Migraine with Aura or Sports-Related Concussion: Case Report, Pathophysiology, and Multidisciplinary Approach to Management

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Abstract
The evaluation and management of athletes presenting with clinical features of migraine headache with aura in the setting of sports-related head trauma is challenging. We present a case report of a 15-yr-old boy with a history of migraine with visual aura that developed acute visual disturbance and headache after a head injury during an ice hockey game. The patient underwent comprehensive assessment at a multidisciplinary concussion program, including neuro-ophthalmological examination, neuro-cognitive testing, and graded aerobic treadmill testing. Clinical history and multidisciplinary assessment was consistent with the diagnosis of coexisting sports-related concussion and migraine with brainstem aura. The authors discuss the pearls and pitfalls of managing patients who develop migraine headache with visual aura after sports-related head injury and the value of a comprehensive multidisciplinary approach to this unique patient population.

Introduction
Sports-related concussion (SRC) is a form of traumatic brain injury (TBI) that can present with a wide range of clinical manifestations. Headaches and visual disturbances are symptoms that commonly occur in the setting of acute SRC but also are features of primary headache disorders including migraine (57). Patients who present with migraine with visual aura in the setting of acute head trauma during sports can present a diagnostic challenge for treating physicians because of the overlap of clinical features between SRC, several forms of headache, and other neuro-ophthalmological conditions that accompany head trauma, including traumatic optic neuropathy (14,18,23–25,30,43,52,62). Although migraine headaches accompanied by visual aura are typically benign and do not require treatment, the evaluation of SRC patients presenting with acute visual disturbance requires a multidisciplinary approach including experts with clinical training in TBI, migraine, and neuro-ophthalmology to arrive at the correct diagnosis and initiate a management plan that limits the risk of cumulative and potentially life-threatening injury (4,60) and promotes neurological recovery (15,61).

Here, we report a young athlete with a history of migraine with visual aura who presented with acute visual disturbance and headache after a head injury sustained during a hockey game. A brief review of the epidemiology, classification, pathophysiology, clinical presentation, diagnostic considerations, and management of migraine with visual aura is presented. Recommendations for SRC patients with this rare clinical presentation also are discussed.

Case Report
The patient is a 15-yr-old boy with a medical history significant for two previous SRC and migraine headaches. The migraine headaches had occurred three to four times and were always characterized by the sudden onset of a fortified scotoma that evolved over 15 to 30 min into either right or left homonymous hemianopsia followed by severe and throbbing headache, nausea, and occasional vomiting. In the past, these migraine headaches were triggered by stress and one episode of severe pain associated with a finger dislocation but never by physical exercise. There was a family history of migraine, and his mother suffered migraines that were often

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preceded by visual auras and were associated with hemiparesis and dysarthria. Neither the patient nor his mother had undergone genetic testing for familial hemiplegic migraine.

During an ice hockey game, the patient sustained a blind-sided body check where the opponent's shoulder impacted the side of the patient's head. The patient reported immediate onset of mild headache, dizziness, blurred vision, and the premonitory sensation that he was going to have a migraine. After being immediately removed from the game, he noted the presence of an incomplete homonymous hemianopsia with the right eye affected more than the left. While on the sidelines he developed severe headache, vomiting, worsening of the homonymous hemianopsia followed by acute confusion and disorientation. The patient was transferred by ambulance to a pediatric emergency department where he underwent urgent computerized tomography (CT) of the brain and spine that demonstrated no abnormalities. Upon later questioning, the patient did not recall events during the time of the ambulance transfer and neuroimaging studies.

Approximately 6 h after the initial onset of symptoms, the patient experienced complete resolution of headaches, visual symptoms, and disorientation and was discharged from hospital after overnight observation.

The patient was referred to a pediatric concussion program and underwent comprehensive multidisciplinary assessment 4 d after the date of injury. The patient underwent initial consultation with a neurosurgeon who serves as the medical director of the pediatric concussion program as well as further evaluation and testing by a neuro-opthalmologist. At the time of consultation, the patient endorsed symptoms of fatigue and drowsiness but no other concussion-related symptoms. The patient denied any blurred vision, diplopia, or visual field deficit. Comprehensive neuro-opthalmological evaluation demonstrated intact visual acuity (20/30-3 OD, 20/25-2 OS) and color vision, normal pupillary function and normal funduscopic examination. Ocular motor examination revealed orthophoric alignment in the primary position at near and at distance and confirmed normal convergence, smooth pursuit, saccades, and vestibulo-ocular reflex (VOR) functioning. Automated visual field testing with Humphrey’s perimetry central 30-2 threshold was normal. The remainder of the physical examination including cranial nerve, motor, sensory, deep tendon reflexes, balance, gait, cervical spine, and cerebellar testing was normal.

Computerized neurocognitive testing using the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) tool was administered by a trained athletic therapist and interpreted by a licensed clinical neuropsychologist. All scores were found to fall within normal limits with no borderline scores (percentiles scores ranged from 32% to 79%). There was no available baseline test to compare postinjury results to in this patient. After neurocognitive testing, the patient underwent physician-supervised Buffalo Concussion Treadmill Testing undertaken by an exercise physiologist and athletic therapist using previously described procedures (32,37). Treadmill testing elicited no symptom-limiting threshold at a maximal blood pressure of 196/74 and maximal heart rate of 200 bpm suggestive of physiological recovery.

Taking into account all clinical data collected during the multidisciplinary assessment, the patient was diagnosed with SRC and migraine with typical and brain stem aura. Given the patient’s clinical status and results of neurocognitive and treadmill testing at the time of consultation, the patient was managed with a graduated return-to-play program resulting in successful return to full contact hockey. Before full medical clearance, the patient was advised of the elevated risk of future concussion given his lifetime history of concussion. The patient also was advised about the potential risk that future hockey activities including head injury could result in future migraines with a similar or more severe clinical presentation.

Discussion
This case demonstrates the value of multidisciplinary assessment in patients with acute visual disturbances after SRC and the challenges of managing patients who present with clinical evidence of migraine headaches after sports-related head injury.

Migraine is the most common primary headache disorder with an estimated prevalence of 15% to 20% in the general population (31). This condition occurs in all age groups with a peak incidence observed within the third and fourth decades of life. Although a strong male predominance is observed among pediatric migraine patients, the opposite is seen in adulthood where the male-to-female ratio is 1:3 (59). Migraine has a strong genetic basis with a family history of migraine frequently reported among migraine patients and rare forms, such as familial hemiplegic migraine, linked to specific gene mutations (28,58). Over the past 10 to 15 yr, the International Headache Society has provided evolving criteria that guide the diagnosis and subclassification of migraine headaches. The most common form of migraine that presents with visual disturbance is migraine with aura, which must meet specific diagnostic criteria. Other described forms of migraine that present with transient visual disturbance include retinal migraine and migraine with brainstem aura (21,22,55). Patients with visual auras that persist for more than 1 h and are associated with neuroimaging evidence of stroke are classified as migrainous infarction (26). In addition to visual disturbance, migraine with brainstem aura can present with decreased levels of consciousness (21). Similarly, alterations of consciousness and confusion can occur during attacks of familial hemiplegic migraine. In children, acute confusional migraine has been reported (52), and slowed cortical activation during acute migraine in children and adolescents can contribute to cognitive dysfunction (63,64). However, there is some debate as to whether confusional migraine in children and adolescents is truly confusional or simply a language disorder (29). Commonly reported triggers associated with migraine headaches include stress, lack of sleep, bright lights, certain foods, and dehydration. In rare cases, migraine-like headaches also can be triggered by head trauma, with a significant proportion of reported cases described in children and adolescents who manifested with headaches and a range of neurological impairments including hemiparesis, blindness, confusion, and coma (24,25).

The pathophysiology of migraine headaches and acute concussion remain controversial but may be closely related. At present, migraine with aura is thought to arise from a wave of spreading depolarization resulting in transient...
suppression of cortical activity (cortical spreading depression [CSD]) advancing 2 to 6 mm min⁻¹ (2,36). The visual aura specifically occurs as a result of CSD that originates in the striate cortex and is associated with alterations in regional cerebral blood flow (5,34,35). CSD in turn, causes the headache phase of migraine by activating trigeminal nociceptors that innervate large meningeal blood vessels, followed by activation of central trigeminovascular neurons (3,54). Similar to migraine, animal model evidence suggests that concussion is mediated by alterations in excitatory neurotransmitter release, neuronal depolarization, and cerebral blood flow (20). Indeed, neuroimaging studies have demonstrated alterations in resting cerebral blood flow and cerebrovascular responsiveness after concussion as well as those with posttraumatic migraine headaches (49,50).

Comprehensive assessment of patients presenting with visual disturbance and headache after sports-related head trauma requires a multidisciplinary approach by experts with clinical training and experience in TBI, migraine, and neuro-ophthalmology. Other conditions that can present with visual disturbance in the setting of sports-related head injury, such as traumatic cranial neuropathy, intraparenchymal hemorrhage, and vestibular dysfunction, must be ruled out before this diagnosis can be made and managed appropriately.

Because the pathophysiology of CSD in migraine is cortical in origin, the hallmark of migraine with visual aura is acute onset of binocular visual disturbance followed by headache. Visual auras can manifest as positive or negative phenomena that generally develop over minutes and persist for up to 1 h in duration. Positive visual auras typically include a fortified scotoma that originates centrally expands temporally in a hemianopic pattern and is associated with a scintillating edge, flashing lights, colors, or waves. Negative visual auras can range from mild blurred vision to a dense homonymous hemianopsia. Visual auras also can be accompanied by other auras that present as sensorimotor or speech deficits. By contrast, retinal migraine is rare. It is typically monocular and manifests as negative, not positive visual phenomena, including complete and incomplete visual loss or scotomata, altitudinal visual field defects, and blurred vision (22,23,55). Many patients including children have difficulty discriminating between visual auras that affect one or both eyes. Because the temporal visual field is larger, patients frequently focus only on the visual phenomena in that field. Likewise, unlike adults, visual auras in children are more likely bilateral, involving both visual fields (29).

Importantly, the visual phenomena that patients with migraine experience must be distinguished from those caused by traumatic optic neuropathy which also can arise in the setting of pediatric acute SRC (18). Visual deficits caused by traumatic optic neuropathy are monocular, usually fixed and can range from mild blurred vision to complete vision loss that often present at the time of injury but in rare cases can present in a delayed fashion (39,44).

The headache that accompanies migraine with aura is typically described as unilateral, pulsating, moderate, or severe in intensity and associated with photophobia, phonophobia, nausea, and vomiting. In the majority of cases, the visual aura precedes the onset of headache but in selected cases can accompany the headache and be prolonged or can occur without headache. Studies have demonstrated considerable overlap between the symptoms of migraine headaches and SRC (30,43). Although acute SRC patients rarely meet the International Classification of Headache Disorders (ICHD)-3-beta criteria for migraine headaches, symptoms, such as headache, nausea, photophobia, phonophobia, and blurred vision, are commonly endorsed by these patients. In contrast, however, acute visual field deficits or loss of vision are not symptoms of SRC and should alert the physician to other coexisting neurological conditions (14,18). After migraine headache, many patients experience a period of fatigue or drowsiness that can persist for days after the initial headache.

The physical examination of patients presenting with visual disturbances after head injury should always include comprehensive assessment of vision, ocular motor, and vestibular function as well as a complete neurological examination. Although in recent years, we have observed the development of clinical and sideline tools that provide abbreviated assessment of some aspects of visual and ocular motor function (19,48), these tools should never be used as stand-alone tools by inexperienced clinicians to provide comprehensive neuro-ophthalmological assessment of SRC patients presenting with visual disturbance. Neuro-ophthalmological examination is frequently normal in patients with migraine with visual aura. In selected patients with retinal migraine examined during an attack, loss of visual acuity is accompanied by focal or segmental constriction of retinal arterioles and retina vein collapse, as well as an ipsilateral relative afferent pupillary defect (22,23,55). Physical examination findings in patients with traumatic optic neuropathy can reveal abnormalities in visual acuity, visual fields and color vision, ipsilateral relative afferent pupillary defect, as well as disc edema on fundoscopy in the affected eye (18,62).

Additional investigations can be useful in the assessment of patients presenting with transient visual disturbance after SRC and patients with suspected migraine headaches. Automated visual field testing is usually normal in patients with migraine but in some cases can detect visual field defects if performed at the time of the visual field symptoms. Neuroimaging in children and adolescents with migraines is often normal (45). As illustrated here, CT studies are typically normal in pediatric patients with sports-related head injury and are only indicated in the emergency department setting (33,51). Magnetic resonance imaging (MRI) studies also are normal in the majority of pediatric SRC patients but should be considered in patients with abnormal CT findings, focal neurological deficits, and persistent or worrisome symptoms (16). Although studies using advanced MRI techniques, such as diffusion tensor imaging (DTI) have demonstrated white matter changes in patients with concussion (8,27,53), similar changes also have been observed in pediatric migraine patients without a history of head injury (47). In general, children and adolescents with a reliable history of migraine headaches who suffer a similar event even in the setting of head trauma and have a normal neuro-ophthalmological examination do not require neuroimaging. However, in patients with an unreliable clinical history, monocular symptoms and those with abnormalities on neuro-ophthalmological examination, MRI of the brain, and optic pathways should be considered. Because the present patient described visual symptoms that were similar to...
previous migraine attacks, had returned to his neurological baseline, underwent a normal neuro-ophthalmological examination and initial CT imaging demonstrated no abnormalities, MRI was not considered in this child.

The management of pediatric migraine headaches continues to evolve. All pediatric migraine patients should be advised to avoid identified migraine triggers as well as maintain regular sleep, exercise, and hydration habits (41). Children and adolescents with rare migraine headaches are usually managed conservatively or with over-the-counter medications, such as acetaminophen or ibuprofen (10,40). Selected triptans also are approved for use in children as abortive medications. In patients with more frequent or severe headaches, prophylactic pharmacological management is considered using medications such as beta-blockers, tricyclic antidepressants, and anticonvulsants (9,42).

The evaluation and management of pediatric patients presenting with migraine headaches in the setting of a sports-related head injury is challenging. At present, there is no gold standard diagnostic test for concussion and no available sideline concussion instruments that can reliably distinguish between SRC and migraine headaches. Some authors suggest that neuropsychological testing can be used as a supplemental tool to aid in the management of SRC patients (46); however, there are limited evidence-based guidelines to direct the use of these tools in children and adolescents (12,13). In addition, studies have suggested that graded aerobic treadmill testing also can serve as a useful tool to assess physiological recovery after SRC (11,15,38). Although the clinical history provided by the patient was most consistent with a migraine headache that was similar to previous attacks, the immediate onset of headache at the time of injury, coupled with the limitations of currently available diagnostic tools, makes it impossible to reliably rule out a SRC in this patient. While the visual aura and accompanying headache are most fitting with a diagnosis of migraine with visual aura, the prolonged period of visual disturbance, confusion, and disorientation make migraine with typical and brainstem aura a more fitting diagnosis. Given the risks of cumulative and catastrophic injury associated with premature return to play after SRC including second impact syndrome (4,60), we recommend that all patients, especially children and adolescents, who develop clinical evidence of migraine headaches with visual aura in the setting of sports-related head injury be managed as concussion patients and undergo comprehensive assessment by physicians with clinical training in TBI, migraine headaches, and neuro-ophthalmology. Our institution, we often consider the use of both neuropsychological and graded aerobic treadmill testing to confirm neurocognitive and physiological recovery in pediatric SRC patients especially in those with multiple concussions and atypical clinical presentations (7,17).

Consequently, in the case presented here, only after documentation of a normal neuro-ophthalmological examination, normal computerized neurocognitive testing, and the absence of a symptom-limiting threshold on graded aerobic treadmill testing, was the patient medically cleared to initiate a physician-supervised graduated return-to-play program that ultimately resulted in successful return to full contact hockey. Because of the patient’s history of two previous concussions and migraine headaches, the athlete was advised about the cumulative risks of future concussions and the potential for future migraine headaches that could recur during hockey activities.

Although not observed in the patient described here, some SRC patients will present with subjective reports of visual disturbance that do not resolve after a period of physical and cognitive rest. In SRC patients who demonstrate objective evidence of vestibular dysfunction, there is some preliminary evidence to suggest that targeted vestibular physiotherapy can enhance symptomatic recovery (1,56). While vision therapy has received increased attention as a potential therapeutic option in TBI patients with documented abnormalities of vergence (6), there are no prospective controlled studies that have examined the clinical benefit of this intervention among SRC patients.

In conclusion, the evaluation and management of athletes presenting with visual disturbance and headache after head injury is challenging. Given the limitations of currently available diagnostic tools in concussion, we recommend that patients who present with clinical evidence of migraine headache with aura after sports-related head injury be managed as concussion patients until proven otherwise and undergo multidisciplinary assessment by physicians with clinical training in TBI, migraine, and neuro-ophthalmology.

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