Central Vestibulopathy in a Female Collegiate Basketball Player: A Case Study

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Abstract: We present a unique case of a collegiate athlete who suffered from a mild head injury following a multi-car motor vehicle accident and was later diagnosed with central vestibulopathy.

A concussion is a mild head injury that involves a physiological disruption of brain function with at least one of four characteristics: (a) any period of loss of consciousness; (b) any retrograde or anterograde amnesia; (c) any immediate alteration of mental status; and (d) any neurological deficits (Kay et al., 1993). A history of brief unconsciousness is considered an indication for hospitalization because of the risk of delayed complications, such as a hematoma which is a collection of blood around the brain (Jennett, 1976). Approximately 90% of new head injury cases each year are diagnosed as a concussion (Gottshall, Drake, Gray, McDonald, & Hoffer, 2003).

While many concussions are diagnosed each year, certified athletic trainers and team physicians often have difficulty detecting and classifying concussions because of the varying signs and symptoms (Kelly & Torg, 1995). The most common signs and symptoms of concussion are headaches, dizziness, fatigue, irritability, anxiety, insomnia, loss of concentration and memory, and noise sensitivity (Dikmen, McLean, & Temkin, 1986; Edna, 1987; Edna & Cappelen, 1987; Minderhoud, Boelens, Huizenga, & Saan, 1980). Evaluation of concussion is difficult with a multitude of signs and symptoms that are often misleading or not reported.

The National Collegiate Athletic Association Injury Surveillance System describes a concussion based on a grading scale with three grades. A Grade I concussion is depicted by no loss of consciousness and a short amount of posttraumatic amnesia. An athlete with a Grade II concussion would likely suffer from loss of consciousness for less than 5 min and amnesia can last up to 30 min. A Grade III is a severe concussion and constitutes loss of consciousness for more than 5 min and extended amnesia beyond 30 min (Cantu, 1986). Neurons or brain cells can become damaged during a concussion. A neuron consists of a nerve cell body, Nissl bodies, nucleus, neuroglial cells, axon, dendrites, myelin sheath, and axon terminal. Following a concussion trauma causes twisting and tearing of the neuronal axon and ultimately leads to death of the cell. Figure 1 illustrates the trauma that occurs to neurons as a result of a concussion. Although the symptoms of a concussion may persist for a prolonged period of time especially when dealing with a severe concussion; evidence supports that some post-concussive symptoms may demonstrate an alternate diagnoses such as vestibular deficits (Gottshall et al., 2003). We intend to present a unique case of a female collegiate basketball player suffering from post-concussive syndrome which resulted in central vestibulopathy.

Background and Injury History

Concussion incident. A 19-year old (height = 177.8 cm, body mass = 75.0 kg) National Collegiate Athletics Association Division I female basketball player sustained a mild traumatic brain injury following a multi-car motor vehicle accident during winter break of her freshman year. The athlete reported being a passenger in the multi-car accident, hitting her head on the visor, and immediately losing consciousness up to 1 min. The Emergency Medical Technicians
(EMTs) arrived on the scene and the athlete refused emergency transportation to the hospital, but was briefly evaluated. During the examination the athlete described feeling “off balance” and “wobbly,” confused, and fatigued. The EMTs assessed the primary complaint as a Grade II concussion. The following day, the athlete boarded an airplane to return to the University and experienced an increase in headaches, light-headedness, and dizziness as a result of air travel. One day following the accident, the athlete reported to the University sports medicine facility where the Certified Athletic Trainer evaluated the condition as a Grade II concussion secondary to mild head trauma and immediately referred her to a local hospital for further evaluation.

The team physician at the local hospital diagnosed the athlete with a Grade II concussion, in agreement with the assessment of the EMTs and the Certified Athletic Trainer. A computer tomography (CT) scan revealed no significant brain trauma. Because headaches and reports of imbalance had not subsided by the time of the evaluation, the physician restricted the athlete from basketball practice and prescribed a series of medications to control her pain and dizziness: Tylenol #3 (acetaminophen and codeine), Medrol (methylprednisolone), Bextra (valdecoxib), and a combination of Cipro (ciprofloxacin) and Mucinex (guaifenesin).

Follow-up evaluations. The athlete reported daily to the sports medicine facility where she was evaluated by the sports medicine team. As her headaches became less severe, she began stationary cycling to maintain her cardiovascular fitness. The athlete progressed to participation in light shooting drill practices. On days when the headaches subsided, the athlete was allowed to participate in passing drills, but the headaches returned and she was again restricted from activity. One month following the initial injury, the headaches and imbalance had not subsided and the athlete was referred to the team physician for additional diagnostic tests (another CT scan and one magnetic resonance imaging scan). The results of the diagnostic testing were unremarkable and did not explain the continued symptoms. Another month passed and the team physician was concerned about the post-concussion syndrome symptoms and the athlete was referred to a Registered Physical Therapist with a national certification in vestibular pathologies.

Methods

The subject of this case report was selected to document an unusual condition encountered by the primary investigator during the clinical education component of her athletic training education program. The subject presented to the primary investigator within a day of the initial motor vehicle accident and the subject was followed throughout her course of injury management to successful return to full activity. A signed release of medical information was obtained in compliance with Florida International University’s Internal Review Board policies. The research design was descriptive research that retrospectively explored medical records including injury reports, head injury questionnaires, medical referral forms, diagnostic reports, evaluation summaries, and progress reports from the university athletic training room. Diagnostic reports including two CT scans and one magnetic resonance image were also collected. Compilations of data from the vestibular specialist included Sensory Organization Test (SOT), a Head Shake SOT in the yaw (vertical axis) and pitch (interaural axis) plane, a Motor Control Test, a Stationary March Test, a bilateral Head-Thrust Test, and a Headshake Test in the yaw and pitch plane. Data were synthesized, interpreted, and presented to contribute to the clinical knowledge base of this unique condition.

Results

Central Vestibulopathy Diagnosis

During the evaluation conducted by the vestibular specialist the athlete reported tinnitus, aural fullness, and pressure in the ears and head (Specialists Notes, 2004). The vestibular
specialist performed a series of tests on the athlete during her first visit. The tests included (a) a SOT revealing a multisensory dysfunction pattern likely originating from the central nervous system (see Figures 2 and 3); (b) a Head Shake SOT in the yaw and pitch plane (Demer, Goldberg, & Porter, 1991) resulting in several falls; (c) a Motor Control Test was within normal limits; and (d) a Stationary March Test with abnormal findings; (e) a bilateral Head-thrust Test, which was positive; and (f) a Headshake Test in the yaw and pitch plane resulting in downbeating nystagmus supports. The final diagnosis vestibular specialist’s final diagnosis was a poorly compensating central vestibulopathy with persistent headaches associated with a retinal slip secondary to a weakness in the vestibule-ocular reflex (Specialists Notes, 2004).

Rehabilitation Program
The vestibular specialist provided exercises to challenge the vestibule-ocular reflex such as standing on a foam pad with feet together, while holding a business card, with one hand, straight out in front of her. The athlete was instructed to focus on one word on the business card and shake her head left to right for a count of ten, while maintaining her focus on the word and staying balanced. The athlete was then instructed to shake her head up and down for a count of ten, while again maintaining her focus on the word and staying balanced. The athlete was prescribed this regimen of exercises two to three times a day with the certified athletic trainer and returned in three weeks for re-evaluation and follow-up. (Specialists Notes, 2004)

Two months later the athlete returned to the vestibular specialist and reported compliance with her home exercise program. The athlete also reported a significant reduction in the intensity and frequency of her headaches and her balance during her course of treatment with the home exercise program. The SOT, Head Shake SOT in the yaw and pitch plane, Motor Control Test, Stationary March Test, Headshake Test in the yaw plane, and bilateral Head-thrust Test were repeated and were all within normal limits. The athlete did continue to demonstrate some deficits with the Headshake Test in the pitch plane, but was improving (Specialists Notes, 2004).

Discussion
The athlete sustained a concussion, ultimately leading to poorly compensated central vestibulopathy (Specialists Notes, 2004). Vestibular disorders are a potentially significant disability often associated with post-concussion syndrome (Gottshall et al., 2003). Vestibulopathy is any abnormality of the vestibular apparatus, which is the receptor organ of the vestibular portion of the eighth cranial nerve that is concerned with balance and equilibrium (Venes, 2004). A slowly developing vestibulopathy can be relatively silent because the function of the vestibular system overlaps other sensory systems (Baloh, Jacobson, & Honrubia, 1989). The athlete’s persistent headaches were associated with a retinal slip secondary to a weakness in the vestibule-ocular reflex. The vestibular-ocular reflex is the only stabilizer for the retina during externally imposed, sudden, head accelerations (Demer, Crane, Tian, & Wiest, 1996). Sudden acceleration and deceleration of the head, especially following a motor vehicle accident, can cause significant retinal slip and may overcome the protective vestibulo-ocular reflex (Herdman, Schubert, & Tusa, 2001). Loss of the vestibular-ocular reflex leads to complaints of imbalance and visual distortion (Baloh et al., 1989). Therefore, mild head trauma resulting in impairment of the vestibular-ocular reflex may present the aforementioned deficits over a prolonged period of time.

In our case, four months of vestibular exercises resulted in complete elimination of the post-concussive symptoms of imbalance. However, the athlete continues to suffer from post concussion syndrome with headaches on occasion although she has been cleared for full participation and rarely abstains from practices or games. Her current course of treatment
includes overall body strengthening and conditioning as well as prescriptions for Tylenol #3 for
minor headaches and Bextra for her more severe headaches. In the general American population,
5% sustain some type of head injury annually. Following head trauma, vestibular dysfunctions
may represent one of the largest groups of delayed complications. Patients, who have
experienced a head injury, exhibit vertigo and dizziness between 20-58% of cases (Rubin,
Woolley, Dailey, & Goebel, 1995). Vestibular injuries, disturbances, or disorders are a
significant disability after head injury or trauma (Gagnon, Friedman, Swaine, & Forget, 2001;
Gottshall et al., 2003; Hoffer, Gottshall, Moore, Balough, & Wester, 2004; Kisilevski et al.,
2001; Mallinson, & Longridge, 1998; Rubin et al., 1995; Tuohimaa, 1978). However; this
significance is rarely reported in the literature among athletes. Vestibulopathies are not part of
entry-level athletic training competencies and proficiencies; therefore, it is important to
recognize the limitations within the athletic trainers’ scope of practice and refer these athletes to
the appropriate medical professionals/specialists for additional testing and treatment. Treatment
can improve the rate of recovery significantly (Hoffer et al., 2004). In this case it was the use of a
vestibular specialist that helped the athlete’s recovery and successful return to competition. It is
also vital to implement the appropriate course of pharmacological medication and treatments for
successful return to athletic competition.

Clinical Implications
We presented this case to demonstrate an association between concussions and central
vestibulopathy. Certified athletic trainers often evaluate concussions based on reports of mild
head injury, headaches, tinnitus, dizziness, and nausea. A concussion is a complex pathology
integrating several aspects of the central nervous system requiring knowledge of neuroanatomy
and pathophysiology. To provide the best care for athletic individuals, certified athletic trainers
must be aware of non-athletic injuries and pharmacology as well as their impact on performance.

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*Figure 1.* Axon Shear (Post-concussion Syndrome) with neurons (brain cells) subject to axonal shearing or post-concussion syndrome. Normal neuron with the nerve cell body, Nissl bodies, nucleus, neuroglial cells, axon, dendrites, myelin sheath, and axon terminal. *Figure 1A.* Trauma causes twisting and tearing of the neuronal axon. *Figure 1B.* Post-trauma condition with death of the brain cell. From Nucleus Medical Art, Inc. (2004). Retrieved December 8, 2004, from http://hon.nucleusinc.com/generateexhibit.php?ID=1870.
Figure 2. Sensory Organization Test. Results of sensory organization testing at two months and at four months following motor-vehicle accident. Composite score was 60 at two months and 84 at four months (normal value = 85).

Figure 3. Sensory Analysis. The sensory analysis indicated a vestibular (VEST) deficit at two months following injury, which dissipated at four months following injury.