brightness sensitivity defect, visual field defect and positive MRI findings. In our study all patients with demyelinating optic neuritis had positive MRI report of high signal intensity lesions in the intra orbital portion of optic nerves in addition to other positive finding of optic nerve damage. Two patients in our study with non demyelinating optic neuritis were diagnosed on clinical basis with reduced visual acuity, color vision and light brightness sensitivity defect, presence of relative afferent papillary defect and swollen optic disc with normal investigations.

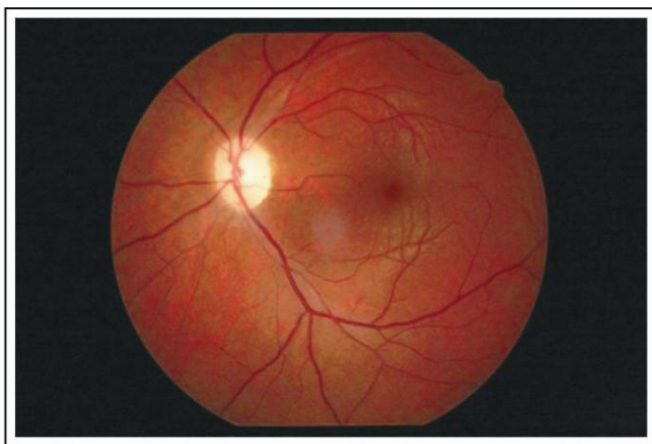
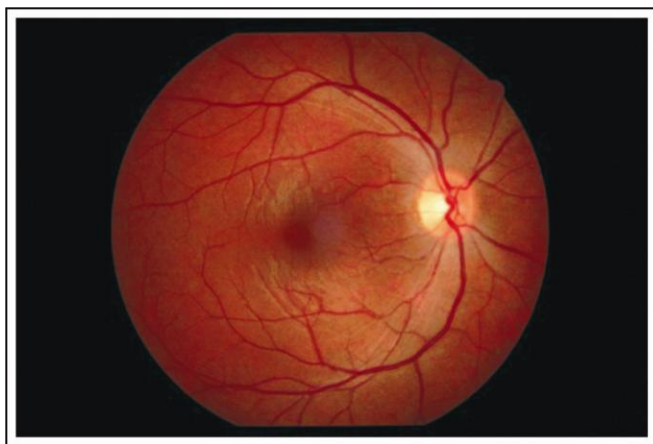
Optic nerve head drusen consists of calcific hyaline-like material within the optic nerve head substance. At an early age these are called as "buried drusen" as these are not usually visible. At later age these enlarge and come closer to the surface of optic nerve head and become more visible. B-scan show high acoustic reflectivity due calcific deposits and is the most reliable method for diagnosis<sup>13</sup>. In our study patient with optic nerve drusen presented with bilateral crowded disc resembling papilloedema but had positive finding on B-scan of high acoustic reflectivity.

Long-standing optic nerve damage such as caused by nutritional or toxic optic neuropathies, compressive, traumatic or hereditary optic neuropathies can give rise to a pale optic disc. This can also occur when an acute inflammatory or ischemic stage of optic neuropathy subsides<sup>14</sup>. Toxic or nutritional and hereditary optic neuropathies selectively affecting the papillo-macular bundle can give rise to temporal optic disc pallor as mentioned by Behbehani R in his study<sup>4</sup>.

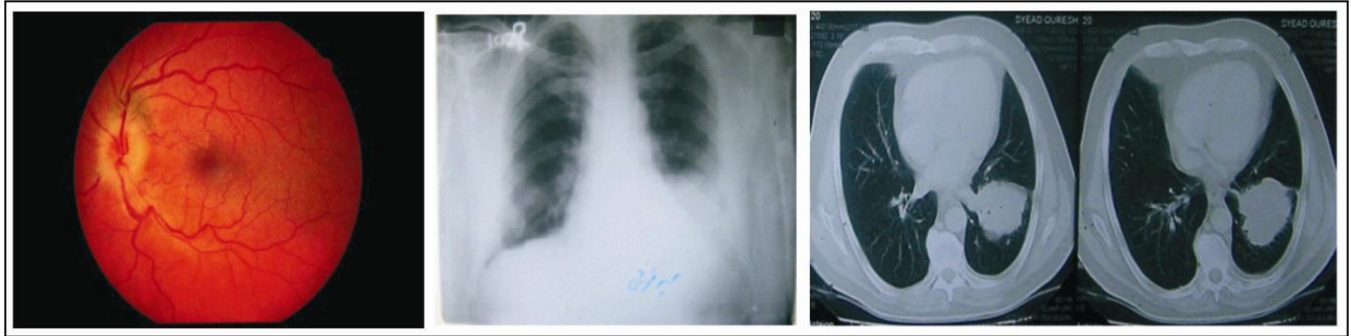
Patients with traumatic optic neuropathy usually

have suffered facial or orbital trauma. Main clue to the diagnosis is presence of RAPD. To detect fractures of the optic canal, and to rule out orbital hemorrhage, CT scan orbit is the investigation of choice<sup>15</sup>. In our study both the patients with traumatic optic neuropathy presented to us very late due to other injuries occurred during road traffic accident. Both had decreased visual acuity, presence of relative afferent papillary defect and optic disc pallor on the affected side with normal CT scan brain and orbits. Figure 1 shows fundus photos of patient with traumatic optic neuropathy.

Different drugs and nutritional deficiencies can also cause Optic nerve damage. Top on the list are ethambutol and amiodarone. Tobacco, methanol and ethanol are also in the list<sup>16</sup>. Tobacco-alcohol amblyopia is the consequence of toxic effect of tobacco superimposed on nutritional deficiency state.<sup>17</sup> Nutritional optic neuropathy mainly occurs due to vitamin deficiency. Deficiency of thiamine (vitamin B1), riboflavin (vitamin B2), niacin (vitamin B3), pyridoxine (vitamin B6), cyanocobalamin (vitamin B12) have all been implicated<sup>16</sup>. In our study among patients with toxic and nutritional optic neuropathies one was heavy smoker with tobacco amblyopia and had bilateral temporal disc pallor with central scotoma on Humphrey's visual fields, one had toxic optic neuropathy resulting from anti-tuberculous drugs and two had nutritional optic neuropathy due to vitamin B12 deficiency. Our criteria for diagnosing nutritional optic neuropathies were reduced visual acuity, temporal or complete disc pallor and serum B<sub>12</sub> level below normal limits. Both our patients with nutritional optic neuropathy due to vitamin B12 deficiency were low in B12 level with bilateral temporal disc pallor.



**Figure 1:** Patient with traumatic optic neuropathy showing left pale disc.

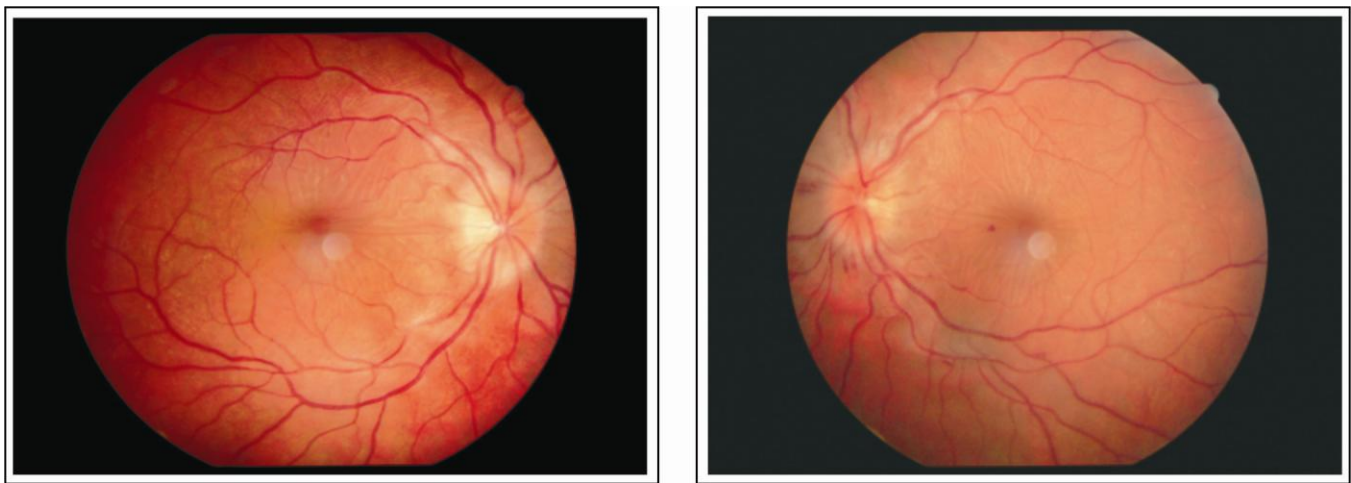


Swollen disc

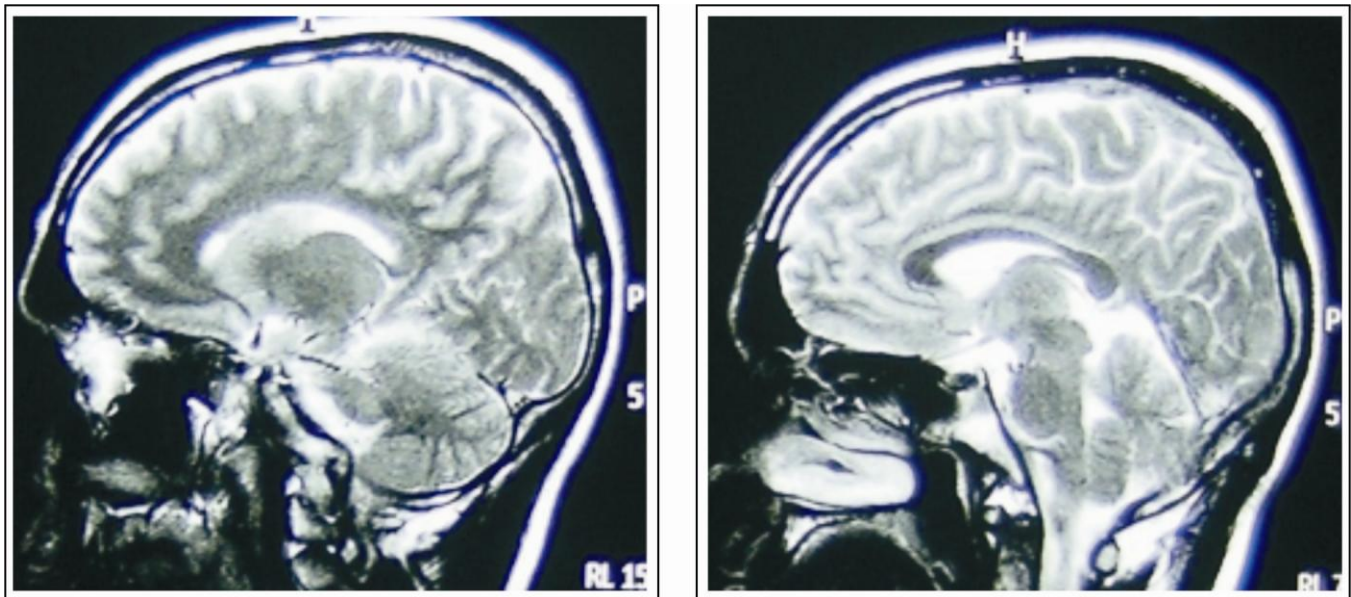
Left lower lobe consolidation

MRI chest showing growth in left lung

**Figure 2:** Patient with paraneoplastic syndrome.



Bilateral swollen discs (Papilloedema)



MRI showing non visualization of superior saggital sinus.

**Figure 3:** Patient with superior saggital sinus thrombosis.

Both small cell and non-small cell carcinoma of the lungs can cause optic neuropathy as a result of paraneoplastic syndrome. Patients usually present with gradual decrease of vision associated with bilateral disc swelling before the signs of the systemic malignancy are evident<sup>18</sup>. In our study patient with paraneoplastic syndrome presented with left swollen disc. Findings were not consistent with local optic disc pathologies so we decided to perform systemic evaluation in collaboration with our physician colleagues. On routine investigations x-ray chest showed left lower lobe consolidation. MRI chest was advised which showed growth in left lung for which biopsy was performed which proved to be squamous cell carcinoma of lung, figure 2.

In our study criteria for diagnosing benign intracranial hypertension was positive clinical history of headache, presence of papilloedema, normal MRI with absence of intracranial mass lesion or enlargement of ventricles, raised opening pressure of CSF on lumbar puncture or clinical trial of acetazolamide with improvement of sign and symptoms in those patients who were unwilling for lumbar puncture. Out of three patients, in our study, two had raised opening pressure of CSF on lumbar puncture while one patient was advised lumbar puncture but was reluctant. Diagnosis in this case was made on clinical basis and showed improvement with oral acetazolamide.

Three patients presented to us with papilloedema, including superior sagittal sinus thrombosis, brain metastasis and one undiagnosed patient who had chronic renal failure and investigations were incomplete due to lack of follow-up. Figure 3 shows papilloedema secondary to superior sagittal sinus thrombosis with positive MRI finding of non visualization of superior sagittal sinus.

Compressive optic neuropathy usually results in gradual and progressive visual loss<sup>19</sup>. Common causes include pituitary adenomas, meningiomas, intracranial aneurysms, craniopharyngiomas and gliomas of the anterior visual pathway<sup>20</sup>. Although knowledge of visual field defects leads in the localization of the lesion, MRI of the brain and orbit is require for accurate diagnosis<sup>19</sup>. In our study seven patients presented with progressive visual loss and bilateral disc pallor which included three cases of pituitary macroadenoma and one each; occipital lobe infarcts, craniopharyngioma, bilateral thalamic ischemia and hydrocephalus. All these cases had

positive findings on humphrey's visual fields and MRI brain and orbits.

## CONCLUSION

Optic nerve diseases may have serious systemic associations and whether localized or associated with systemic illnesses, it has serious ophthalmic and systemic sequel so every patient with optic nerve disease must be properly examined and proper investigations must be performed for accurate diagnosis and management.

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