

Neuro-ophthalmological Findings in Closed Head Trauma

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Visual abnormalities following closed head trauma are common and can affect any part of the visual pathway. We reviewed 181 consecutive patients referred with visual complaints following closed head trauma. A motor vehicle accident was the most common etiology of trauma in 57% of cases, direct trauma to the head in 15%, and injuries sustained from a fall in 13%. Sixty-three percent of patients lost consciousness and 26% suffered a skull fracture. Thirty-five percent of all patients had visual field defects with functional (tunnel) fields the most common. Over 88% of eyes had 20/20 or better visual acuity. Thirty-three percent of patients suffered a cranial nerve palsy, with 75% resolving without intervention. The severity of head injury was directly related to the lack of proper seat belt and helmet use. Most visual complaints were improved with a simple refraction. Most binocular diplopia cleared with time, with only 15 cases requiring surgical correction.

Key Words: Closed head trauma—Tunnel visual fields—Cranial nerve palsy—Basilar skull fracture.

Neuro-ophthalmological abnormalities are common after closed head injuries, and can be challenging in both diagnosis and management. Many visual complaints of victims of closed head injury are sometimes ignored or misdiagnosed for a long period of time, hampering neurological, emotional, and vocational rehabilitation. Many such patients are ultimately referred to the neuro-ophthalmology clinic for vague visual complaints that appear to hamper the patient's rehabilitative progress. These referrals are often not initiated by a physician, but by a family member or rehabilitative personnel. The cause of visual complaints is often quite simple to resolve, but occasionally surprising in origin.

After several such surprises, we decided to retrospectively review a series of consecutive referrals of closed head trauma victims to the neuro-ophthalmology clinic, observing the reason for the referral, as well as the neuro-ophthalmological findings. This review is not a comprehensive compilation of all acute neuro-ophthalmological abnormalities in a series of consecutive closed head injuries, because our patients represent a select group, most of which were delayed referrals for specific ocular complaints. Therefore, it should not be concluded that our data represent an overall rate of occurrence of ophthalmic findings following closed head injury.

MATERIALS AND METHODS

The outpatient records of 188 consecutive closed head trauma patients referred for neuro-ophthalmologic examination to the Dean A. McGee Eye Institute from July 1, 1986 to January 21, 1991, were reviewed retrospectively. Seven were excluded for preexisting ocular disease (Table 1). The remaining 181 patients underwent a thorough

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TABLE 1. Reasons for exclusion of patients from study (*n* = 7) (patients with associated "other" ocular diseases)

Eales' Disease	1
Bilateral phthisis	1
Pseudotumor cerebri	1
Multiple sclerosis	1
Brainstem tumor	1
Myasthenia gravis	1
Congenital ocular abnormality	1

neuro-ophthalmologic history and physical examination. Length of time between injury and examination, type of trauma incurred, extent of injuries, loss of consciousness, and associated skull fractures were noted for each patient.

Those patients with associated abnormalities were followed for an average of 6 months (range from 1 month to 3 years) until resolution of their problem or appropriate treatment with correction was rendered.

RESULTS

Of the 181 patients included in this study, 125 were male and 56 were female. Patients ranged in age from 5 to 74 years, with a mean age of 31 years. The average time from injury to time of examination was 8.5 months, with a range of 3 weeks to 3 years.

A motor vehicle accident (MVA) was the most common cause of closed head trauma in 57% of patients. This was followed in frequency by direct trauma to the skull (i.e., assault with a fist or instrument) in 15%, fall from a platform or ladder in 13%, and a motorcycle accident (MCA) in 9% (Table 2). Of the 103 patients involved in motor vehicle accidents, 77% were drivers. Interestingly, 87 (84%) of all MVA victims were not wearing seat belts. Only 16 of the 103 patients involved in a motor vehicle accident were wearing safety belts. All seat-belted patients did well both neurologically and visually, with only 5 losing consciousness for a brief time. Only 2 seat-belted victims had

TABLE 2. Etiology of closed head trauma (*n* = 181)

Motor vehicle accident	103	(57%)
Blow to head (assault, etc.)	27	(15%)
Fall (platform, ladder etc.)	23	(13%)
Motorcycle accident	16	(9%)
Three- or four-wheeler	5	(3%)
Bicycle	3	(2%)
Train accident	2	(1%)
Mountain-climbing accident	1	(<1%)
Explosion	1	(<1%)

a cranial nerve palsy (unilateral and bilateral sixth). None had visual field defects or associated skull fractures.

Of the 23 patients involved in falls, the average estimated distance of the fall was 20 feet, usually onto a concrete surface.

Most of the victims of MCA were male (81%; mean age 29 years), and 14 of 16 did not wear helmets. Only 5 patients, with a mean age of 18 years, were involved in three- or four-wheeler accidents. None of these 5 were wearing helmets.

One hundred fifteen (64%) of the 181 patients experienced a loss of consciousness from a few minutes to 5 months in length. Forty-seven (26%) of the 181 patients had associated skull fractures, 10 of which were basilar skull fractures. Only 19 patients with a skull fracture required surgical debridement or decompression.

The most common presenting complaint was that of blurred or decreased vision in 46% of patients. This was followed by diplopia in 30% and headaches in 13% (Table 3). A headache history consistent with postconcussive migraine was elicited in 29% of patients.

Presenting visual acuity was 20/20 or better in 319 (88%) of 361 eyes (181 patients). One individual underwent enucleation at the time of his injury secondary to severe ocular trauma. Seventeen eyes were 20/30–20/60, and the remaining 26 eyes were 20/100 or worse. Optic atrophy was present in 7 eyes of the 20/30–20/60 group and in 19 eyes of the 20/100 or worse group. Amblyopia, cataract, cortical blindness, and functional visual loss were the remaining causes of visual loss.

All patients underwent Goldmann perimetry visual field testing followed by tangent screen testing if abnormal or otherwise indicated. Sixty-four (35%) of the patients had visual field defects. Much to our surprise, of those 64 with field defects, functional (tunnel) fields were the most common (41%). The next most frequent field defects in-

TABLE 3. Presenting chief complaint in closed head injuries

Blurred or decreased vision	83	(46%)
Double vision	55	(30%)
Headaches	23	(13%)
Trouble reading	10	(6%)
Eye irritation	5	(3%)
Oscillopsia	4	(2%)
Irregular pupils	4	(2%)
Sagging lids	4	(2%)
Flashes of light	4	(2%)

Note: Some patients have more than one chief complaint (i.e., double vision and headache).

cluded arcuate and homonymous hemianopsia (Table 4). A complicating observation was the association of postconcussive migraine and functional visual loss in 14 of the 26 patients with functional visual fields. Of the 38 "nonfunctional" visual field defects, 34 (89%) were associated with loss of consciousness.

Sixty (33%) patients were found to have suffered a palsy of at least one of the cranial nerves III, IV, and VI. Thirty-two (53%) of these 60 cases were associated with skull fractures, including all 10 of the basilar skull fractures. Ninety-one percent of patients having a cranial nerve palsy experienced a loss of consciousness. Of those suffering a cranial nerve palsy, the fourth cranial nerve was involved either unilaterally or bilaterally in 40% of cases, the third cranial nerve in 33% of cases, and sixth cranial nerve in 27% of cases (Table 5). There were no motility disturbances solely attributable to orbital wall or floor fractures.

Only 15 (25%) of patients with a cranial nerve palsy required surgical correction. The remaining 45 resolved without sequelae. The most common palsies requiring surgical correction involved the third and fourth cranial nerves (Table 6). All but one of the cases with unilateral involvement were corrected with one surgery, while 6 of the 8 cases with bilateral involvement required two or more surgeries for correction. Aggressive care consisting of extensive lubrication and a possible tarsorrhaphy were required in patients with peripheral seventh nerve palsies, particularly when associated with a sensory fifth nerve palsy (2 patients).

Funduscopy examination was found to be normal in 343 (95%) of 361 eyes (181 patients). Optic atrophy was found in the remaining 18 eyes. Optic nerve contusion was presumed the cause of atrophy in 14 eyes, chronic papilledema in 2 eyes (one bilateral case), and chiasmal trauma in 2 eyes.

Other associated neuro-ophthalmological findings were 6 patients with a decompensated phoria, 4 patients with vertical gaze palsy, and 3 patients with bilateral internuclear ophthalmoplegia (Table 7). One patient with internuclear ophthalmoplegia required strabismus surgery.

TABLE 4. Visual field defects (n = 64)

Functional (tunnel)	26 (41%)
Optic nerve (arcuate)	17 (26%)
Homonymous hemianopsia	13 (20%)
Homonymous quadrantanopia	6 (9%)
Inferotemporal island	1 (2%)
Cortical blindness	1 (2%)

TABLE 5. Cranial nerves III, IV, VI affected following closed head trauma (n = 60)

Cranial nerve	Unilateral	Bilateral	Total	Percent
3	10	10	20	33%
4	15	9	24	40%
6	8	8	16	27%

DISCUSSION

Visual abnormalities following closed head trauma are not uncommon and can affect any part of the visual system. As in our review, the most common cause of closed head trauma in the United States is a motor vehicle accident (1-4). The relationship of seat belt use to the lack of severe injury was impressive in our study, but not surprising (3). The practice of not wearing a helmet, for whatever reason, while riding a motorcycle, or three- or four-wheeler, was associated with a much more severe neurologic and neuro-ophthalmologic injury.

The severity of head injury in motorcycle accidents is well known, and controversies with state helmet laws continue, in spite of the obvious relationships (5,6). All 19 of our unhelmeted motorcycle accident patients suffered a loss of consciousness, with 16 remaining comatose for longer than a week. Fourteen (74%) of these MCA victims sustained multiple cranial nerve palsies, with the majority suffering skull fractures (63%).

Our findings suggest that there is a higher association of neuro-ophthalmological abnormalities in patients who do not wear seat belts or helmets and that their more severe closed head injuries are associated with a higher incidence of coma, skull fracture, and cranial nerve palsy. The association of basilar skull fracture and multiple cranial nerve palsies was particularly high.

Closed head injury resulting in loss of consciousness often causes permanent neurologic sequelae (7). Fifty-nine (51%) of our patients with loss of consciousness were left with a cranial nerve palsy, and nearly all of our patients with nonfunc-

TABLE 6. Strabismus surgery for cranial nerve palsy (n = 15)

Bilateral 4th nerve	4
Unilateral 4th nerve	5
Bilateral 3rd nerve	3
Unilateral 3rd nerve	1
Bilateral 3rd and 4th nerves	1
Bilateral 6th nerve	1

TABLE 7. Associated neuro-ophthalmologic findings

Decompensated phoria	6
Vertical gaze palsy	4
Bilateral internuclear ophthalmoplegia	3
Unilateral internuclear ophthalmoplegia	1
Convergence insufficiency	4
Downbeat nystagmus	3
See-saw nystagmus	1
Macro square wave jerks	1
Dorsal midbrain syndrome	1
Horner's syndrome	1
Skew deviation	1

tional visual field defects experienced coma (87%). A head blow that is severe enough to cause brainstem contusion and therefore disrupt reticular formation function, resulting in unconsciousness, can often result from coup contra coup-type injury. This certainly could explain the association of coma, cranial nerve palsy, and parieto-occipital lobe visual field defects seen in many of our patients.

The most common chief complaint elicited was that of blurred or decreased vision. Yet, over 88% of our patients's eyes had 20/20 or better best corrected visual acuity. This can best be explained by the fact that many (if not all) of these patients had either an incorrect refraction or no refraction at all. This simple explanation has been noted previously (8). Although diplopia is sometimes misinterpreted as blurred vision by the patient, our experience suggests that most complaints of blurred or decreased vision can be cured with a sometimes difficult but accurate refraction. Care should be taken to separate monocular from binocular diplopia. Monocular diplopia usually results from a refractive error, cataract, or dry eye state.

A history consistent with postconcussive migraine was elicited in almost 30% of our patients. The development of migraine headaches following closed head trauma is well recognized. Many patients have a history of migraine headaches which are exacerbated by the head trauma, while others have a new onset of migraine headaches following head trauma. Over 25% of the patients in our study with postconcussive migraine were associated with functional (tunnel) fields. This association can make not only the diagnosis, but the treatment of these patients very challenging.

Interestingly, the most common visual field defect in our study was "tunnel" or functional visual fields. Functional visual fields were only suspected when no other organic pathology was present to explain the field defect, such as an abnormal pupil or retinal exam. Visual field defects following closed head trauma are not uncommon (35% in our

series). At its most severe extent, cortical blindness has been observed (9,10). Other defects, ranging from a quadrantanopia or bitemporal hemianopia to a complete homonymous hemianopia, are not uncommon (11-13). Our study included patients with all of the above-mentioned visual field problems.

The closed space within the cranium, with its rigid bony case, leads to the susceptibility of pressure effects. Therefore, contusions commonly occurring in closed head trauma can bleed or develop edema resulting in pressure effects. Skull fractures also have a potential for intracranial damage, especially where the cranial nerves travel along the base of the skull. Their rather long course through the cranium also makes them susceptible to damage. Orbital fractures can lead to muscle entrapment and subsequent muscle imbalances, which were not prevalent in our review.

Cranial nerve palsy following head trauma involving one nerve or multiple nerves has been well documented, often in combination (14-19). In fact, head trauma is considered the most common cause of paralysis in fourth nerve palsy (16,18). Damage to other cranial nerves following head trauma including the first, fifth, and seventh cranial nerves has been observed (8). This seems to suggest that any cranial nerve palsy or combination of palsies can occur following closed head trauma.

Nearly 75% of the cranial nerve palsies in our series resolved without intervention. This is a higher recovery rate than previously reported (17). This higher rate could be explained by a longer recovery period prior to surgical intervention. All but one of the cases of unilateral third or fourth nerve palsies required only one surgical procedure, while 6 of the 8 bilateral cases required two or more procedures. Although the cases with bilateral involvement of the third and fourth cranial nerves can be a significant surgical challenge, resolution of diplopia in primary gaze is a realistic and obtainable goal. In our study, the number of cases of cranial nerve palsies that resolved spontaneously prior to their initial presentation is unknown because of the delay in referral.

The optic nerve is commonly injured following closed head trauma. It can be affected in up to 13% of survivors of severe head injury (20). A pathohistological review of 84 patients who died of acute closed head injury showed ischemic necrosis and shearing lesions of the anterior visual pathway in 44% (21). The most common area of injury to the optic nerve is in the intracanalicular portion where the nerve is tethered to the dura and is relatively immobile (22-24). Indirect and direct injury to the

optic nerve have been described as possible mechanisms of the nerve damage. The etiology of indirect optic nerve injury may be secondary to a stretching, tearing, torsion, or contusion of the nerve caused by the momentum of the eyeball itself and by skeletal distortion caused by forces away from the initial impact. These injuries may cause direct injury to the nerve itself or vascular insufficiency leading to nerve damage (25,26). In our study, 10% of all eyes were found to have optic atrophy secondary to indirect injury.

Other associated neuro-ophthalmologic findings have been described, including unilateral and bilateral internuclear ophthalmoplegia, pretectal syndrome, and skew deviation (27). We also found these conditions in our study, as well as cases of vertical gaze palsy, convergence insufficiency, and nystagmus. We believe these were all manifestations of posterior fossa injury.

In summary, visual complaints following closed head trauma can be of wide variety and usually require careful neuro-ophthalmologic examination. A large number of our patients had complained for months to years of visual disturbances that were either misunderstood, ignored—or worse yet—misdiagnosed. The post-traumatic patient can be a challenge to examine, with patience and thoroughness of examination often being the key to success. We found that most patients will benefit immensely from a good refraction. These refractive errors are often misinterpreted by patient and lawyer alike as being injury-related, when in fact they are not. It is not uncommon for patients to complain of diplopia many months after their injury. Fortunately, given more time (6 months to 1 year), most cases of diplopia tend to resolve without sequelae. However, if diplopia persists, it can be a real handicap to rehabilitation. The patients must understand that they do not have to “live with” diplopia (as they are often told) and that single binocular vision is often a very realistic goal. There are a variety of visual field defects found in closed head trauma, but in our experience, functional (tunnel) fields are quite common. Ultimately, closed head trauma patients should be approached with the idea that their condition can be improved; the examiner need not be intimidated by the vast possibilities of injuries.

We have consistently found that the vast majority of our closed head injury victims and their families respond with renewed hope when confronted with a physician who takes the time to address their chief complaints and provides a positive, persistent, and realistic therapeutic approach.

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