Visual Profile of Patients Presenting with Brain Trauma

Lynn Fishman Hellerstein, OD, FCOVD, FAAO
Theodore S. Kadet, OD, FCOVD

Visual abnormalities following acquired brain injury (ABI) are not uncommon and can affect any part of the visual system. Clinical findings on this patient population include binocular, ocular motor, and accommodative dysfunctions; visual field deficits; ocular pathology; and visual processing information deficits. In-depth visual evaluation of the patient with ABI is critical for documentation purposes as well as for possible treatment regimens. Key Words: acquired brain injury (ABI).

INTRODUCTION

Patients with acquired brain injury (ABI) present a diversity of visually-related characteristics and symptoms. Many visual symptoms are sometimes ignored or misdiagnosed for a long period of time, hampering neurological, emotional, and vocational rehabilitation. Factors including type of injury (open or closed head), severity (mild, moderate, or severe), etiology, presence (including duration) or absence of coma, injuries to other anatomical systems, psychological effects, etc., will affect the patient’s awareness of and ability to report visual changes.

The optometrist may be challenged to identify ABI-related visual dysfunctions. In the case history, all possible visual anomalies must be carefully explored. Many brain injury survivors report visual malaise, with only a vague awareness of specific problems. Disorientation, dizziness, nausea, headaches, and other asthenopic symptoms may be the only clue to underlying visual dysfunctions. Some patients do not even relate the observed changes to the brain trauma. The most common visual symptoms include: blurred or decreased vision, diplopia, headaches, trouble reading, eye irritation, oscillopsia.

Cohen lists the most common vision problems from TBI as:

- binocular dysfunctions (convergence insufficiency, acquired strabismus, diplopia)
- ocular motility deficits
- visual field loss
- visual perceptual-motor dysfunctions

Falk adds accommodative dysfunction. Kowal observes oculomotor cranial nerve palsies. Hellerstein found problems in near point of convergence, stereo acuity, and fusion ranges, as well as abnormal visually evoked cortical potential (VECP). Padula includes spatial disorientation, balance and postural difficulties, objects appearing to move, visual memory problems, staring behavior, and asthenopic symptoms as part of his Post Trauma Vision

Correspondence regarding this article should be addressed to Lynn Hellerstein, 7180 E. Orchard Rd, Suite 103, Englewood CO 80111.

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Syndrome. Bohnen describes the post-concussion syndrome to cover quasi-organic and subjective symptoms as headaches, dizziness, tiredness, insomnia, lowered tolerance to noise and light, irritability, and difficulties with memory or concentration.

This article discusses the types of visual problems associated with ABI. The goal for early identification of the visual sequelae is to emphasize the timely evaluation and prescribing of appropriate visual treatment. The referenced articles are suggested for elaboration and in-depth study.

DISCUSSION

Binocular and Ocular Motor Dysfunction

Beck notes ocular motor disturbances including strabismic and non-strabismic conditions. Strabismus includes esotropia, exotropia, and hypertropia in all its forms, variations and complicating conditions. Non-strabismus conditions encompass esophoria, exophoria, hyperphoria, and convergence insufficiency. Constant or periodic diplopia results from the acquired loss of binocular alignment and fusion. Strabismus or high phoria present prior to head trauma may be exacerbated or changed by the injury.

Post-trauma strabismus or high phoria is presumed due to partial or complete paralysis of Cranial Nerves III, IV, and VI. Kruger describes paresis or paralysis of the third cranial nerve as characterized by palpebral ptosis, inability to rotate the involved eye upward, downward and inward, with exo and slightly hyper deviation of the eyeball and dilated, non-reactive pupils. Beck states that fourth cranial nerve involvement presents with hyper deviation and head tilt while the sixth nerve produces an abduction deficit with an eso deviation greater at distance than near. Baker estimates that cranial nerve injuries occur in 13% of brain-injured patients, but the portion of ABI patients demonstrating some form of ocular motility disturbance is most likely higher than this.

Various authors postulate that small hemorrhages or hematomas in the vicinity of the III, IV and VI cranial nerve nuclei are responsible for the loss of binocular function. These may be the result of compression or "coup-contrecoup" impacts from the acceleration and deceleration forces to the head. Shearing of blood vessels and nerve tissue in the area of the midbrain is also suspected. Whiplash injuries seem especially prone to causing trauma-induced binocular deficits.

Falk feels that ocular motility deficits secondary to TBI are common; deficits in pursuit movements (both versions and ductions), saccades (voluntary and stimulus generated), and the presence of nystagmus and jerk nystagmus may be observed monocularly and binocularly. Bouska adds loss of range of motion to the extremes of gaze and Aksionoff discusses the inability to integrate ocular motility with visual motor. Neger includes fixation (defined by him as the maintenance of an object of conscious regard on the fovea) dysfunctions in this category.

Various areas of the brain affect ocular motility performance, including the brain stem, frontal lobe, midbrain superior colliculi, parietal lobe, temporal lobe, occipital lobe, reticular system and cerebellum. Paresis and paralysis of Cranial Nerves III, IV, and VI also impact ocular motility.

Accommodative Dysfunctions

It is common that survivors of ABI often exhibit a reduction in accommodation endurance and facility below age related norms. The reductions are usually binocular. Harrison notes disruption to the fusional vergence reflex will often affect accommodation. Burke reports the most common abnormality from whiplash injuries are disturbances of convergence and/or accommodation. Padula includes accommodative dysfunction in his Post Trauma Vision Syndrome. Horwich notes the likelihood of third cranial nerve involvement in the loss of accommodation. Vogel reports midbrain and brainstem lesions can disrupt accommodative/convergence relationships.

Visual Field Deficits

Visual field loss is common in ABI; the extent of the loss varies widely. Cohen lists total, sector, central, peripheral, and altitudinal losses along with congruous, incongruous, and homonymous defects. Fowler notes left hemisphere lesions will primarily affect the right
visual fields and vice versa. The terms macular sparing, obliteration and splitting are used to describe macular involvement in the field loss. Scotomas can be monocular or binocular and of any size and shape. Warren notes superior horizontal field defects are predominant in traumatic brain injury while homonymous hemianopsia is a more frequent sequel to cerebral vascular accident (stroke). In a retrospective study, 35% of the patients with brain injuries referred to Sabates for a neuro-ophthalmologic examination had visual field defects. Of those with field defects, functional (tunnel) fields were the most common (41%). Langerhorst proposes that this type of constriction of the visual field may reflect an organic disturbance in attentional mechanisms revealed when executing dual task commands.

Visual field losses may not be subjectively observed by the patient, as the person's awareness of this type of impairment can be dramatically compromised. Symptoms including disorientation, bumping and tripping over objects, unsteady movement, missing food on the plate, fear of moving in space, etc. will lead the examiner to suspect visual field changes.

Ocular Pathology

Optometry generally enters the ABI paradigm at the post-acute stage and into recovery. Diagnosis and treatment of the functional sequelae from the brain and other structural insults becomes the optometrists’ primary role. Involvement at an acute level requires knowledge of standard medical protocols for all tissues of the eye and orbit. Other professions may assist the optometrist in diagnosing underlying pathology. More efficient and effective prognoses and treatment outcomes result from this knowledge.

Visual Information Processing

Aksionoff proposes a “Model for Information Processing” which categorizes input, integrative and output stages. Visual perception takes place at the integrative stage and includes spatial relations, visual sequencing, visual memory, visual closure, form constancy, figure ground analysis, visual auditory integration, and visual motor integration. Warren adds visual attention and scanning, pattern recognition, and visual cognition.

Padula observes spatial disorientation, balance and posture difficulties and staring behavior. Robert presents visual disorders of higher cortical function which includes hemi-inattention, visual agnosia, and alexia. Patients with ABI often present with post-trauma losses in some or all of the above visual perception skill areas. Family members, friends and other professionals may be more aware of these losses than the survivor. Short term memory function and speed of information processing skills are exceptionally affected by ABI.

Bohnen reports on visual and auditory hyperesthesia, the inability to endure intense light and sound. A further study found 25% of TBI patients were not able to endure intense stimuli by five weeks. Tierney indicates symptoms of anterior pathway disease include dimness of vision, poor visual acuity, reduced color intensity, positive visual phenomena (photopsia, photophobia), sound-induced photism, and increased visual disturbance in fluorescent lighting.

CONCLUSION

Patients with ABI frequently exhibit varied forms of visual dysfunction. Vision evaluation and management can substantially contribute to understanding the patient’s injury, recovery, and rehabilitation.

REFERENCES


