Neuro-ophthalmic Manifestations of Head Trauma

M A Rani Sujatha¹, Sakina Irum²

¹Professor and Head, Department of Ophthalmology, Dr. B. R. Ambedkar Medical College and Hospital, Bengaluru, Karnataka, India,
²Post-graduate student in the Department of Ophthalmology, Dr. B. R. Ambedkar Medical College and Hospital, Bengaluru, Karnataka, India

Abstract

Introduction: 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.

Purpose: To describe the neuro-ophthalmic findings in a group of patients with head trauma.

Materials and Methods: A retrospective chart review of patients who were given a diagnosis code of cerebral concussion and cerebral contusion, in a tertiary hospital between 2014 and 2016. A total of 50 consecutive patients were reviewed. Age ranged from 2 to 86 years. The mean age was 30 years. Motor vehicle accident was the most common cause of head trauma, occurring in 74% patients.

Results: Ophthalmological examination was undertaken in head trauma patients, and an abnormal neuro-ophthalmic examination result was noted in 9.5% of total patients, 40% of ophthalmologically examined patients. Traumatic optic neuropathy was the most common manifestation, followed by oculomotor, abducens, trochlear, facial nerve palsy, Terson syndrome, and internuclear ophthalmoplegia. Loss of consciousness and cerebral concussion were not associated with any outcomes, but the presence of neuroimaging (computed tomography, magnetic resonance imaging) abnormality, particularly intracranial hemorrhage (ICH) and skull fracture, was significantly associated with neuro-ophthalmic deficits.

Conclusions: Head trauma causes a number of neuro-ophthalmic manifestations. ICH and skull fracture may be a reliable predictor of specific neuro-ophthalmic outcomes.

Key words: Head trauma, Neuro-ophthalmic manifestations, Visual loss

INTRODUCTION

Neuro-ophthalmic deficits occur commonly after head trauma.¹⁻¹³ The afferent and efferent visual systems are susceptible to injury from a variety of mechanisms. Such patients pose a diagnostic and therapeutic challenge, due to frequently vague nature of their visual complaints and their coexistent neurologic deficits. Although the association between head trauma and neuro-ophthalmic deficits is clear, there is no definite consensus regarding the relative frequency of specific neuro-ophtalmic deficits, both afferent and efferent that may accompany head trauma.

In this review, we studied the frequency of various neuro-ophthalmic deficits that occurred after head trauma and their relationship to the nature of the head injury.

MATERIALS AND METHODS

A retrospective review of all consecutive patients seen between 2014 and 2016 at the tertiary hospital and given a diagnosis code of head trauma was performed. All patients underwent a standardized neuro-ophthalmic history and examination. Visual fields were tested using a Humphrey automated perimeter.

Duration of time from injury to examination, type of trauma sustained, extent of injuries (including neuroimaging, if available), and loss of consciousness (LOS) were noted for each patient. For patients diagnosed with optic neuropathy, a distinction among mechanisms of injury (indirect, direct, and papilledema) was made on the basis of visual acuity, visual field, and optic disc...
appearance. Cause of injury, sex, LOS, and the presence of any neuroimaging abnormality were all evaluated as potential predictors of outcome, with outcome being defined as the presence of a neuro-ophthalmic deficit or a combination of neuro-ophthalmic deficits.

The association between the predictors and outcomes was evaluated using a chi-squared test.

RESULTS

A total of 50 patients were reviewed. They included 28 (56%) men and 22 (44%) women. Age ranged from 2 to 86 years, with a median of 30 years. Most patients were admitted to the emergency department at the time of their evaluation in our unit. The neuro-ophthalmic examination was part of a systematic evaluation obtained before their discharge and included both symptomatic and asymptomatic patients. Only patients cooperative enough to perform a complete neuro-ophthalmic examination, including formal visual field testing, were evaluated.

Motor vehicle accident (MVA) was the most common cause of head trauma, occurring in 29 patients (58%). Of these, 20 were passengers or drivers, and the remaining patients were pedestrians struck by a moving vehicle. Falls and projectile injuries were less common, accounting for 31.2% of patients in total. Time from injury to examination ranged from 2 days to 2 years, with a mean of 73.5 days ± 291.8 days. 20 patients (39.9%) experienced an LOS ranging from several minutes to a prolonged coma of several month's duration at the time of injury. Clinically relevant neuroimaging findings at the time of injury were noted in 25 patients (50%). We considered the following abnormalities as clinically relevant: Intracranial hemorrhage (ICH) (epidural, subdural, or intraparenchymal), basal skull fracture, and radiographically evident contusion. ICH was the most common finding, occurring in 31 patients (62.1%). In 19 patients (34.3%), neuroimaging was normal.

An abnormal neuro-ophthalmic examination was found in 31 patients (62%). Of the patients with abnormal neuro-ophthalmic examinations, 13 (50%) had afferent pathway deficits, and 18 patients (58.9%) had efferent pathway deficits. Among afferent deficits, retrochiasmal visual field defects were the most common, occurring in 7 patients (50.5%).

Optic neuropathies occurred in 6 patients (43%) and included indirect optic nerve injury and optic nerve injury secondary to previously elevated intracranial pressure with papilledema (15%). Chiasmal injury and Terson syndrome were uncommon, each occurring in just 1 patient (3.2%). Among efferent deficits, ocular motor cranial nerve abnormalities predominated, with 14 patients (77.1%) having at least one ocular motor nerve palsy (Table 1). Among these patients, 3 (22.6%) had multiple ocular motor nerve palsies. Trochlear nerve palsy was the most common deficit, followed closely by oculomotor nerve palsy. 7 patients had bilateral oculomotor motor palsies, with bilateral trochlear nerve palsy occurring most frequently. Supranuclear ocular motor deficits were less common and included convergence insufficiency (14.7%), supranuclear gaze palsy (two patients), and dorsal midbrain syndrome (two patients). Horner syndrome was uncommon, occurring in only one patient. Central vestibular nystagmus was documented in only two patients.

Cause of injury was not significantly associated with any outcome. Female sex was significantly associated with trochlear nerve palsy ($P = 0.05$) but not with any other outcome. Male sex was not significantly associated with any outcome. The presence of any neuroimaging abnormality was significantly associated with indirect optic nerve injury ($P = 0.05$) and oculomotor nerve palsy ($P = 0.05$).

The presence of an ICH was significantly associated with oculomotor nerve palsy ($P = 0.003$) and bilateral trochlear nerve palsy ($P = 0.02$). The presence of a basilar skull fracture was significantly associated with trochlear nerve palsy ($P = 0.05$). The presence of a radiographically evident contusion was not significantly associated with any outcome (Graphs 1-4).

DISCUSSION

Head trauma is common. In one survey, the incidence of traumatic head injury requiring hospitalization ranged from 109 to 322/100,000 people.\(^4\) The advent of motorized transportation has increased the clinician's exposure to head trauma and its complications. Indeed, MVA is the most frequent cause of head injury. Young men are most frequently affected\(^4,5\) as noted in our study.

The relationship between head trauma and neuro-ophthalmic injury has been recognized for some time. Hutchinson's studies,\(^6\) in the 1880s, documented a clear relation between head trauma and ocular motility deficits. The relative frequency of neuro-ophthalmic deficits is highly variable, and this finding may be explained by differences in the patient population. However, a few studies reviewed patient populations that were homogeneous in age or clinical characteristics; for example, Lepore\(^11\) studied motility deficits in patients with known heterodeviations and subjective diplopia, thereby perhaps missing patients with only afferent, or afferent and efferent, deficits. Furthermore,
in some reviews, certain aspects of the neuro-ophthalmic examination (such as visual field defects) were not addressed. Finally, referral bias may play a role in that patient with only mild neuro-ophthalmic deficits or patients whose neurologic status renders them unable to communicate symptoms may not be referred to the neuro-ophthalmologist. It is very likely that we had such a bias because not all patients with head trauma were evaluated by us; however, we think that our series is most likely representative of the population with severe head trauma. This neuro-ophthalmic evaluation was part of a routine evaluation performed before their discharge. Only 56.7% of our patients had an abnormal neuro-ophthalmic examination and suggesting that not only symptomatic patients were referred to us.

Closed head injury may damage the optic nerve through a variety of mechanisms, including direct injury from a penetrating wound, indirect injury from concussive forces transmitted to the nerve, and disc edema from elevated intracranial pressure. Indirect optic nerve injury is the most common form of traumatic optic neuropathy (TON). The incidence of indirect TON ranges from 2.8 to 26.1%. This large range is most likely related to several factors: (1) TON may be difficult to diagnose in the acutely ill, head-injured patient; (2) distinction between the mechanisms of TON (direct vs. indirect injury, papilledema) requires adequate historical information and accurate and reliable visual field testing, both of which may be difficult to obtain in neurologically impaired patients; and (3) attention is often directed to efferent pathway deficits because these are more amenable to management. Our study showed a higher incidence of indirect TON than many previous studies and suggesting that TON may be under-diagnosed in the head trauma population. Although the management of indirect TON is controversial, patients with indirect TON seen within the first few hours may benefit from treatment with high-dose corticosteroids. Therefore, indirect TON should always be suspected in patients with severe head trauma.

Traumatic chiasmal injury is uncommon and is often associated with frontal head trauma. Typical features include bitemporal hemianopia (often complete), cerebrospinal fluid rhinorrhea, and diabetes insipidus. The mechanism

<table>
<thead>
<tr>
<th>Nerve injured</th>
<th>Number of patients with unilateral nerve injury</th>
<th>Number of patients with bilateral nerve injury</th>
</tr>
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<tbody>
<tr>
<td>Optic nerve</td>
<td>7</td>
<td>6</td>
</tr>
<tr>
<td>Oculomotor nerve</td>
<td>14</td>
<td>7</td>
</tr>
<tr>
<td>Trochlear nerve</td>
<td>16</td>
<td>12</td>
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Graphs 1: Ratio of males: Females evaluated
Graphs 2: Motor vehicle accidents
Graphs 3: Afferent deficits
Graphs 4: Efferent deficits
of injury is unclear but may involve physical disruption of the chiasm or diffuse axonal injury.1

Because a relatively large proportion of neural tissue is dedicated to the primary retrochiasmal visual system, it makes intuitive sense that diffuse brain injuries should frequently damage these pathways; however, the relative frequency of traumatic retrochiasmal visual field defects is difficult to ascertain in the literature because many studies do not specifically address this issue. This observation may again reflect the difficulty in assessing visual fields in neurologically impaired patients, particularly using the automated perimetry. We were able to obtain a reliable visual field test in all our patients, using the Humphrey automated perimeter. More than half of our patients with afferent pathway deficits had retrochiasmal visual field defects, a much higher incidence than that reported in comparable studies. Because these visual field defects may have substantial financial and legal implications (regarding driving and employment), accurate visual field testing is critical in head-injured patients. Because accurate testing is often not possible in the immediate period after the injury and may not be possible for weeks or even months after the injury, it is important that patients be monitored at regular intervals until an accurate assessment can be made.

The ocular motor nerves may be damaged at any point in their course from brainstem nucleus to extraocular muscle. The most likely mechanism of injury is axonal shearing resulting from the differential acceleration of the skull and brain;2,3 however, focal lesions such as hemorrhage at the brainstem exit site16 or avulsion of the nerve root12 may also occur. In some cases, minor head trauma may unmask a pre-existing, otherwise unrecognized mass lesion.17

The trochlear nerve is the smallest and longest of the ocular motor nerves and is closely associated with the rigid tentorium, making it susceptible to traumatic injury. It is particularly susceptible to injury as it emerges from the dorsal surface of the brainstem; damage at this location often causes bilateral injury.1

The trochlear nerve has been variably reported to be the most and least frequently involved ocular motor nerve after head trauma.1,16,19 Indeed, the diagnosis of trochlear nerve palsy may be difficult, particularly in uncooperative, neurologically impaired patients. In our study, trochlear nerve palsy was the most common ocular motor nerve palsy, occurring in 51.2% of patients with ocular motor nerve palsies. A high proportion (32.5%) was bilateral, reinforcing the concept that a bilateral trochlear nerve palsy should be suspected in any patient with what appears to be a unilateral injury. Further, a certain proportion of posttraumatic trochlear nerve injury may represent decompensation of a pre-existing congenital trochlear nerve palsy. Indeed, in one study, congenital trochlear nerve palsy was the most common cause in children.20 Determination of vertical fusion amplitude and examination of old photographs usually help to distinguish congenital from acquired trochlear nerve palsy.

Trauma is the second or third leading cause of oculomotor nerve palsies in adults and the leading cause of acquired third nerve palsies in children.1,21 The site of injury is often difficult to localize unless other neurologic findings are present. Among our patients with ocular motor nerve palsies, oculomotor nerve injury was the second most common deficit and found in 46.4% of patients with ocular motor nerve palsies; only 7.7% were bilateral.

Acquired abducens nerve palsy is the most commonly recognized ocular motor nerve palsy in any age group.1,19,21 This finding probably relates more to the ease of diagnosis rather than frequency of occurrence because acquired abducens nerve palsies are relatively rare in certain age groups such as young adults.22 The abducens nerve has a long course and is susceptible to injury at any point; it is particularly vulnerable to the effects of elevated intracranial pressure. The abducens nerve was the least commonly affected ocular motor nerve in the previous studies, and this finding is in agreement with our series.

Convergence insufficiency is a supranuclear motility disorder characterized by a remote near point of convergence, poor convergence amplitudes, and an exodeviation at near. Convergence insufficiency may be seen in a variety of clinical settings, but its association with head trauma has been well documented.3 Similar to previous studies, convergence insufficiency was the most common supranuclear ocular motility deficit among our patients, seen in 14.7% of patients with efferent deficits. Other supranuclear motility disorders (such as supranuclear gaze palsies, skew deviation, and dorsal midbrain syndrome) are less commonly reported in association with head trauma. This finding may again reflect difficulty in diagnosing these deficits in uncooperative patients. In addition, some of these disorders may be relatively asymptomatic and therefore not prompt referral to a neuro-ophthalmologist. Furthermore, these supranuclear disorders, particularly convergence insufficiency, may be masked by coexistent ocular motor deficits.11

Knowing if specific neuro-ophthalmic deficits were more common after certain types of head injury might allow the clinician to have a higher index of suspicion for certain abnormalities when examining these patients. LOC implies head injury severe enough to cause brainstem reticular formation or bihemispheric dysfunction through
diffuse axonal injury, parenchymal contusion, or ICH. Several studies have found a correlation between LOC and particular neuro-ophthalmic deficits; however, LOC was not significantly associated with any outcome in our study. This finding would suggest that although LOC may be an indirect indicator of the severity of head injury, it may not be a reliable predictor of neuro-ophthalmic outcomes. The presence of a neuroimaging abnormality (skull fracture, contusion, or ICH) would suggest a greater severity of head trauma and perhaps a more frequent occurrence of certain neuro-ophthalmic deficits. In our study, the presence of an ICH was significantly and strongly correlated to unilateral oculomotor palsy. The presence of a skull fracture was significantly, but weakly, correlated to unilateral abducens nerve palsy. The specific location and extent of the neuroimaging abnormalities were not available in all patients; however, the information available would suggest that the presence of a neuroimaging abnormality, especially ICH, should increase the index of suspicion for an ocular motor nerve injury, particularly of the oculomotor nerve.

CONCLUSION

Finally, it is worth mentioning that more than one-third (34.3%) of our patients with neuro-ophthalmic deficits had normal neuroimaging. Therefore, even in the absence of any neuroimaging abnormality, the prevalence of neuro-ophthalmic findings is high.

REFERENCES


Source of Support: Nil, Conflict of Interest: None declared.