Peripheral Vestibular Disorders in Children and Adolescents with Concussion

Jacob R. Brodsky, MD1,2, Talia N. Shoshany1,2, Sophie Lipson1,2, and Guangwei Zhou, ScD1,2

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Abstract

Objective. To review peripheral vestibular disorders in pediatric patients with dizziness following concussion.

Study Design. Case series with chart review.

Setting. Pediatric vestibular clinic and pediatric multidisciplinary concussion clinic at a tertiary level pediatric hospital.

Subjects and Methods. We retrospectively reviewed 109 patients seen for dizziness following a concussion between September 2012 and July 2015. Patients were ≤20 years of age at the time of concussion. Incidences of specific peripheral vestibular disorders were assessed along with timing of diagnosis relative to the date of injury, diagnostic test findings, and treatment interventions associated with those diagnoses.

Results. Twenty-eight patients (25.7%) were diagnosed with peripheral vestibular disorders. None of these disorders were diagnosed prior to evaluation in our pediatric vestibular clinic or our multidisciplinary concussion clinic, which occurred a mean of 133 days (95% confidence interval, 89.2-177.3) after injury. Benign paroxysmal positioning vertigo was diagnosed in 19 patients, all of whom underwent successful canalith repositioning maneuvers. Other diagnoses included temporal bone fracture (n = 3), labyrinthine concussion (n = 2), perilymphatic fistula (n = 2), and superior semicircular canal dehiscence (n = 2). Both patients with perilymphatic fistula and 1 patient with superior semicircular canal dehiscence underwent successful surgical management, while 1 patient with superior semicircular canal dehiscence was managed nonsurgically.

Conclusion. Peripheral vestibular disorders may occur in pediatric patients with dizziness following concussion, but these disorders may not be recognized until symptoms have persisted for several weeks. An algorithm is proposed to guide the diagnosis and management of peripheral vestibular disorders in pediatric patients with concussion.

Keywords

concussion, postconcussion syndrome, vertigo, benign paroxysmal positional vertigo, BPPV, pediatric

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Every year, approximately 1.1 million people in the United States experience a concussion, or mild traumatic brain injury.1 Concussion is defined as a complex of pathophysiologic processes resulting from direct or indirect traumatic forces to the head that disrupts the function of the brain.2 Approximately 1 in every 220 pediatric patients seen in US emergency departments is diagnosed with a concussion, which represents about 144,000 concussion-related visits annually.3 Most individuals will report symptom resolution within a month after a concussion, but some will have sustained symptoms lasting for weeks, months, or even years after their injury.4 The World Health Organization defines postconcussion syndrome (PCS) as the persistence of symptoms for greater than 4 weeks after a concussion.5 The reported incidence of PCS among concussion patients varies widely, although estimates from large-scale studies generally range between 12% and 30%.6-9

Dizziness is the second most common symptom of concussion and of PCS. Also, dizziness has been shown to be the only on-field symptom of sports-related concussion that is independently predictive of a prolonged recovery, yielding a 6-fold increased risk of developing PCS compared to those without dizziness at the time of injury.10,11 Generally, dizziness after concussion is attributed to the concussive effects on the brain.12 Traumatic ailments involving the peripheral vestibular system are known to occur following major traumatic brain injury,13 but the role of peripheral vestibular disorders (PVDs) in dizziness

1Department of Otolaryngology and Communication Enhancement, Boston Children’s Hospital, Boston, Massachusetts, USA
2Harvard Medical School, Boston, Massachusetts, USA

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Corresponding Author:
Jacob R. Brodsky, MD, Department of Otolaryngology and Communication Enhancement, Boston Children’s Hospital, 300 Longwood Avenue, Boston, MA 02115, USA.
Email: jacob.brodsky@childrens.harvard.edu
following concussion has not been well studied, particularly in the pediatric population. Many medical care providers on the front lines of pediatric concussion evaluation and management (eg, sports medicine physicians, primary care pediatricians, and pediatric neurologists) are not well trained in the identification and management of PVDs. The objective of this study was to review the clinical aspect of PVDs in children and adolescents with dizziness in the setting of concussion and PCS.

**Methods**

We retrospectively reviewed our internal database of all patients seen at the Brain Injury Center multidisciplinary concussion clinic (MCC) and/or at the Balance and Vestibular Program clinic in Boston Children’s Hospital from September 2012 through July 2015. The Brain Injury Center MCC includes same-day evaluations by multiple pediatric specialists, including an otolaryngologist (J.R.B.), ophthalmologist, optometrist, physical therapist, and a psychologist, as well as either a sports medicine physician or a neurologist (alternates monthly).

All patients 20 years or younger who presented with symptoms of dizziness following a concussion were identified. Concussion was diagnosed based on the diagnostic criteria outlined by the 2012 International Consensus on Concussion in Sports. The electronic medical records of 109 patients who met inclusion criteria were reviewed, including clinical notes by all providers involved in the patients’ concussion-related care. Records of patients diagnosed with PVDs were further reviewed to determine timing of diagnosis relative to injury date, features of clinical presentation, diagnostic testing results, and modes of treatment. This study was approved by the Institutional Review Board of Boston Children’s Hospital.

A number of patients underwent a vestibular test battery due to symptoms and/or examination findings that were suggestive of peripheral vestibular dysfunction. The specific tests included in the battery varied between patients and were selected by the treating providers based on a number of factors, including patient age, level of cooperation, and the specific disorders that were suspected for a given patient. Vestibular tests were conducted by a licensed audiologist (G.W.Z.) with the aid of a trained assistant. Video head impulse testing (VHIT) was conducted using the ICS Impulse system (GN Otometrics, Taastrup, Denmark). Rotary chair and videonystagmography (VNG) were performed using the Micromedical System 2000 (Micromedical Technologies, Chatham, Illinois) with multifrequency sinusoidal harmonic acceleration. Bithermal caloric testing was conducted using water stimulation with the Micromedical VisualEyes with Aqua Stim system (Micromedical Technologies). Computerized dynamic posturography was conducted with the NeuroCom SMART EquiTest (Natus Medical, San Carlos, California). Cervical vestibular evoked myogenic potential testing (cVEMP) was recorded with Bio-logic Navigator Pro Evoked Potential system (Natus Medical).

Statistical analyses were performed using the Statistical Package for the Social Sciences (SPSS v.23; SPSS, Inc, an IBM Company, Chicago, Illinois). Patients were divided into groups of those with PVDs and those without (non-PVDs) for comparison.

**Results**

Twenty-eight patients (25.7%) were diagnosed with a PVD out of 109 patients seen for dizziness following a concussion. Demographics of included patients are outlined in Table 1. Mean age for the entire sample was 14 years (95% confidence interval [CI], 13.8-14.9). Mean age for those with a PVD was 14 years (95% CI, 12.6-15.0), and sex distribution was 17 males (61%) and 11 females (39%). For those diagnosed with a PVD, evaluation in our clinic occurred at a mean of 133 days (95% CI, 89.2-177.3) after their injury. No patients in the study sample were diagnosed with a PVD prior to being evaluated in our clinic.

The injury was sports related in 14 of the patients with a PVD, with 5 resulting from impact with another player and the other 9 resulting from impact with a ball or the ground. Injuries in the remaining patients with PVDs resulted from a motor vehicle crash in 4 patients and from impact with a table, door, or wall in the remaining 10 patients.

Results of vestibular testing are shown in Table 2. Of the 28 patients who were diagnosed with a PVD, 20 underwent vestibular testing (71%). Of the 81 patients who were not diagnosed with a PVD, 39 underwent vestibular testing (48%). The distribution of specific peripheral vestibular diagnoses is summarized in Figure 1. The most common PVD was benign paroxysmal positioning vertigo (BPPV), which was identified in 19 patients. Patients with BPPV were seen in our clinic and subsequently diagnosed with BPPV at a mean of 19 weeks after the inciting injury. All patients with BPPV reported exacerbation of dizziness with head movements when supine. No patients with BPPV had hearing loss. All BPPV patients were diagnosed with posterior canal involvement, with the exception of 1 patient with lateral canal BPPV and 1 patient with superior canal BPPV. Posterior canal BPPV was diagnosed by a Dix-Hallpike test that was positive for torsional and up-beating nystagmus.

<table>
<thead>
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<th>Table 1. Demographics.</th>
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<tr>
<td>Age, y</td>
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<tr>
<td>Mean (95% CI)</td>
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<tr>
<td>Sex, No. (%)</td>
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<tr>
<td>Female</td>
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<tr>
<td>Male</td>
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<tr>
<td>Time to evaluation, d</td>
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<tr>
<td>Mean (95% CI)</td>
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<td>History of concussion, No. (%)</td>
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Abbreviations: CI, confidence interval; PVD, peripheral vestibular disorder.
accompanied by subjective vertigo with the affected ear down. Lateral canal BPPV was diagnosed by a head roll test that was positive for geotropic, horizontal nystagmus accompanied by subjective vertigo with the affected ear down. Superior canal BPPV was diagnosed by a Dix-Hallpike test that was positive for torsional and down-beating nystagmus accompanied by subjective vertigo with the affected ear up. All patients who were diagnosed with BPPV were successfully treated with canalith repositioning maneuvers.

Perilymphatic fistula (PLF) was diagnosed in 2 patients, both of whom had normal vestibular testing, and a normal temporal bone computed tomography (CT). Both also had symptoms of sound- and pressure-induced vertigo in the affected ear only. One patient had a moderate conductive hearing loss and was found on middle ear exploration to have a stapes footplate fracture with associated incudal dislocation and perilymphatic leak from the oval window via the footplate fracture, while the other patient with PLF had a leak from the round window. Both PLFs were successfully repaired with perichondrial grafts secured with fibrin glue.

Superior semicircular canal dehiscence (SSCD) was diagnosed in 2 patients, both of whom had symptoms of sound-induced vertigo, conductive hyperacusis, and a positive Tullio’s phenomenon. Both patients with SSCD had normal hearing but showed reduced thresholds and augmented amplitudes on cervical VEMP testing in the affected ear. Both patients had SSCD confirmed radiologically on fine-cut temporal bone CT with short- and long-axis oblique reformats through the plane of the superior semicircular canal. One patient with SSCD was treated surgically with resolution of symptoms postoperatively, while the other opted not to undergo surgery due to relatively mild symptoms that resolved with a course of vestibular rehabilitation.

An occult temporal bone fracture was identified by CT of the temporal bone in 3 patients. The impacts associated with these injuries were consistent with major traumatic brain injury (TBI); however, they were originally diagnosed as concussion by referring providers. The fracture involved the vestibular labyrinth in all 3 cases. Labyrinthine concussion was diagnosed in 2 patients, which was defined as a complete unilateral peripheral vestibular loss demonstrated on vestibular testing with no evidence of fracture on temporal bone CT and no signs or symptoms of a PLF. All patients with temporal bone fractures had injuries in the region of the ear and had symptoms of ear pain at the time of injury that had resolved at the time of their evaluation in our clinic. A moderate sensorineural hearing loss was identified in 1 of the 3 patients diagnosed with a temporal bone fracture and in 1 of the 2 patients diagnosed with a labyrinthine concussion. All patients with a temporal bone fracture or a labyrinthine concussion were managed with a course of vestibular rehabilitation with a physical therapist. All patients with a temporal bone fracture or a labyrinthine concussion demonstrated evidence of unilateral peripheral vestibular loss on rotary chair, caloric, VHIT, and/or cervical VEMP testing (Figure 2).

Discussion

A concussion requires direct or indirect traumatic forces to the head sufficient to impact the brain. The proximity of the inner ear structures to the brain within the skull puts it at high risk for damage from a concussive impact. Inner ear lesions affecting the vestibular system may result in vertigo

Table 2. Vestibular Testing Results for Patients Who Underwent Vestibular Testing.

<table>
<thead>
<tr>
<th>Testing</th>
<th>PVD patients</th>
<th>Non-PVD patients</th>
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<tbody>
<tr>
<td>Videoystagmography (oculomotor)</td>
<td>11</td>
<td>8</td>
</tr>
<tr>
<td>Videoystagmography (positional)</td>
<td>1</td>
<td>10</td>
</tr>
<tr>
<td>Rotary chair test</td>
<td>12</td>
<td>4</td>
</tr>
<tr>
<td>Video head impulse test</td>
<td>5</td>
<td>7</td>
</tr>
<tr>
<td>Caloric testing</td>
<td>1</td>
<td>0</td>
</tr>
<tr>
<td>Computerized dynamic postuography</td>
<td>13</td>
<td>4</td>
</tr>
<tr>
<td>Subjective visual vertical testing</td>
<td>4</td>
<td>5</td>
</tr>
<tr>
<td>Cervical vestibular evoked myogenic potential</td>
<td>15</td>
<td>4</td>
</tr>
<tr>
<td>Ocular vestibular evoked myogenic potential</td>
<td>4</td>
<td>2</td>
</tr>
<tr>
<td>Dynamic visual acuity</td>
<td>4</td>
<td>4</td>
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Abbreviation: PVD, peripheral vestibular disorder.
that could be very difficult to differentiate from dizziness resulting from the concussive impact to the brain itself. The latter will often resolve spontaneously, but traumatic peripheral vestibular impairments may require additional interventions to resolve, such as head repositioning procedures for BPPV, vestibular rehabilitation for labyrinthine concussion or temporal bone fracture, or even surgical intervention for PLF or SSCD. Children and adolescents with PCS are typically managed by pediatricians, sports medicine providers, or neurologists, many of whom may not have had training or experience with the identification and management of inner ear disease, which may result in delayed treatment. In this study, we identified a traumatic PVD in 28 pediatric patients with sustained dizziness following a concussion. This may be a major contributing factor to the findings from prior studies that dizziness is the second most common symptom of PCS and that dizziness is the only on-field symptom that is predictive of a prolonged recovery from pediatric concussion.\(^{10,11}\)

Nineteen patients were diagnosed with BPPV, based on a positive Dix-Hallpike test, which resulted in the immediate performance of repositioning maneuvers during their otolaryngology visit in the MCC. A study of adult military patients evaluated within a few days after concussion found that approximately one-third of those with dizziness had evidence of BPPV that was successfully treated with repositioning maneuvers, resulting in a much more rapid return to duty than other patients with dizziness in the study.\(^{15}\) The patients in that study were all adults and were evaluated by an otolaryngologist within days of their injury, resulting in prompt diagnosis and treatment of BPPV. In contrast, the pediatric patients in our study were not evaluated by an otolaryngologist and diagnosed with and treated for BPPV until a mean of 19 weeks after their injury. This long delay may reflect a lack of awareness of BPPV among pediatric providers, since BPPV is rarely described in the pediatric literature and most nonotolaryngology providers managing concussion patients may have little to no experience with BPPV. However, once a patient’s neck is cleared, providers should perform the Dix-Hallpike as early as possible to reduce time to diagnosis and treatment.

We found 2 patients whose symptoms and test findings were consistent with SSCD as the cause of their postconcussion dizziness. Minor\(^{17}\) observed that approximately 13% of patients with SSCD experienced the onset of symptoms only after an inciting head trauma. He proposed a 2-hit model, where patients are initially predisposed to the dehiscence by preexisting thinning of the bone overlying the superior semicircular canal, which is further disrupted by the traumatic injury. Therefore, it is important for providers to be aware of SSCD as a possible cause of the onset of dizziness after concussion, even though the head injury itself may not be the direct cause of the syndrome.

Symptoms of sound-induced vertigo, hearing loss, or hyperacusis are clues to the diagnosis of SSCD or PLF, while positional vertigo is a clue to the diagnosis of BPPV. The symptoms of PVD in concussion patients that are due to temporal bone fracture or labyrinthine concussion may be more subtle and primarily consist of room-spinning vertigo that is exacerbated by head movements and oscillopsia (sensation of movement of the visual fields). Hearing loss is also often present. Vestibular testing and audiometry should be considered when these symptoms are present followed by temporal bone CT if unilateral peripheral vestibular loss is identified.

The focus of this study was on PVD in concussion patients, but 81 patients in this group did not have PVD.
It is possible that these patients had dizziness due to central postconcussive vestibulopathy, although we cannot be certain. Hoffer and colleagues 18 described 3 categories of central postconcussive vestibulopathy. The first is exertional dizziness, which is triggered only by exertion and is generally associated with normal examination and test findings. Exertional dizziness is primarily treated by hydration and a gradual reconditioning regimen. The second type is migraine-associated dizziness, which is similar to vestibular migraine and is also typically associated with normal examination and test findings. Migraine-associated dizziness is managed with treatments directed specifically at migraine, including behavioral changes and medications. The third type is spatial disorientation–related dizziness, which causes symptoms of motion sensitivity and disequilibrium that are constant but not associated with findings of PVD on examination or testing. Spatial disorientation–related dizziness is managed with vestibular rehabilitation.

We have developed an algorithm to help guide the identification and timely management of these disorders in children and adolescents with dizziness in the setting of PCS (Figure 2). Bedside maneuvers are used to diagnose and treat patients with BPPV. Screening for other otologic symptoms may prompt further objective vestibular testing and/or imaging that can help to identify the possible presence of surgically treatable conditions, such as SSCD and PLF, or conditions such as labyrinthine concussion or temporal bone fracture that may benefit from vestibular rehabilitation. Pediatric providers involved in the care of children with concussion should be routinely trained in the identification of traumatic PVD, particularly BPPV, so that the appropriate treatments are not delayed. This approach may help facilitate recovery from concussion and therefore reduce the vestibular symptoms of PCS in children and adolescents.

Limitations

This study was limited by its retrospective design. The clinical data available for us to review were limited for many patients in this study, since their initial postconcussion care occurred outside of our hospital in some cases. Similarly, follow-up was limited on many patients, since they were followed at outside institutions, so information on disease and symptom resolution was not available to us for many patients in this study.

Conclusions

Although often not frequently recognized by pediatric concussion providers, PVDs are common contributing factors for protracted symptoms of dizziness and imbalance following concussion. Often, a blow to the head may cause trauma to the vestibular organs in the inner ear rather than the brain alone, causing disorders such as BPPV, PLF, and SSCD. Many of these disorders are treatable when appropriately diagnosed. The included algorithm may help to guide the diagnosis and management of PVD in children and adolescents with prolonged dizziness and imbalance symptoms following concussion.

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Author Contributions

Jacob R. Brodsky, study conception/design, data analysis/interpretation, drafting/revising manuscript, final manuscript approval, agreement to accountability; Talia N. Shoshany, data acquisition/analysis, drafting/revising manuscript, final manuscript approval, agreement to accountability; Sophie Lipson, data interpretation, drafting/revising manuscript, final manuscript approval, agreement to accountability; Guangwei Zhou, data interpretation, drafting/revising manuscript, final manuscript approval, agreement to accountability.

Disclosures

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