Indirect optic nerve injury in two-wheeler riders in northeast India

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Purpose: To investigate the association of posterior indirect traumatic optic neuropathy and superior temporal orbital rim injury in two-wheeler riders and documentation of the clinical profile of such cases.

Design: Retrospective observational study.

Materials and Methods: Records of all patients reporting with cranio-orbital injury and vision loss following road traffic accidents between October 1994 and April 2006 were reviewed and from them cases with vision loss solely from indirect optic nerve injury were taken up for study. The prognostic significance of different presenting features, role of intravenous methyl prednisolone (IVMP) and relative risk of superior orbital rim injury to posterior indirect traumatic optic neuropathy (at 95% confidence interval) was calculated.

Results: Out of 129 consecutive cases of cranio-orbital injury, 35 had posterior indirect traumatic optic neuropathy with minor ipsilateral superior temporal orbital rim trauma and none used any protective headwear. Presenting clinical features like relative afferent pupillary defect (P= 0.365), optic disc status (P= 0.518) and visual evoked potential (VEP) (P= 0.366) were disproportionate to visual loss. Only VEP had prognostic significance. The IVMP did not provide any added therapeutic benefit. The remaining 94 cases sustained direct blinding ocular trauma and 28 of them had associated intracranial pathology. The relative risk of superior temporal orbital rim injury to posterior indirect optic nerve trauma was 2.25.

Conclusion: Superior temporal orbital rim injury, even when minor, carries a potential risk for development of blindness from indirect posterior indirect traumatic optic neuropathy in two-wheeler drivers. Presenting signs do not correlate with visual status. Only VEP has prognostic significance and the condition is untreatable.

Key words: Orbital rim injury, posterior indirect traumatic optic neuropathy, protective headwear, two-wheeler

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Posterior indirect traumatic optic neuropathy and visual system injury is a serious and infrequently found condition where treatment is controversial and permanent visual loss is almost evident. It can occur following an innocent ipsilateral injury over the superior temporal orbital rim and is characterized by vision loss without external or internal ophthalmic evidences of injury to the eye and its nerve. Posterior indirect traumatic optic neuropathy is seen in up to 5% of all the cases of closed head trauma and only a few series of such reports are available in the literature. In India it is estimated that over 500,000 people suffer from some form of head trauma every year and even if a 2% incidence is considered, over 10,000 of them will develop posterior indirect optic nerve trauma and severe visual loss. Till the treatment is known, identification of risk factors and prevention will have a considerable role to play in its management. The aim of the study was to evaluate the profile of posterior indirect traumatic optic nerve neuropathy in two-wheeler riders with injury over the superior temporal orbital rim, in the northeastern part of our country.

Materials and Methods

A retrospective study was conducted at a tertiary eye care center, where case records of 129 consecutive patients presenting with history of accidental forehead injury and vision loss occurring between October 1994 and April 2006 in two-wheeler riders following road traffic accidents (RTA) were reviewed. The association of cranio-orbital injury and vision loss due to indirect optic nerve injury was investigated. The cases reporting were divided into patients with posttraumatic vision loss due to any organic ocular etiology and associated unconsciousness and neurological pathology (Group I) and those with ipsilateral vision loss without ocular injury following outer orbital rim injury, without any associated unconsciousness and neurological pathology (Group II). The cases in Group I were excluded at the very beginning. The Group II cases were taken up for further analysis in this study and three aspects were analyzed in detail – viz., i) Clinical presentation and its relation to visual loss, ii) investigation with predictor value in visual loss and iii) response to any treatment received. Being a tertiary eye care hospital, the patients have reported at different periods of time interval from the injury either for a second opinion or after being referred by the primary attending consultant. Thirty-five cases were included for the present study as per the following inclusion criteria. The set institutional
Diagnostic criteria for posterior indirect traumatic optic neuropathy were decreased or total loss of vision following closed head trauma, with various grades of relative afferent pupillary defect (RAPD) at presentation, normal or optic disc pallor and positive Visual Evoked Potential (VEP) findings. Recommended investigations were VEP, high-resolution computed tomography (HRCT) and magnetic resonance imaging (MRI) of the orbit, and brain.

Institutional trauma scheme for examination, investigation and follow-up of such cases was, recording of detailed clinical history, including mode of injury, treatment received elsewhere for trauma and vision loss, preexisting ocular condition, conventional ocular examination and neuro-ophthalmological evaluation with special emphasis on visual acuity, color vision, pupillary size, direct, consensual and accommodative pupillary reaction, and swing flash test and RAPD grading, dilated fundus examination including optic disc study and its comparison to other eye. Before diagnosis of posterior traumatic indirect optic neuropathy, fictitious amblyopia, previously undiagnosed ocular pathology and any neuropathology were excluded. Investigation schedule was recording of VEP, visual field, HRCT and MRI. The RAPD grading that was followed was Grade I; a weak initial constriction and greater redilatation, Grade II; initial still and greater redilatation, Grade III; immediate pupillary dilatation, Grade IV; immediate pupillary dilatation following prolonged illumination of the good eye for 6 sec and Grade V; immediate pupillary dilation with no secondary constriction. Follow-up protocol was from Day one of the injury, repeated examination every 48 h for the first week, weekly for one month, monthly for three months, every three months for one year then yearly thereafter with provision of lateral entry at any stage. The findings from the reviewed documents were recorded in the proforma of the study. Minimum time interval for primary analysis of a case in this study was three months. The relative risk (RR) of superior temporal orbital rim injury to posterior indirect traumatic optic neuropathy and its 95% confidence interval in the study were calculated. The statistical significance of the RAPD, optic disc status and with that of the initial visual acuity was calculated by student’s t test and their prognostic significance was analyzed. Statistical calculation was done using Microsoft Excel and Statistical Software.

Results

Case records of 129 consecutive cases of cranio-orbital injury were reviewed. Out of them 35 cases with superior temporal orbital rim injury over the eyebrow and vision loss-associated posterior indirect traumatic optic neuropathy, fulfilling the criteria were included in the study [Table 1]. Thirty-two patients were male (91%) and three were female (9%) with an average age of 39 years (age range 10-63 years) [Figure 1]. Right and left laterality of the injury was 11 (31%) and 23 (69%) respectively. Site of injury was typically located on the ipsilateral superior temporal orbital rim over eyebrow at the extreme lateral end. Horizontal or vertical cut [Figure 2]. Black eye, edema, abrasion alone or in combination was the presenting nature of the wound. In selected cases, circumstances leading to injury were accidental without any vehicular collision and the drivers were in casual mode driving without using any protective headwear. Majority of them skidded and fell due to unfavorable road conditions. Eleven of them received first aid and all could drive back home or went to their destinations and considered the matter very minor and felt no necessity for any ophthalmology consultation. The remaining 94 cases had history of vehicular collision. All had some form of direct ocular injury accounting for visual loss [Table 2]. Twenty-eight cases out of them were unconscious and were under neurological intensive care for a variable period of time after the accident and 13 cases of them had residual non-visual neurological defect also. Incidentally, except seven, the rest (n=87) of the cases were using protective headwear during driving. The most interesting fact observed in the study was that medial superior orbital rim injuries were not associated with posterior indirect traumatic optic neuropathy [Figure 3]. The relative risk (RR) of superior temporal orbital rim injury to posterior indirect traumatic optic neuropathy was 2.25 and its 95% confidence interval (CI) was 1.25 - 4.04 in this study.

Presenting clinical features of the cases (selected for the study) were as follows. Only three patients reported within five days following injury and the mean interval between injury and the presentation was 37.26 days [Table 1] and one case presented as late as 180 days. Delayed presentation was due to the relatively trivial nature of the injury and had usually received initial treatment elsewhere. Visual acuity ranged from no perception of light in 12 (34%) cases to 20/30 in one (3%) case [Figure 4]. All had abnormality in pupillary status. The RAPD grading was Grade IV - 29 (83%) cases, Grade I - 4 (11%) cases, Grade II - 2 (6%) cases; however Grade III and Grade V RAPD were not found in any cases.

Variable amount of optic disc changes was found. It revealed generalized pallor in 23 (66%) cases, normal disc appearance in five (14%), temporal pallor in four (11%) cases, primary looking optic atrophy in two (6%) cases, doubtful pallor in one (3%) case, hyperemic disc in one (3%) case. All cases with normal disc appearance reported within 14 days of injury but detectable disc pallor was there on 10th day post injury also. The exact time of onset of the disc changes could not be ascertained from the study.

Flash VEP of the affected eye showed an extinguished and flat response in 21 (60%) cases (one had extinguished response even for 16 x 16, 32 x 32, 64 x 64 ), reduced amplitude in seven (20 %) cases, increased latency in six (17 %) cases and normal response was seen in one (3 %) case.

Figure 1: Age distribution

![Image](https://via.placeholder.com/150)
The HRCT and MRI of the cases did not reveal any abnormal findings, except in two cases - one had hairline fracture in the orbital plate of the frontal bone and the other had diffuse swelling of the optic nerve [Figure 5].

Records showed that 18 cases received intravenous methyl prednisolone (IVMP) (1000 mg daily for three consecutive days) at random of which two cases improved to best corrected visual acuity of 20/20 but still had temporal disc pallor during final checkup. Seventeen cases received no treatment but two of them showed similar improvement during the final checkup.

Significant visual recovery was observed in four cases (out of total seven cases with visual improvement) [Figure 4]. Out of these four cases, two cases had an extinguished response in VEP at presentation but still recovered to have a good visual acuity later on. Twenty of the 21 cases with extinguished response had poor or no final visual recovery. The patient, who had a hyperemic disc on presentation, regained maximum visual acuity of 20/20 on follow-up with a subnormal VEP. Three cases that had a good visual acuity on follow-up had temporal pallor of the optic disc and one case had a pale disc. Five cases that had a normal disc appearance had poor visual acuity at presentation with a delayed VEP response. These show no definite correlation between clinical features. The

**Discussion**

Traumatic optic nerve damage after craniofacial injury was first described by Hippocrates.\(^{10}\)

Indirect injury to the intra-canalicular part of the optic nerve following head trauma occurs as a result of transection of nerve fibers, interruption of blood supply or secondary hemorrhage and edema following shearing or avulsion of the nutrient vessels or by the pressure transmitted along the bone of the optic canal.\(^{5,11-17}\)

The present study revealed that automobile collisions may lead to life-threatening injury to two-wheeler drivers and associated visual loss in them is either due to organic damage to the eye in the form of open globe injury, vitreous hemorrhage etc. or injury to the intracranial part of the visual system [Table 2]. Accidental fall in the road from two-wheeler without any vehicular collision causes an injury, many a times trivial, over the superior temporal orbital rim in particular, if the rider does not use any proper protective helmet. When

**Figure 2:** Typical location of injury at the orbital rim seen in indirect optic nerve trauma

**Figure 3:** Midline injury of the forehead causes direct ocular trauma and vision loss

**Figure 4:** Graph showing initial and final vision

**Figure 5:** MRI findings

RAPD, optic disc status and VEP had no statistical significance (student's t) with presenting visual acuity ($P=0.365$, $P=518$, $P=0.360$ respectively). But the VEP finding was observed to be the only predictor for final visual recovery.
such civil accident occurs the rider reflexly grips the handle more firmly resulting in the unprotected head striking the road surface. The lateral aspect of the forehead (over the orbital rim) is the most vulnerable point of such injury. All 35 cases of the present study had injury over the lateral (temporal) aspect of the eyebrow. Similar findings were observed in the International Optic Nerve Trauma Study.12 Lessell4 found that the commonest mode of such an injury was fall from bicycle, closely followed by automobile collision. However, similar indirect optic nerve injury is also possible following a blow to the face, forehead or less commonly to the temple.5,11 Analysis of circumstances leading to such accidents revealed that the minor accidents were due to bad road condition and none of the riders were using a protective helmet at that time of the accident. Absence of indirect optic nerve injury in helmet user indicates it protective role. All cases of indirect optic nerve injury in helmet users had associated head injury indicating very severe impact.

Table 1: Profile of the cases

| Sex | Age in years | Eye | Interval between injury and presentation (days) | VA at presentation | IVMP | Final VA | RAPD grade | Optic Disc status | VEP |
|-----|--------------|-----|---------------------------------------------|-------------------|------|----------|------------|-------------------|-----|
| M   | 40           | OS  | 60                                          | FC 1/2 M          | No   | FCCF     | 4          | GP E              |     |
| F   | 38           | OD  | 11                                          | FC 2M             | No   | FC 2M    | 2          | GP IL             |     |
| M   | 36           | OS  | 19                                          | 20/400            | Yes  | 20/400   | 1          | TP IL             |     |
| M   | 26           | OS  | 30                                          | 20/60             | No   | 20/60    | 2          | TP IL             |     |
| M   | 17           | OS  | 10                                          | FCCF              | No   | FCCF     | 1          | GP IL             |     |
| M   | 50           | OD  | 104                                         | P L negative      | Yes  | P L negative | 1         | GP IL             |     |
| M   | 51           | OS  | 15                                          | 20/60             | No   | 20/60    | 4          | GP RA             |     |
| F   | 29           | OD  | 30                                          | FC 2M             | No   | FC 2M    | 4          | GP RA             |     |
| M   | 60           | OD  | 5                                           | FCCF              | Yes  | P L negative | 4         | N E               |     |
| M   | 18           | OS  | 180                                         | HMCF              | Yes  | HMCF     | 1          | GP E              |     |
| M   | 40           | OS  | 5                                           | HMCF              | Yes  | CF 1/2 M | 4          | GP IL             |     |
| M   | 43           | OD  | 8                                           | 20/80             | Yes  | 20/20    | 4          | TP N              |     |
| M   | 30           | OS  | 45                                          | P L negative      | No   | P L negative | 4         | GP E              |     |
| M   | 33           | OD  | 120                                         | FC 2M             | No   | NR       | 4          | GP E (10X16)       |     |
| M   | 27           | OS  | 14                                          | 20/120            | No   | 20/30    | 4          | GP RA             |     |
| M   | 35           | OS  | 60                                          | 20/400            | No   | 20/20    | 4          | H RA              |     |
| M   | 39           | OD  | 31                                          | P L negative      | Yes  | P L negative | 4         | GP E              |     |
| M   | 25           | OD  | 19                                          | P L negative      | No   | P L negative | 4         | GP E              |     |
| M   | 30           | OD  | 71                                          | FC 1M             | No   | P L negative | 4         | POA E             |     |
| M   | 22           | OS  | 81                                          | P L negative      | No   | P L negative | 4         | GP E              |     |
| M   | 30           | OS  | 12                                          | 20/30             | Yes  | 20/20    | 1          | TP RA             |     |
| M   | 15           | OS  | 14                                          | P L negative      | Yes  | P L negative | 4         | N E               |     |
| M   | 36           | OS  | 11                                          | P L negative      | No   | P L negative | 4         | N E               |     |
| M   | 51           | OS  | 4                                           | P L negative      | Yes  | P L negative | 4         | DP E              |     |
| M   | 23           | OS  | 14                                          | PL positive       | Yes  | P L negative | 4         | GP E              |     |
| M   | 24           | OD  | 22                                          | P L negative      | No   | P L negative | 4         | GP E              |     |
| M   | 20           | OS  | 14                                          | FCCF              | No   | P L negative | 4         | POA E             |     |
| M   | 17           | OS  | 18                                          | FC 2M             | Yes  | FC 2M    | 4          | GP E              |     |
| F   | 63           | OS  | 20                                          | HMCF              | Yes  | FCCF     | 4          | N E               |     |
| M   | 10           | OS  | 33                                          | FCCF              | Yes  | FCCF     | 4          | GP RA             |     |
| M   | 35           | OD  | 15                                          | P L negative      | Yes  | P L negative | 4         | N E               |     |
| M   | 32           | OD  | 60                                          | P L negative      | Yes  | P L negative | 4         | GP E              |     |
| M   | 38           | OS  | 14                                          | 20/60             | No   | P L negative | 4         | GP RA             |     |
| M   | 38           | OS  | 105                                         | P L negative      | Yes  | PL negative | 4         | GP E              |     |
| M   | 31           | OS  | 30                                          | PL positive       | Yes  | FC 1F   | 4          | GP E              |     |

M = Male, F = Female, VA = Visual acuity, IVMP = Intravenous Methyl Prednisolone, RAPD = Relative Afferent Pupillary Defect, VEP = Visual Evoked Potential, OD = Oculus Dexter, OS = Oculus Sinister, FC = Finger Counting, M = Metre, FCCF = Finger Counting Close to Face, PL = Perception of Light, HMCF = Hand Movement Close to Face, NR = No Reaction, F = Foot, E = Extinguished, RA = Reduced Amplitude, IL = Increased Latency, N = Normal, GP = Generalized Pallor, TP = Temporal Pallor, N = Normal, POA = Primary looking optic atrophy, DP = Doubtful Pallor, H = Hyperemic disc.
The HRCT and MRI of the orbit and head were negative in all of our cases except two who had orbital roof fracture and diffuse thickening of the optic nerve. So the exact mechanism of optic nerve injury in the present series of cases remained ill understood. Lessell found craniofacial fracture in 17 of 33 imaged cases, and seven had fracture intersecting the optic canal. But neither the presence nor the location of a fracture correlated with the severity of the optic neuropathy. Superior orbital rim injury even when minor carries a potential risk for development of indirect posterior indirect traumatic optic neuropathy and blindness in two-wheeler riders. Presenting signs do not correlate with visual status and VEP showed significance as a prognosis predictor. The condition is untreatable but can be prevented by using protective headgear.

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