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Ethambutol has been used to treat TB since the 1960s. The original formulation was a racemic mixture. However, when it was discovered that the L-form was predominantly responsible for its toxicity, and the D-form for its therapeutic effects, the L-form was withdrawn. Despite this, cases of irreversible visual loss have been reported in the literature. We report 3 cases of ethambutol-associated optic neuropathy, and recommend that ethambutol be used with caution, proper patient education, and proper ophthalmologic monitoring. This consists of baseline (pre-treatment) best corrected visual acuity, color vision, and Humphrey visual field (HVF). Subsequent monitoring in asymptomatic patients is also important. We also postulate that in cases of ethambutol associated chiasmopathy, ethambutol initially causes an optic neuropathy, and later progresses to involve the optic chiasm.

**MATERIALS AND METHODS**

A prospective, randomized, controlled study included 120 eyes of 60 patients divided into ethambutol A and control (who did not receive) groups B. Patients were evaluated in terms of BCVA, visual fields (VF), contrast sensitivity (CS), color vision, fundus and pupillary reaction before starting therapy and monthly till 2 months of cessation. Therapy was stopped in case of drop in VA by 2 lines on Snellens chart, fundoscopic changes of optic neuritis, drop in CS by 0.3 log units, VF changes, subjective blurring of vision.

**Case 1**

A 60 yr old male, diabetic and hypertensive, diagnosed with pulmonary TB, and had been taking rifampicin, isoniazid and ethambutol for the past 13 months, came with history of persisting poor vision after bilateral cataract surgery. Examination revealed visual acuity OD-FC 3m and OS 6/60 with no improvement through a pinhole. There was no RAPD. Color vision (Ishihara) was abnormal bilaterally; only the test plate was seen.

Confrontation visual field testing was normal by finger counting, but revealed red desaturation temporally when tested with the red mydriacyl cap. There was bilateral optic disc pallor. Contrast sensitivity dropped by 0.3 log units.
Blood tests for autoimmune diseases (ESR), ANA titres, ds DNA and syphilis (VDRL) were negative. The HVF showed bitemporal hemianopia and CT brain was normal. There was no SOL in the para-sellar region. He was diagnosed with ethambutol-associated optic neuropathy and the medication was stopped immediately.

He was followed up for 18 months. There was initial worsening of his visual fields over 2 months despite stopping ethambutol. On his last visit, visual acuity was OD 6/60 and OS 6/18, improving on looking through a pinhole to 6/24 and 6/12 respectively.

Case 2
A 45 yr old female was diagnosed with tuberculous arthritis of her knee, for which she was started on rifampicin, isoniazid, ethambutol and pyrazinamide. Baseline visual acuity was normal and color vision was normal. She was on regular review with the ophthalmologist, with visual acuities of 6/6 in both eyes. 9 months after the commencement of TB treatment, VA both eyes 6/6; her optic discs were pink. HVF showed scattered defects. Contrast sensitivity was normal. This was communicated to her attending physician and ethambutol was stopped. On review 2 months later, visual acuity had worsened; OD 6/60 in the right eye and OS FC 5 m with no improvement on pinhole. Her optic discs now showed temporal pallor. HVF showed progression with a suggestion of bitemporal field loss. A CT of the brain, performed as a result of the visual field progression, was normal. She has now been followed up for 24 months. Visual acuity remains poor at OD 6/36 and 6/18 on the left eye with bilateral pale discs.

Case 3
A 70 yr old female was started on ethambutol and isoniazid for pulmonary TB. She had complaints of floaters, blurring of vision bilaterally 4 months after starting the AKT. Examination revealed VA of 6/6 bilaterally, normal color vision and bilateral pink optic discs. There was no evidence of toxic optic neuropathy at that time. The HVF showed a junctional scotoma. Drop in contrast sensitivity was noted. Ethambutol was stopped immediately MRI of the brain was normal. She was diagnosed with ethambutol-associated optic chiasmopathy. After cessation of ethambutol however, her visual acuity continued to worsen. Her visual field and CS improved slowly over the next 9 months. On her last visit, visual acuity was 6/6 bilaterally. CS and HVF had normalised.

RESULTS
Patients on ethambutol showed deterioration of VA(1), VF(3), CS(2). Maximum recovery occurred in 6 -8 weeks after stopping therapy.
In conclusion Gr A toxicity was seen in 3(10%). VF in all 3(10%) and CS in 2(6%) of patients. VF and CS are more sensitive than VA for early detection and more resistant to recovery. Group B patients did not show any changes in any parameters.

**DISCUSSION**

Ethambutol-associated optic neuropathy is an established “ocular” drug complication. These cases remind us that ethambutol usage is associated with a real risk of toxic optic neuropathy and permanent visual loss. Although isoniazid may also be responsible, ethambutol-associated optic neuropathy is more widely recognized. In addition, ethambutol toxicity has frequently been reported to present with central or centroceceal scotomas. However, bitemporal field defects have occasionally been reported in the early stages of ethambutol-associated optic chiasmopathy, the optic nerves are initially affected. If ethambutol is continued, the damage spreads to the anterior chiasm and later, to the whole chiasm resulting in a bitemporal hemianopia.

However, centrocecal scotomas can be confused with bitemporal hemianopias as reported.

Due to the nature of the tuberculous bacilli, it would be difficult to eradicate the disease altogether and the use of ethambutol will most likely continue. How we can make it safer for our patients. This requires the patient, the physician and the ophthalmologist to work closely together. The first step is to identify patients in whom ethambutol is relatively contraindicated. These include patients who are unlikely to notice or describe visual symptoms, such as patients with dementia, mental retardation and children. Others include patients with pre-existing ophthalmological diseases with poor baseline vision. These patients should not be treated with ethambutol.

The second step is to educate all patients treated with ethambutol on its side effects. Ethambutol causes loss of visual acuity, color vision and visual field. The occurrence of ocular toxicity is dose related, loss of vision most likely to occur in patients receiving 25 mg/kg/day or more. However, vision loss has been documented in approximately 1% of patients receiving the recommended therapeutic dose of 15 to 25 mg/kg/day. This rarely occurs before the patients have been on treatment for 2 months, with 7 months being the average. 2 Patients with impaired renal function from renal tuberculosis may be more prone to ethambutol-associated optic neuropathy; perhaps because ethambutol depends on the kidneys for excretion. It is also important for the clinician to be aware that there are reports of rapid onset, severe, bilateral visual loss despite treatment with therapeutic doses of ethambutol. Patients taking ethambutol should be instructed to discontinue the drug immediately at the onset of any visual symptoms and seek medical consult.
Thirdly, all patients commencing treatment with ethambutol should have a baseline (pretreatment) ophthalmological examination. This comprises bestcorrected visual acuity, color vision and visual field.

These parameters are usually monitored periodically (every 1 to 3 months) during the treatment of asymptomatic patients. However, there is no consensus regarding the specific visual test and testing intervals appropriate for monitoring asymptomatic patients during treatment.19

In summary, ethambutol is associated with a real risk of permanent visual loss. In cases of ethambutol-associated chiasmopathy, ethambutol initially affects the optic nerve and subsequently, the optic chiasm, and can result in a bitemporal hemianopia. All physicians prescribing the drug should be aware of this and the drug should be used with proper patient education and ophthalmological monitoring.

Clinical Study of Ocular Manifestations in Head Trauma

Dr. Ashis Ghosh, Dr. (Col) R. P. Gupta, Dr. Renu Magdum, Dr. Kanchan Sawant, Dr. (Col) S Patra

Neuro-ophthalmological deficits commonly follow head trauma. The afferent and efferent visual systems are susceptible to injury from a variety of mechanisms. These patients can be a diagnostic and therapeutic challenge, in large part secondary to the frequently vague nature of their visual complaints and their coexistent neurologic deficits1. Head trauma may also affect the pupillo-motor pathways, the cranial nerves and the internuclear and supra-nuclear gaze pathways. About 1/3rd to 1/2 of patients of head trauma have abnormal neuro-ophthalmological findings.

The World Health Organization (WHO) has projected that by 2020, road accidents will be a major killer in India accounting for 546,000 deaths and 15,314,000 disability-adjusted life years lost.2,3

Traumatic brain injury (TBI) continues to be an enormous public health problem, even with modern medicine in the 21st century. Most patients with TBI (75-80%) have mild head injuries; the remaining injuries are divided equally between moderate and severe categories.

Ocular manifestations in head injury are of common occurrence. They are often of great clinical importance in localizing the lesion and in the management of the patients. These signs are also of great prognostic value.4
MATERIALS AND METHODS

This study was conducted in the Dept. of Ophthalmology at Dr. D Y Patil Medical College Pimpri, Pune from April 2009 to December 2010. The study consisted of 100 cases of head trauma who were attended by the Dept. of Ophthalmology/Neurosurgery in our institute. We included both in patient and out patient cases which were presented to us either with open head injury or closed head injury.

The cases with direct orbital trauma and eye injuries were excluded from the study.

Complete ophthalmic evaluation was done for all the patients with head trauma including assessment of visual acuity, color vision, visual fields, fundus and motility testing. Radiological investigations like X-ray orbit and skull, Neuro-imaging CT scan and MRI of brain were done to confirm the extent of the trauma.

RESULTS

Table 1: Age distribution

<table>
<thead>
<tr>
<th>Age</th>
<th>No</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>&lt; 20</td>
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<td>12%</td>
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<tr>
<td>21 – 30</td>
<td>32</td>
<td>32%</td>
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<tr>
<td>31 – 40</td>
<td>34</td>
<td>34%</td>
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<td>41 – 50</td>
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<tr>
<td>&gt;50</td>
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<td>10%</td>
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<tr>
<td>Total</td>
<td>100</td>
<td>100%</td>
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Table 2: Sex distribution

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<thead>
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<th>Sex</th>
<th>Cases</th>
<th>Percentage</th>
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<tbody>
<tr>
<td>Male</td>
<td>68</td>
<td>68%</td>
</tr>
<tr>
<td>Female</td>
<td>32</td>
<td>32%</td>
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<tr>
<td>Total</td>
<td>100</td>
<td>100%</td>
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</table>

Table 3: Causes of trauma

<table>
<thead>
<tr>
<th>Causes</th>
<th>Cases</th>
<th>Percentage</th>
</tr>
</thead>
<tbody>
<tr>
<td>RTA</td>
<td>82</td>
<td>82%</td>
</tr>
<tr>
<td>Assault</td>
<td>11</td>
<td>11%</td>
</tr>
<tr>
<td>Fall from height</td>
<td>04</td>
<td>04%</td>
</tr>
<tr>
<td>Others</td>
<td>03</td>
<td>03%</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100%</td>
</tr>
</tbody>
</table>
Table 4: Type of head injury

<table>
<thead>
<tr>
<th>Type</th>
<th>Cases</th>
<th>Percentage</th>
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</thead>
<tbody>
<tr>
<td>OHI (Open head injury)</td>
<td>29</td>
<td>29%</td>
</tr>
<tr>
<td>CHI (Closed head injury)</td>
<td>71</td>
<td>71%</td>
</tr>
<tr>
<td>Total</td>
<td>100</td>
<td>100%</td>
</tr>
</tbody>
</table>

Table 5: Ophthalmic manifestations

<table>
<thead>
<tr>
<th>Manifestations</th>
<th>Cases</th>
<th>Percentage</th>
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</thead>
<tbody>
<tr>
<td>VI CN Palsy</td>
<td>24</td>
<td>24%</td>
</tr>
<tr>
<td>Traumatic Optic Neuropathy</td>
<td>12</td>
<td>12%</td>
</tr>
<tr>
<td>Papilloedema</td>
<td>18</td>
<td>18%</td>
</tr>
<tr>
<td>Hutchinson pupil</td>
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<td>06%</td>
</tr>
<tr>
<td>Lid Ecchymosis</td>
<td>28</td>
<td>28%</td>
</tr>
<tr>
<td>Subconjunctival Haemorrhage</td>
<td>17</td>
<td>17%</td>
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<tr>
<td>IV CN Palsy</td>
<td>19</td>
<td>19%</td>
</tr>
<tr>
<td>Terson syndrome</td>
<td>01</td>
<td>01%</td>
</tr>
<tr>
<td>III CN Palsy</td>
<td>08</td>
<td>08%</td>
</tr>
<tr>
<td>Anterior displacement of eyeball</td>
<td>01</td>
<td>01%</td>
</tr>
</tbody>
</table>

DISCUSSION

In our study, 66% of the cases were in the age group 20-40 years which indicates that trauma is common in the 3rd and 4th decades of life. Our study showed a marked male preponderance in the incidence of head trauma and motor vehicle accidents were the most common cause (82%).

Males form the major earning group of society and are more exposed to the outdoors in 3rd and 4th decades of life and thus accounting for the gender and age bias.

In our study Closed head injury was seen in 71% cases which is corroborating with many of the previous studies done by Raju N4 and Kulkarni et. al.5 but Radke N et. al. 6 in their study of 83 patients reported that open head injury patients outnumbered closed head injury patients (46.37%).

We found that VI CN Palsy was seen in 24%, IV CN Palsy in 19% and III CN Palsy in 8% but Moster et. al. 7 in their study of 46 patients reported VI cranial nerve palsy in 22%, IV cranial nerve palsy in 26% and III cranial nerve palsy in 30% cases.

In our study traumatic optic neuropathy (TON) was seen in 12% of cases but Moster et. al. 7 in their study of 46 patients reported that TON was seen in 18%. The presence of RAPD was the only objective sign in Traumatic Optic Neuropathy (12%) as fundoscopy was most often normal in acutely ill patients.
Homonymous hemianopia (15%) and Horner’s syndrome (7%) as studied by Moster et al.\(^7\) and Cortical blindness (0.4-0.6%) as studied by Banks et al.\(^8\) were not seen in our study.

We found that pupillary signs are of grave importance in indicating the site and severity of injury and in the prognosis of head injury. It aids in localizing the site of supratentorial injuries, extradural and subdural haemorrhages, and pontine lesions. Hutchinson’s pupillary signs indicate progressive coning and the need for emergent life-saving intervention. In our study, Hutchinson’s pupil was seen in 6% while Radke N et al.\(^6\) reported Hutchinson’s pupil in 2.70%.

In our study it was found that lid ecchymosis was seen in 28% which was the most common finding and sub conjunctival haemorrhage was seen in 17%, while Radke N et al.\(^6\) in their study of 83 patients reported that lid ecchymosis and subconjunctival haemorrhage were seen in 13.51%.

Papilloedema and Terson’s syndrome were seen in 18% and 1% respectively in our study. But Radke N et al.\(^6\) reported that Papilloedema was seen in 18.91% and Tersons syndrome was seen in 2.70%.

We found one case of marked anterior displacement of eye ball of right side due to torn extra ocular muscles, there was also association of medial wall and orbital floor fractures. Patient was having associated multiple injuries due to fall from height.

In summary, our study showed that RTA was the most common cause of head trauma and the age group between 20-40 years was most commonly affected with a male preponderance. Most common ocular manifestation was lid ecchymosis and 2nd most common manifestation was VI CN palsy which was commonly associated with papilloedema. In our study 22% of our patients with neuro-ophthalmic deficits had normal neuro imaging. Therefore we concluded that even in the absence of any neuro-imaging abnormalities the prevalence of neuro-ophthalmic findings is high. Although the association between head trauma and neuro-ophthalmic deficits is clear, there is as yet no definite consensus regarding the relative frequency of specific neuro-ophthalmic deficits, both afferent and efferent, that may accompany head trauma.

REFERENCES
Visual loss following non-ocular surgery is a very rare, but dreaded complication. Perioperative visual loss (POVL) could be due to cortical infarction, retinal vascular occlusion and anterior or posterior ischemic optic neuropathy.\textsuperscript{1,2} POVL has been described in a variety of operative procedures the commonest being spinal and cardiac surgery. Posterior ischemic optic neuropathy is more common following spinal surgeries while anterior ischemic optic neuropathy has been described more often following cardiac surgeries.\textsuperscript{1}

The incidence of perioperative ischemic optic neuropathy reported in the literature varies between 0.028 and 1.3%.\textsuperscript{3,4} Perioperative optic neuropathy (PON) following cardiac surgeries is mostly an anterior ischemic optic neuropathy. Patients notice vision loss in one or both eyes typically upon waking after the surgery. They have typical features of anterior ischemic optic neuropathy (AION).

Typically PON has been reported more often with conventional coronary artery bypass graft surgery with the use of cardiopulmonary bypass. There have been three reports of PON associated with off pump cardiac surgery. To the best of our knowledge, this is the first case controlled study of patients developing PON following off-pump coronary artery bypass graft (OPCABG).

**MATERIALS AND METHODS**

We retrospectively identified patients who had developed PON following off-pump cardiac surgery between August 1, 2008 and May 31, 2009 (10 months) at the Narayana Hrudayalaya Institute of Cardiac Surgery. For each case,
two age and risk-matched controls who did not experience perioperative visual loss were selected from the group of patients undergoing off-pump cardiac surgery exactly 2 weeks before the case. An approval was obtained from the Institutional Review Board of Narayana Nethralaya for this retrospective study. The medical records of all these patients were reviewed and data collected. The study group was compared with the control group and matched for age, gender, risk factors for vascular disease, and type of surgery to determine the incidence of and potential risk factors for PON. The mean arterial blood pressure and hematocrit were specifically observed in the preoperative, intraoperative and postoperative periods.

All patients had complained of vision loss on waking up in the intensive care unit. An immediate ophthalmic consult was obtained and a single ophthalmologist had examined all patients. Examination at the bedside included assessment of the visual acuity, pupillary reflexes and a dilated examination of the fundus. All patients underwent an MRI of the brain to rule out associated intracranial co-morbidities. All of them underwent serial ESR measurements to rule out the possibility of an arteritic AION. All patients underwent a thorough ophthalmic examination in the clinic including a visual field assessment and fundus photography once they were ambulatory.

The preoperative data were analyzed using Mann-Whitney test and Chi-square test. The mean arterial pressure (MAP) and the hematocrit values of both the cases and controls in the perioperative period were analyzed using repeated measures ANOVA. SPSS 15 statistical software package was used for the analysis.

**RESULTS**

PON was identified in 06 (0.42%) out of 1442 patients who underwent OPCABG in this period. Only 4 patients were considered for statistical analysis, as there was insufficient data documentation of 2 patients.

We found that there was no statistically significant difference between the cases and controls in the demographic parameters, presence of co-morbid diseases and preoperative characteristics.

There was a statistically significant change in both the hematocrit and mean arterial pressure (MAP) from the preoperative period to the postoperative period in both the case and the control groups but not between the groups (Table 1 and 2).

**DISCUSSION**

Ischemic optic neuropathy so far has been mostly described in patients who have undergone cardiac surgery with cardiopulmonary bypass (CPB). In our
We found a significant change in both the hematocrit and MAP from the preoperative to the postoperative period in both the case and the control group but not between the groups. It is likely that there are individual susceptibility risk factors independent of the external factors that may predispose a patient under duress to ischemic optic neuropathy.

The pre-existing optic nerve morphology and presence of a “disc at risk” may predispose a patient to the disorder, although this has not been proven. We could not comment regarding the “at risk” nature of the discs in our patients since there was bilateral involvement in three out of four and one of them had optic atrophy secondary to diabetic retinopathy. Although embolization has been implicated as a factor in CPB-related PON, it is unlikely in the absence of comorbidities like brain infarcts. Also, a retinal artery occlusion is a more likely result of embolization than ischemia of the optic nerve.

It is likely that there may be genetic predispositions for a complication like this. Patients in a single family have been described to have NAAION, not related to surgery. It is similarly possible that patients may have a genetic predisposition to PON.

Hypotension, either in the intra or post operative period has been implicated as a possible risk factor for PON. This could be either due to an abnormal autoregulation or an anatomic circulatory variation, both of which could lead to an inability to adequately compensate for decrease in perfusion pressures. There is a lack of data in the literature to quantify what level of hypotension
is potentially dangerous. In our patients, the average MAP was lower during night as compared to day in both the groups, although it is difficult to comment on the clinical significance of this in our series.

Although epiphenomena associated with CPB are associated with the risk of PON, it is likely that there are additional factors that play a role in this setting. And precisely because of this, it may not be completely possible to prevent this complication. However it is probably imperative to explain to the patients the possible risk of visual loss.

Although clear risk factors that dominated in cases Vs controls were not seen in this series, it does highlight the important fact that patients undergoing OPCABG, despite avoiding the risks of CPB, could develop PON.

REFERENCES

Analyze the Retinal Nerve Fibre Layer Thickness in Traumatic Optic Neuropathy

Dr. Benazir Ansari, Dr. Rashmin Gandhi

Optical coherence tomography (OCT) is a non-invasive optical imaging technique that provides high-resolution, cross-sectional, imaging of the human retina from which estimates of retinal layers thickness can be made. There have been various studies that have documented Retinal Nerve Fibre Layer Thickness (RNFL) and macular thickness measurement as being able to identify axonal loss in diseases like glaucoma, band atrophy of optic nerve. OCT has also been used to assess retinal ganglion cell volume in multiple sclerosis.

In relation to traumatic optic neuropathy (TON), previous studies have documented in single case reports that OCT can be used to document RNFL
loss \(^5\) as well as to monitor the axonal loss longitudinally. \(^6\) There have also been studies to evaluate longitudinally both the RNFL and macular thickness measurements following TON to document progressive retinal axonal loss. \(^7\)

A previous study by Liu YC et. al. \(^8\) analyzed 15 cases of traumatic optic neuropathy using OCT and evaluated the relationship between the optic nerve atrophy and visual acuity or visual field. They concluded that traumatic optic atrophy begins in the temporal area of optic disc. The nasal area’s atrophy occurs at the last and is the mildest. The more serious is the optic nerve atrophy, the worse is the vision function impairment.

The purpose of this study was to evaluate:

i) If there exists any sectoral preponderance or sparing during axonal loss in TON.

ii) Is there any significant difference in the RNFL thickness between patients who are PL negative and those with vision of PL+ and above.

We also aimed to find if there exists any correlation between RNFL thickness and –

i) duration of precedent injury, and

ii) best corrected visual acuity.

MATERIALS AND METHODS

Prospective single centre case review of 33 cases (33 eyes, 33 patients) of Traumatic Optic Neuropathy (Diagnosed on the basis of history and ophthalmic examination). The records were studied for the duration of precedent injury, best corrected visual acuity (BCVA) at presentation and retinal nerve fibre layer (RNFL) thickness at presentation. These were divided into two groups:

i) Group I- BCVA 6/6 to Perception of Light + (PL+)

ii) Group II- BCVA Perception of Light – (PL-)

Best corrected visual acuity was taken by Snellens visual acuity chart and then converted to LogMar equivalent for the purpose of statistical analysis.

RNFL thickness was measured using spectral-domain OCT (3D OCT-1000, Topcon, Tokyo, Japan) at the time of presentation. The eye was dilated using topical tropicamide drops. An internal fixation was used in patients with good vision. An external fixation target was used for the other eye in patients with poor vision in the study eye. Circle scans (diameter 3.4mm) centered on the optic nerve head containing 1024 A-scans were obtained. The axial resolution was 6 microns and transverse resolution was 10 microns with a scanning speed of 18,000 A-scans/second.

Statistical analysis was done using SPSS (version 14.0 for windows).
Independent T test was used for analysis of RNFL thickness in the two groups. Pearson coefficient of correlation was used to analyse the correlation between a) RNFL thickness and duration of injury b) RNFL thickness and duration of injury.

RESULTS

In patients with BCVA of (PL) and above (21 patients, 21 eyes):

i. Gross thinning was seen in the inferior quadrant (11 eyes, 52.4%) followed by superior (8 eyes, 38%) and temporal quadrant (5 eyes, 23.8%) while nasal quadrant was least affected (1 eye, 4.8%).

ii. Mean of average RNFL thickness ± SD in patients with PL+ and above was 70.20 µm ± 18.65 (Range 44.5 µm-103.25 µm).

iii. A negative correlation was found, in these patients, between RNFL thickness and duration of injury, however this was statistically not significant.

iv. BCVA did not correlate with RNFL thickness

In patients without PL (12 patients, 12 eyes):

i. Gross thinning was seen in the superior quadrant (12 eyes, 100%), followed by inferior (11 eyes, 91.7%) and temporal (7 eyes, 58.3%). Nasal quadrant was normal in 11 eyes (91.7%) and borderline thinned in 1 eye (8.3%).

ii. Mean of average RNFL thickness ± SD in patients without PL was 55.39 µm ± 6.7 (Range 44.75µm- 69.25µm).

iii. A negative correlation was found, in these patients, between RNFL thickness and duration of injury, however this was statistically not significant.

iv. BCVA did not correlate with RNFL thickness.

The difference in RNFL thickness in the two groups was statistically significant with a P value of .002

In conclusion inferior quadrant is the first to be affected and nasal quadrant is the last to be involved. There exists a significant difference in the RNFL thickness between the two groups of patient in our study. There exists no correlation between RNFL thickness and BCVA in either group. In PL negative patients a negative correlation exists between RNFL thickness and duration of injury, however this was statistically not significant.

REFERENCES


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**Incidence of Cerebral Venous Sinus Thrombosis (CVST) in Papilloedema**

**Dr. Saurin P. Gandhi, Dr. Kiruba E. Paul, Dr. Kartik P. Panikkar**

Cerebral (dural) venous sinus thrombosis is an uncommon condition, but its clinical presentation is varied and often dramatic. It often affects young-to-middle-aged patients, and more commonly women. According to British death certification data from 1952 to 1961, the average mortality from venous sinus thrombosis was 0.4/10^6/year over this period. Assuming a mortality rate of 10–20% over this period produces an incidence figure of 4–8/10^6/year, which is likely to be an underestimate. Venous blood from the brain flows via the superficial (cortical) and the deep cerebral veins into the venous (dural) sinuses. There are numerous connections between the cortical veins and dural sinuses, and also with the venous system of the meninges, scalp and nasal sinuses. This facilitates the spread of thrombus or infection between these vessels, but also may allow the opening of collateral draining vessels in the
event of an occlusion. The superior sagittal and lateral sinuses are commonly (70%) individually involved by thrombosis. In 30%, both are affected, in addition to cortical and cerebellar veins. Also in CVST patients, 21.2% present with ocular symptom as the initial presentation, 30.5% present with ocular symptom as well as the other symptoms, and 48.3% present with non-ocular symptoms as the initial onset.

MATERIALS AND METHODS
The medical records of all patients presenting with a clinical picture of papilloedema to Aravind Eye Hospital, Coimbatore (Tamilnadu) between the time frame of Feb 2010-2011 were retrospectively reviewed. The other factors concurrently evaluated included the patients visual acuity (Snellen's acuity), central fields (Bjerrum screen), color vision (Ishihara charts), MRI+MRV (brain) results, detailed anterior segment and fundus evaluation and also the presence of any specific false localizing signs (with laterality if present) were specifically sought for. No age limit for exclusion was decreed. Basic information including the name, age, sex, date of presentation and presence of any associated systemic illnesses was also recorded. The only exclusion criteria was the patients failure to undergo neuroimaging.

RESULTS
We had about 122 (m:f=42:80) patients with papilloedema in stated duration of which 22 did not undergo MRI+MRV brain. Out of the other 100 patients (m:f=30:51) we had 20 patients(20%) with cvst (m:f=11:9). No other correlating ocular(e.g. false localizing sign, retinal vascular disease) or systemic findings (Diabetes,Hypertension etc.) were noticed in patients with cvst. Also other significant findings on neuroimaging in these patients were features suggestive of idiopathic intracranial hypertension in 22 patients (22%), hypoplastic sinus in 8 patients (8%), intracranial space occupying lesions in 33 patients (33%) and the remaining 17 patients (17%) were normal on neuroimaging. Visual acuity at the time of presentation ranged between 6/6 and HM. Neuroimaging revealed the pattern of thrombosis showing a combination of the superior sagittal, transverse, sigmoid sinus and internal jugular vein being most commonly affected en bloc with no particular laterality taking precedence.

<table>
<thead>
<tr>
<th>Pathology</th>
<th>&lt;30 yrs male</th>
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<th>&lt;30 yrs female</th>
<th>&gt;30 yrs female</th>
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</thead>
<tbody>
<tr>
<td>Idiopathic intracranial hypertension</td>
<td>1</td>
<td>2</td>
<td>9</td>
<td>10</td>
</tr>
<tr>
<td>Intracranial space occupying lesions</td>
<td>5</td>
<td>10</td>
<td>11</td>
<td>7</td>
</tr>
<tr>
<td>Hypoplastic sinuses</td>
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<td>2</td>
<td>4</td>
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<tr>
<td>Cerebral venous sinus thrombosis</td>
<td>7</td>
<td>4</td>
<td>6</td>
<td>3</td>
</tr>
</tbody>
</table>
CVST patients, 21.2% present with ocular symptom as the initial presentation, 30.5% present with ocular symptom as well as the other symptoms, and 48.3% present with non-ocular symptoms as the initial onset. Also more than 100 causes of cerebral venous sinus thrombosis have been recorded in the scientific literature. However, even with extensive investigation, no cause is identified in 20–25% of cases. Infective causes related to the middle ear, facial skin infection or penetrating head trauma probably occur less commonly with modern aggressive antibiotic treatments, and may now only account for <10% of cases. Largely inherited prothrombotic tendencies such as factor V Leiden mutations, protein S and C and antithrombin III deficiencies are an important cause, accounting for perhaps 10–15% of cases. Various drugs have been implicated, including androgens, ecstasy, HRT and the oral contraceptive pill. Pregnancy and the puerperium have long been recognized as periods of increased susceptibility. Interestingly, most occurred in the peripartum period. A recent study by the Dutch Venous Sinus Thrombosis study group looked at prognostic factors in a series of 59 cases. Poor outcome at 12 weeks (death or Oxford handicap score >3) was associated with patients presenting with papilloedema, coma or altered consciousness, age >33, diagnostic delay >10 days, intracerebral haemorrhage and involvement of the straight sinus. Good outcome was associated with an isolated intracranial hypertension presentation and a delta sign on CT (perhaps because this led to early diagnosis). Other studies have suggested poor outcome with these and other parameters including involvement of the cerebellar veins, uncontrolled seizures, pulmonary embolism, infectious or malignant aetiology. However, even in those surviving ‘intact’, a further paper from the Dutch group looking 1 year later found 35% had cognitive impairments, 6% were dependent, 40% had symptoms that led to restrictions in lifestyle, and 40% could not resume their previous level of economic activity. This suggests there may be more morbidity after sinus thrombosis than reported previously, and interestingly, there was no significant effect of treatment on this later outcome data.

Little is known about the long-term outcome or risk of recurrence of cerebral venous sinus thrombosis, although one study reports as high a risk as 12%. Patients also have an increased risk (14%) of venous thrombosis elsewhere (DVT, PE).

Studies following recanalization of the venous sinuses have shown it may be incomplete in some cases. Raised intracranial pressure may also persist following the acute presentation of the thrombus. Although one study had suggested this results in no significant cognitive or visual morbidity, patients with persistent papilloedema and therefore raised intracranial pressure should have serial visual field assessment and be considered for a CSF shunting procedure.
Serial Monitoring in a Patient of Non Arteritic Ischaemic Optic Neuropathy

Dr. Khyati Shah, Dr. Mohana K.P., Dr. Parveen (Kharbanda) Sen, Dr. Rashmin Gandhi

To evaluate the correlation of optical coherence tomography (OCT), multifocal visual evoked potential (MFVEP), and Humphrey’s visual field (HVF) in a patient of Nonarteritic ischemic optic neuropathy (NAION) on serial monitoring.

MATERIALS AND METHODS

This was a retrospective study in a patient diagnosed clinically as NAION. The patient underwent the below mentioned tests at 5 visits over a period of 11 months.
The patient underwent Visual acuity, color vision and perimetry by the static threshold strategy and the SITA-standard 24-II program Humphrey’s visual field [2007 Carl Zeiss Meditech HFA II 750-9382= 4.2.2/4.2.2], Optical coherence tomography [Stratus OCT RNFL thickness average analysis report - 5.0.1 (0376) (Zeiss)] and multiple visual evoked potential [Veris™ Science 5.2.2X].

Mean RNFL thickness was recorded separately for the superior, inferior, nasal and temporal quadrants. Each Visual Field was divided into 6 zones based on the optic disc–Visual field map described by Garway-Heath et. al. The mean p1 latency and n1 amplitude of multifocal visually evoked potential was determined separately in superior, inferior, nasal and temporal quadrants.

Humphrey visual fields and MFVEP amplitude were compared with the RNFL thickness of the corresponding quadrant of the retina.

**RESULTS**

The visual acuity from the presentation till the last visit remained stable.

The central part of the visual field was the earliest to be affected which showed a centrocaecal scotoma with corresponding amplitude of 0.7 on the MFVEP and RNFL thickness of 80 µm, which worsened to involve the whole central field with marked thinning of 37 µm and amplitude of 0.9.

The superior had stable visual fields initially, later fields worsened with a corresponding amplitude change from 0.7 to 0.9 and decrease in RNFL thickness from 163- 92 µm.

The temporal field was less affected by the changes of NAION and showed relatively stable visual field but a decrease from 0.4 to 0.1 of MFVEP amplitude and RNFL thickness from 112 to 78 µm.

**DISCUSSION**

Nonarteritic anterior ischemic optic neuropathy (NAION) is an acute generalized or sectoral swelling of the optic disc resulting in visual field (VF) loss with or without reduction in visual acuity.\(^1\) Nonarteritic anterior ischemic optic neuropathy is thought to be due to vascular insufficiency within the optic nerve head microcirculation, but the precise mechanism and specific location of vascular compromise remain unproven. Histological studies have shown that NAION induces a thinning of the retinal nerve fiber layer (RNFL) in the affected sectors.\(^1\)

Once the acute swelling resolves clinically, visible optic disc pallor and RNFL defects are evident.\(^1\)

There was correlation between mean global RNFL on OCT and mean deviation on Humphrey VF, the ROC curve drawn between these two variables showed that when RNFL thickness decreases to less than 114.9 µm, causes of RNFL drop out such as optic atrophy should be investigated.\(^2\)
Garway-Heath et. al. produced the first high resolution map from structure to function. Using this map allowed a detailed comparison of structure (GDx image) and function (SAP) measures. This mapping allowed correlation of sectors of disc RNFL with sectors of the VF.³

Hood et. al. determine the relationship between the mfVEP amplitude and HVF loss. The agreement between the simple model and the data suggested that the decrease in the signal portion of the mfVEP response was, to a first approximation, proportional (or linearly related) to HVF loss.⁴

This correlated with our results which showed a similar picture of corresponding decrease in amplitude and RNFL thinning associated with visual field defects and mean deviation.

In conclusion the structural decrease in the RNFL thickness in the involved quadrant corresponded to the functional increased latency and decreased amplitude in MFVEP.

REFERENCES


2. Soltan-Sanjari et. al. RNFL and VF in Optic Atrophy; Journal of Ophthamlic and vision research 2008; vol.3, no. 29


Retinal Nerve Fibre Layer Thickness Provides Direct Assessment of Axonal Loss in Optic Neuritis

Dr. Rupak Kanti Biswas, Dr. Subhrangshu Sengupta, Dr. Partha Biswas, Dr. Ajoy Paul, Dr. Sourav Sinha

Optical coherence tomography (OCT) is part of the wave of advances in ocular imaging that has introduced new opportunities to visualize ocular anatomy and quantify the effects of retinal nerve fiber layer (RNFL) damage. OCT employs low-coherence interferometry to generate non-invasive, in vivo, high resolution (<10 µm), cross sectional images of the RNFL by measuring backscatter of infrared light.¹³
Our study aims to assess Retinal Nerve Fibre Layer Thickness (RNFLT) by OCT 3 in patients with Optic Neuritis (ON).

MATERIALS AND METHODS

Eighteen patients with recent onset clinically diagnosed Optic Neuritis between 19-55 years was included in the study. All patients were administered five day course of Intravenous Methyl Prednisolone at presentation after clearance by physician and testing of adequate blood parameters.

All eighteen patients were assessed for RNFLT (OCT 3; Carl Zeiss, USA) using the fast RNFL thickness software protocol at 2-3 weeks after initial onset of symptoms. The RNFLT of the affected eye was primarily analyzed. The fast RNFL thickness scan protocol was used. Good scans were defined according to specifications in the OCT-3 users’ manual: signal strength of >= 7 (maximum, 10) and uniform brightness across the scan circumference. 4 Scans meeting the above criteria only were selected.

MRI with gadolinium enhancement was also performed in all the patients.

Patients were divided into 2 groups based on MRI Report:

Group A(10 patients) with no MRI proven periventricular white matter lesion;

Group B(8 patients) with periventricular white matter lesion.

RNFLT analysis was done in 18 age matched controls who were assigned to Group C.

RESULTS

The average age of the eighteen patients is 41 years and twelve patients are females. The best corrected visual acuity at presentation in the affected eye ranged from 20/120 to Finger Counting at 1 metre.

Affected eyes of patients in Group B had thinnest RNFLT (average=39.75µ) followed by Group A(average=44.25µ). Patients in Group C had the highest average RNFLT values (average=80.75µ). The RNFLT difference was statistically significant between Group A and C.

DISCUSSION

In a pilot study Parisi et. al. used OCT to compare RNFL values between 14 MS patients and normal control subjects. They reported a significant reduction in RNFL measures among MS patients and a predilection for RNFL thinning in the maculopapillary nerve fiber bundle.

A second study compared RNFL measures to tests of visual function among 25 patients with incomplete recovery after ON and reported reduced RNFL values, which correlated with diminished visual acuity, color vision, and visual field function among patients.
Fisher et al.\(^7\) reported the results of OCT testing in a heterogenous MS cohort, which included 90 MS patients (with and without a history of ON) and 36 disease free controls. The average RNFL thickness was significantly reduced in all MS eyes as compared to normal subjects, with the lowest RNFL values noted in MS patients with a prior clinical history of ON.

Costello et al.\(^8\) found in a prospective study of 54 patients with ON, significant RNFL thinning occurred in the majority (74\%) of clinically affected eyes, often within 3 to 6 months of the acute event.

Our study also shows that patients with ON, irrespective of detection of periventricular white matter lesion on MRI, have thinner RNFL compared to age matched controls. The average RNFLT values are lowest in patients with periventricular white matter lesions.

OCT is a relatively inexpensive, non invasive, fast and reliable diagnostic modality and RNFL being unmyelinated, RNFLT provides direct evidence of axonal loss in optic neuritis patients and may prove to be a valuable diagnostic and prognostic tool in development of multiple sclerosis. Retinal nerve fiber layer thinning in non-ON eyes should be further studied as a possible subclinical indicator of disease.

REFERENCES


Cerebrospinal Fluid (CSF) Dynamics in Papilloedema

Dr. Nitika Ashok Poyam, Dr. A.H Madan, Dr. Dilipkumar Govinda Kumre

Patients with increased intracranial pressure (ICP) often develop papilloedema. Normally ICP should be < 20 mm Hg and cerebral perfusion pressure (CPP) should be ≥ 60 mm Hg. This potentially vision-threatening ophthalmological condition results from transmission of the increased ICP to the subarachnoid space (SAS) of the optic nerve (ON). Recent evidence states that the flow of CSF between intracranial SAS and ON SAS is neither continuous nor bidirectional.

Pathogenesis: Increase Cerebrospinal fluid (CSF) volume in the perioptic space results in unfolding of the ON sheath, and it was believed that this compresses the nerve, causing stasis of axonal transport, thus producing swelling of the ON axons. Increased ICP and papilloedema may leads to CSF segregation and the development of a biologically unfavorable environment from a reduced CSF recycling time within the perioptic space. CSF diversion procedure such as a lumboperitoneal, ventriculoperitoneal shunt or an ON sheath fenestration (ONSF) may help in resolving papilloedema.

Aim of the study is to determine cerebrospinal fluid (CSF) dynamics between intracranial CSF spaces and CSF in the subarachnoid space (SAS) of optic nerve.

MATERIALS AND METHODS

This was hospital based prospective, case-control study carried out in Government Medical College, Nagpur from July 2010 to August 2011. In this study 12 patients from 45 years to 65 years were selected, out of which 10 patients had papilloedema (Case Group) and 2 patients without papilloedema (Control Group). After detailed history, neurological and ophthalmological examinations, patients were subjected to Lumber puncture and MRI Phase contrast study. In Case Group, 3 were men and 7 were women while in case of Control Group one was man and other was woman. In Case Group 6 had bilateral papilloedema and 4 had unilateral papilloedema. In bilateral papilloedema 2 patients had CVE, 2 patients had Infection. 1 patient had Traumatic brain injury. One patient had Hydrocephalus. In case of unilateral papilloedema, 2 patients had papilloedema on Right side due to Astrocytoma and space occupying lesion and rest 2 patients had papilloedema on Left side due to Idiopathic intracranial hypertension and Metastatic involvement. All patients were subjected to complete neurological and complete ophthalmological (pupillary reaction and fundus) examination. Lumbar puncture (to measure
initial opening pressure and to rule out infectious causes), MRI phase study (with contrast medium) and Visual field (automated perimetry) testing were done to establish the diagnosis and to document increased ICP. After obtaining result, statistical analysis was done and conclusion was drown.

RESULTS

Contrast density was measured in Hounsfield Units (HU). Measurements were performed intracranially in the basal cisterns (pituitary and prepontine cisterns) as well as in the SAS along the orbital portion of the ON. Multiplanar reconstruction images were obtained in the axial, coronal and sagittal planes with a 1 mm slice thickness. Axial sections were used, and the diameter of the portion of the ON sheath adjacent to the globe was measured always at its widest site to standardise the measurement of the diameter of the ON sheath.

<table>
<thead>
<tr>
<th>Case group (10)</th>
<th>Control group (02)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Contrast density in Hounsfield Units (HU)</td>
<td>Unilateral (04)</td>
</tr>
<tr>
<td>ON sheath diameter</td>
<td>7.8 TO 10.2 mm</td>
</tr>
<tr>
<td>Initial opening pressure (LP)</td>
<td>Increased</td>
</tr>
</tbody>
</table>

In conclusion Unilateral and asymmetrical papilloedema in the patients with elevated ICP are well diagnosed after examining SAS of optic nerve. Compression over optic nerve due to raised ICP causes damage to the axon, mitochondria and pia septal blood supply which result in visual loss. Finally, we would emphasise that MRI phase study can help in understanding mechanism and optic nerve damage in papilloedema. Early treatment can prevent or reverse the visual loss.

Figure A showed the fundus photograph of papilloedema. Figure B and C showed the thickening of optic nerve. Figure D showed the decreased contrast loaded CSF fluid around optic nerve. Intracranial space occupying lesion with decreased contrast loaded CSF fluid was found in Figure E, F and G showed the MRI flow study in normal patient.
REFERENCES

Assessment of RNFL Thickness in Cases of Retrobulbar Neuritis

Dr. Padmavathy Maharajan, Dr. Aditi Jain

Retrobulbar neuritis is an inflammation of the optic nerve characterized by normal looking fundus with RAPD and decreased vision. Following treatment the optic disc may show pallor as a result of the axonal insult. Changes in RNFL thickness represent changes in optic nerve axonal integrity, due to the absence of myelin. Retrobulbar damage to the optic nerve causes retrograde axonal degeneration, which manifests as visible RNFL defects and optic disc pallor. OCT a non invasive imaging technique, can reliably quantify RNFL thinning in glaucoma and other optic neuropathies, and previous studies have correlated RNFL measures with tests of visual function among ON and MS patients. OCT may represent a biomarker for optic nerve axonal integrity, with the potential to quantify RNFL damage early after an ON event and predict to what extent visual recovery will ensue.

MATERIALS AND METHODS

This was a prospective study conducted at a tertiary eye care centre between July 2010 till April 2011 with patients presenting to us with a diagnosis of retrobulbar neuritis in that time period who were followed up over a period of 3 months. All the patients underwent complete neuro ophthalmic evaluation with imaging.

10 patients who were diagnosed to have unilateral presentation of retrobulbar neuritis, without involvement of the other eye. One eye of 10 healthy patients were included as age matched controls.
The OCT (Stratus version 4; Zeiss Humphrey, Dublin, CA) was used to obtain circular peripapillary scans (Fast RNFL protocol), which included three 3.4mm diameter retinal scans averaged to provide the RNFL thickness with a signal strength >7. The considered parameters were average RNFL thickness and RFNL thickness in 4 quadrants.

The patients were given a treatment of a 3 day regimen of intravenous solumedrol followed by oral steroids and reviewed after 1 week, 1 month and 3 months from time of presentation. Statistical analysis was done using STATA version 11.0 software. P values were from Kruskal-Wallis equality of populations rank test and Wilcoxon on matched pairs signed rank test where p value <0.001 was taken to be significant.

**RESULTS**

3 groups of patients included in the study

1) 10 affected eyes of patients with the disease (AE)
2) 10 unaffected eyes of patients with the disease (UE)
3) 10 normal eyes (Control group-N)

70% of patients were females with a mean age of 35.1 years ranging from 22 – 52 years.

Visual acuity at presentation was variable from 6/18 to PL +

After treatment 80% showed improvement in vision with a significant LogMAR VA of p<0.005.

### Affected eye: RNFL thickness before and after treatment

<table>
<thead>
<tr>
<th></th>
<th>Before Treatment</th>
<th>After Treatment</th>
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<tbody>
<tr>
<td></td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
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<tr>
<td>S</td>
<td>143.3</td>
<td>17.56</td>
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<td>94.4</td>
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<td>I</td>
<td>133</td>
<td>18.19</td>
<td>123.6</td>
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<tr>
<td>T</td>
<td>61.3</td>
<td>11.25</td>
<td>59.2</td>
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### Unaffected eye: RNFL thickness - before and After treatment

<table>
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<tr>
<td></td>
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<td>SD</td>
<td>Mean</td>
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<tr>
<td>S</td>
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<tr>
<td>I</td>
<td>135.8</td>
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<td>T</td>
<td>65</td>
<td>8.87</td>
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**Affected Eye and Unaffected Eye: RNFL thickness after treatment**

<table>
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<tr>
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<th>Unaffected Eye</th>
<th>P-Value</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td></td>
</tr>
<tr>
<td>S</td>
<td>136.6 (21.5)</td>
<td>136.3 (20.2)</td>
<td>0.91</td>
</tr>
<tr>
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<td>I</td>
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<td>T</td>
<td>59.2 (13.88)</td>
<td>66.7 (11.29)</td>
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**Affected Eye and Normal Control: RNFL thickness after treatment**

<table>
<thead>
<tr>
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<th>Affected Eye</th>
<th>Control</th>
<th>P-Value</th>
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<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
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</tr>
<tr>
<td>S</td>
<td>136.6 (21.5)</td>
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<tr>
<td>N</td>
<td>94.4 (22.6)</td>
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<td>I</td>
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<td>T</td>
<td>59.2 (13.88)</td>
<td>71.2 (11.83)</td>
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**Unaffected Eye and Normal Control: RNFL thickness after treatment**

<table>
<thead>
<tr>
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<th>Control</th>
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<tbody>
<tr>
<td></td>
<td>Mean (SD)</td>
<td>Mean (SD)</td>
<td></td>
</tr>
<tr>
<td>S</td>
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<td>127.1 (14.19)</td>
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<td>N</td>
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<td>I</td>
<td>134.7 (15.83)</td>
<td>127.8 (17.64)</td>
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<tr>
<td>T</td>
<td>66.6 (11.29)</td>
<td>71.2 (11.83)</td>
<td>0.326</td>
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</table>

Average RNFL thickness in the affected eyes before treatment was $110.07 \pm 17.3$ $\mu$ and after 3 months was $103.45 \pm 17.4\mu$. RNFL thickness in the unaffected eyes before treatment was $107.4\pm14.7\mu$ and after a period of 3 months was $106.77 \pm 15.3\mu$.

**DISCUSSION**

Our study demonstrates though there was no statistically significant change in the average RNFL thickness in the affected eyes before and after treatment, there was significant reduction in the RNFL thickness of the nasal and inferior quadrants but not statistically significant. Moreover on comparing with the controls there was thinning of the temporal quadrant thickness but not statistically significant. In our study there was no evidence of subclinical involvement of the other eye.

Pro et. al. described statistically significant temporal thinning in a relatively short follow-up (1-3 months).
Bertuzzi *et al.* demonstrated direct correlation between logMAR VA and RNFL thickness in temporal quadrant in ON Group 1. The limitation in our study was small sample size, one episode of ON with a short period of follow-up.

Costello *et al.* reported statistically significant thinning in all the quadrants between the affected and the non-affected eyes.\(^2,3\) On correlating the log MAR with that of RFNL thickness all the correlation coefficient are positive, which means a decrease in LogMAR is associated with a decrease in the thickness measures. But none of the correlation coefficients are statistically significant. This could be due to the fact that the sample size is too small to show a significant relationship.

Trip *et al.* demonstrated functionally relevant changes indicative of axonal loss and retinal ganglion cell loss in the RNFL and macula, respectively, after optic neuritis.\(^4\) This noninvasive RNFL imaging technique could be used in trials of experimental treatments that aim to protect optic nerves from axonal loss.

In another study Trip *et al.* showed that axonal loss contributes to optic nerve atrophy following a single attack of optic neuritis. By inference, axonal loss due to other post-inflammatory brain lesions is likely to contribute to the global MRI measure of brain atrophy in multiple sclerosis.\(^5\)

In conclusion OCT helps to track and quantify RNFL loss in cases of optic neuritis. When investigating permanent damage after ON, RNFL thickness is a promising biomarker. It shows good diagnostic validity and good correlations with functional tests in discriminating affected from unaffected eyes. Retinal nerve fiber layer thinning in non-ON eyes should be further studied as a possible subclinical indicator of disease.

**REFERENCES**


Precipitous Reduction in Blood Pressure-Potential for Ischemic Optic Neuropathy

Dr. Sujatha Rathod, Dr. Roma Johri, Dr. Kumar Ravi, Dr. Nishchitha

To create awareness about the ocular adverse effects of precipitous reduction in blood Pressure in malignant hypertension.

MATERIALS AND METHODS

Presenting a 24-year-old man, not a known hypertensive or diabetic, was referred to us from the department of Nephro urology, as a case of Chronic Renal Failure-End Stage Renal Disease (CRF-ESD) on dialysis with complaints of sudden painless loss of vision since 2 days. His BP=210/180 mm Hg.

- O/E: Vision in both Eyes was counting fingers close to face.
- A/E – within normal limits.
- Posterior segment examination revealed:
  - Bilateral Total bullous retinal detachment with hyperaemic disc.
- On investigation:

His renal parameters were altered. Accelerated hypertension was diagnosed and patient was referred back to physician for control of BP.

He presented after 2 days of treatment with a combination of IV NTG and T. Nicardia, when his blood pressure was brought down to 110/70 mm Hg. This time his vision had dropped down to Hand movements in both eyes.

- Posterior segment examination shows picture of multiple peripapillary hemorrhages and persistant bullous retinal detachment in the inferior quadrant. suggestive of bilateral ischemic optic neuropathy and inferior bullous RD.
Inference
- Precipitous reduction in bp – Potential for ischemic optic neuropathy.
- Malignant hypertension is a rare syndrome consisting of rapid and severe elevation of BP, with the systolic component >200 mmHg or the diastolic >140 mmHg.
- Nearly 1% of hypertensive patients develop malignant hypertension.¹
- The average age at diagnosis is 40 years, with men affected more than women.

Clinical Features
Divided into three distinct categories: hypertensive retinopathy, hypertensive choroidopathy, and hypertensive optic neuropathy.

Pathophysiology
- Retinopathy is usually the earliest finding in malignant hypertension and is manifested by arteriolar constriction->occlusion->ischemia, and resultant smooth muscle necrosis. This is followed by loss of autoregulation, vasodilation, and downstream transmission of high BP.
- The choroid does not autoregulate, acute rise in BP -> choroidal ischemia -> retinal pigment epithelium necrosis and serous retinal detachment. 2.1
- Hypertensive optic neuropathy is a late finding and is due to optic nerve head ischemia resulting in optic nerve edema. 2.2

Treatment, Course and Outcome
- Lowering BP in a controlled fashion minimizes end-organ damage. The actual level of blood pressure is less important in gauging the urgency of the situation than is the ongoing end-organ damage
- Too rapid a decline can lead to ischemia of the optic nerve head, brain, and other vital organs, resulting in permanent damage
- Malignant hypertensive crisis represents a medical emergency. Untreated, the mortality rate is 50% at 2 months and 90% at 1 year 3,4

Take Home Message
BP should be lowered in a slow, deliberate, controlled fashion to prevent end-organ damage

REFERENCES
Tracking RNFL Changes after IIH – A prospective Study using Optical Coherence Tomography

Dr. Padmavathy Maharajan, Dr. Aditi Jain, Dr. Kunal Rana

Idiopathic intracranial hypertension (IIH) causes swelling of the optic nerve secondary to raised intracranial tension without any organic cause. Various techniques have been employed to monitor the optic disc edema in IIH like serial fundus photo evaluation.

Optical coherence tomography is one of the promising non-invasive methods of investigation which helps us to analyze, quantify and monitor the retinal nerve fibre layer (RNFL) thickness. Our aim was to analyze the changes in peripapillary RNFL thickness using OCT in patients diagnosed with IIH and monitor the course of RNFL changes on serial follow-up.

MATERIALS AND METHODS

This was a prospective study done in a tertiary centre of 23 patients (46 eyes) diagnosed as IIH from October 2009 to October 2010. All patients underwent routine neuroophthalmic examination, visual fields testing and neuroimaging. Stratus OCT (3.0), the fast RNFL thickness 3.4 protocol was used for scanning. RNFL thickness average analysis and RNFL serial analysis programmes were used for evaluating RNFL thickness and subjects were compared with age matched and sex matched controls. All the patients included in the study were medically managed for their condition under the supervision of neurologist and were followed up at 2 weeks, 1 month and 3 months from the time of presentation with clinical assessment and RNFL analysis by OCT done at each visit.

RESULTS

Out of 23 patients with IIH, 18 (78.3%) were females, 5 (21.7%) were males. The average age at presentation was 33.5 ± 11.3 yrs. 73.9% of subjects had normal visual fields and 26% had blind spot enlargement. After 3 months the fields were within normal limits. Comparison of mean RNFL thickness between affected and normal eyes is as shown in Tables 1, 2 and 3.
Table 1: Comparison of mean RNFL thickness in IIH group between affected eyes and normal eyes

<table>
<thead>
<tr>
<th>Quadrant</th>
<th>IIH</th>
<th>Normal</th>
<th>Difference of means</th>
<th>E'</th>
<th>d.s</th>
<th>Significance</th>
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</thead>
<tbody>
<tr>
<td></td>
<td>No.</td>
<td>Mean</td>
<td>SD</td>
<td>Mean</td>
<td>S.D</td>
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<td>SUP</td>
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<td>249.1</td>
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<td>137.9</td>
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<td>240.8</td>
<td>75.2</td>
<td>128.6</td>
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<td>TEMP</td>
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<td>154</td>
<td>114.3</td>
<td>62.4</td>
<td>9.3</td>
<td>91.6</td>
</tr>
<tr>
<td>AVERAGE</td>
<td>46</td>
<td>211.3</td>
<td>80.4</td>
<td>103.3</td>
<td>8</td>
<td>108</td>
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</table>

Table 2: Mean RNFL thickness in each quadrant in IIH group

<table>
<thead>
<tr>
<th>Quadrant</th>
<th>No.</th>
<th>1st visit</th>
<th>2nd visit</th>
<th>3rd visit</th>
<th>4th visit</th>
<th>friedman coeff</th>
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</thead>
<tbody>
<tr>
<td></td>
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<td>Mean</td>
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<td>Mean</td>
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<tr>
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<td>129.2</td>
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<td>92.2</td>
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<tr>
<td>AVERAGE</td>
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<td>80.5</td>
<td>174.7</td>
<td>70.2</td>
<td>135.9</td>
<td>135.9</td>
</tr>
</tbody>
</table>

Table 3: Comparison of Mean RNFL thickness of diseased eye with normal eye at IV visit, IIH group

<table>
<thead>
<tr>
<th>Quadrant</th>
<th>Diseased</th>
<th>Normal</th>
<th>Difference of means</th>
<th>d.s</th>
<th>Significance</th>
</tr>
</thead>
<tbody>
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<td>SD</td>
<td>Mean</td>
<td>SD</td>
<td>d.s</td>
</tr>
<tr>
<td>SUP</td>
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<td>23.1</td>
<td>134.8</td>
<td>11.6</td>
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<tr>
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<tr>
<td>TEMP</td>
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<tr>
<td>INF</td>
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<td>18.5</td>
<td>60.2</td>
<td>9.6</td>
<td>10.7</td>
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<tr>
<td>Average</td>
<td>102.2</td>
<td>18.4</td>
<td>101</td>
<td>10.1</td>
<td>1.2</td>
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</table>

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Table 1 is a comparison of RNFL thickness of the eyes of IIH patients with normal eyes at presentation. It shows highly significant increase in RNFL thickness in all four quadrants.

Table 2 shows that the average thickness of RNFL is significantly reduced over the study period (from 1st visit to 4th visit) with treatment.

Table 3 shows that although superior and inferior quadrants recorded some loss, there was no statistically significant difference between the affected eyes and the normal eyes after resolution of disc edema.

**DISCUSSION**

Primary focus was to objectively record the changes in the RNFL thickness over the course of the disease in patients with IIH. Demonstrable changes in RNFL thickness, correlating with the clinical picture, could be recorded in an objective and reproducible manner. In cases with IIH, the average thickness of RNFL showed significant change, as compared with the normative data as well as the baseline measurement on serial follow up over a period of three months, with treatment. Sanchez - tocino et. al. did a similar study to show the utility of OCT in diagnosis and follow up of IIH in paediatric age group. They observed that the RNFL thickness was increased 2-3 times over the normal expected values and returned to normal after treatment was started.

In conclusion OCT has a potential role to monitor the progression and regression of disc edema by quantification through serial RNFL measurements.

**REFERENCES**


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**Rutin**
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**Lutein & Zeaxanthin**
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- Increases MPOD (Macular Pigment Optical Density) in DR patients\(^3\)

**References:**
2. Clinics in Dermatology (2009); 27: 195-201