Traumatic Optic Neuropathy: A Potentially Unrecognized Diagnosis after Sports-Related Concussion

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Abstract

Traumatic optic neuropathy is a rare cause of visual disturbance after head injury that can be difficult to distinguish from coexisting vestibulo-ocular dysfunction because of the overlap in presenting symptoms in patients with these conditions. We present a case report of a 13-year-old girl who sustained a head injury during a ringette game leading to blurred vision and diplopia persisting 5 months after injury. Clinical history and physical examination findings were consistent with a traumatic optic neuropathy, convergence insufficiency, and postconcussion syndrome. Neuroimaging was normal. The patient was managed using a multidisciplinary approach. At 6 months of follow-up, neuro-ophthalmological examination demonstrated evidence of permanent partial optic nerve injury, and formal neuropsychological testing fell primarily within normal limits. The patient was advised to retire from collision sports. The authors discuss the value of a comprehensive multidisciplinary approach to the evaluation and management of concussion patients presenting with persistent visual symptoms.

Introduction

Sports-related concussion (SRC) is a form of traumatic brain injury (TBI) that can lead to a wide spectrum of clinical presentations. Among the most debilitating symptoms that can occur after SRC are those that affect vision including blurred vision, diplopia, gaze instability, and motion sensitivity. In 24% to 90% of adult TBI (2,6) and 29% to 62% of pediatric SRC patients (8), visual disturbances are associated with dysfunction of the vestibular and oculomotor neurological subsystems. In rare cases, however, visual symptoms can occur as a result of direct injury to the visual processing pathways including the optic nerves (11,13,19). Distinguishing between these important causes of visual disturbance in TBI and SRC patients can be challenging but has important implications for prognosis and management.

Here we report a young athlete who presented with visual disturbance after SRC and clinical evidence of traumatic optic neuropathy and vestibulo-ocular dysfunction. A brief review of the anatomy, pathophysiology, clinical evaluation, and neuroimaging assessment of traumatic optic neuropathy is presented. The value of a multidisciplinary approach to complex concussion patients also is discussed.

Case Report

A 13-year-old girl with no previous history of concussion or orbital trauma sustained a head injury during a ringette game, a Canadian sport similar to hockey wherein players use straight sticks instead to propel a rubber ring into a hockey net. The patient was wearing a helmet and reported colliding with another player and then hitting the back of her head on the ice resulting in a brief period of anterograde amnesia. Her initial symptoms included headache, dizziness, fogginess, and difficulty focusing. Over the next four weeks, the patient began to experience blurred vision and diplopia. She also reported decreased peripheral vision and "squiggly lines" in the left eye. Magnetic resonance imaging (MRI) was normal but visualization of the optic pathways was limited because of the artifact from the patient's braces. The patient was referred to our pediatric multidisciplinary concussion

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program and underwent formal assessment by a neurosurgeon and neuro-ophthalmologist 2 months after the injury. At the time of clinical assessment, the patient continued to report intermittent headaches, difficulty reading, intermittent diplopia and blurred vision, and fixed decreased peripheral vision in the left eye. She also complained of fatigue, photophobia, excessive sleep, and feeling more emotional. Physical examination demonstrated evidence of a left relative afferent pupil defect, slight dyschromatopsia in both eyes (recognizing seven out of eight Ishihara plates), and left optic nerve pallor on undilated fundoscopy. Visual acuities with glasses were 20/20–1 (right eye) and 20/20 (left eye). Near point of convergence was 20 cm. Extraocular movement testing with cross-cover test showed no strabismus at distance and four-prism-diopter exotropia at near (*i.e.*, convergence insufficiency). Smooth pursuit testing was normal. Testing of horizontal saccades and vertical saccades as well as vestibulo-ocular reflex testing revealed normal movements but evoked pronounced vestibular symptoms. Automated visual field testing revealed restrictive pattern of reduced visual sensitivity, more so superiorly in the left eye. Optic coherence tomography (OCT) demonstrated reduced nerve fiber layer thickness inferiorly in the left eye and normal thickness of the ganglion cell layer complex. Visual evoked potentials were normal in both eyes. The remainder of the neurological examination, including cranial nerves, motor, sensory, deep tendon reflexes, balance, gait, cervical spine, and cerebellar testing, was normal. Taken together, the clinical history and physical examination findings were consistent with the left traumatic optic neuropathy, convergence insufficiency, and postconcussion syndrome (PCS).

To assess the structural integrity of the optic pathways, the patient had her braces removed and underwent repeat MRI of the brain and optic nerves, which were normal (Figure). Diffusion tensor imaging (DTI) of the optic nerves was acquired, but quantitative analysis and tractography could not be accomplished because of the small caliber of the optic nerves and artifact from the eye movements and nearby sinuses.



Figure: MRI obtained in this adolescent with a left traumatic optic neuropathy after SRC. No evidence of traumatic injury to the brain or optic nerves was detected. An axial T2-weighted image is shown here.

After neuroimaging, the patient underwent Buffalo Concussion Treadmill Testing that elicited a symptom-limiting threshold at 10 min at a maximal heart rate of 131 bpm, consistent with a diagnosis of physiologic postconcussion disorder (PCD). The patient was managed using a multidisciplinary approach whereby the traumatic optic neuropathy was managed conservatively, the patient's convergence insufficiency was addressed with targeted vision therapy, and the patient's physiologic PCD was treated with submaximal aerobic exercise therapy. At 2 months of follow-up, the patient reported no blurred vision and diplopia. The patient's left eye visual field defect was also reportedly resolved. On examination, the patient's near point of convergence was 3 cm. Her exotropia at near (*i.e.*, convergence insufficiency) was reduced to a two-prism diopter, and she denied any difficulty or blurred/double vision with reading. Visual acuity was 16/20 in both eyes. Repeat visual field testing demonstrated slightly reduced but improved sensitivity superotemporally in the left eye. The patient recognized eight out eight plates with both eyes when she was tested with Ishihara color vision test. There was still a slight relative afferent pupillary defect and slightly pale optic nerve head in the left eye. Taken together, these clinical findings indicated that her convergence insufficiency was resolved and her traumatic optic neuropathy improved significantly. However, repeat OCT showed that the reduced neurofibrillary layer inferiorly in the left eye was unchanged compared with her first OCT, which suggests that a permanent scar was left in the left optic nerve even after her clinical symptoms were resolved. The patient was transitioned from the submaximal exercise program to a sport-specific return-to-play program. At 6 months of follow-up, the patient was asymptomatic at rest and during intensive exercise but continued to endorse intermittent difficulty with visual memory. To determine whether these subjective reports were associated with objective cognitive deficits related to concussion, the patient underwent formal neuropsychological testing. Performance on objective measures of effort was intact, suggesting that the overall neuropsychological profile provided an accurate reflection of the patient's neurocognitive functioning. More comprehensive neuropsychological testing revealed intact intellectual functioning, attention, processing speed, mental flexibility, auditory memory, and higher order neurocognitive functioning. Despite her history of visual disturbances, visual tracking and speed fell well within or above normal limits. Reading comprehension and immediate recall and recognition of visual information (*i.e.*, simple visual designs and pictures of social settings) fell within expected (*i.e.*, normal) limits. Performance on a single test of incidental visual memory revealed poor immediate and delayed recall of information, whereas recognition was intact. The overall neuropsychological profile was not suggestive of persistent neurocognitive deficits secondary to concussion. In the absence of evidence-based guidelines directing return-to-play decision-making in cases of traumatic optic neuropathy, the neurosurgeon, neuro-ophthalmologist, and neuropsychologist medically cleared the patient to return to competitive running and swimming but advised the patient to avoid future contact and collision sports.

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Discussion

The present case demonstrates the importance of comprehensive neurological assessment in patients with focal or persistent visual disturbances after SRC and the value of a multidisciplinary approach to the management of complex SRC patients.

Anatomically, each optic nerve is composed of four parts including the intraocular, intraorbital, intracanalicular, and intracranial segments. The intraocular segment including the optic head derives its main blood supply from the posterior ciliary arteries (15). The intraorbital segment is supplied by the ciliary branches and central retinal artery arising from the ophthalmic artery, and the intracranial segment is supplied by the superior hypophyseal artery (25). Vascular supply to the intracanalicular segment is less robust and consistent among cadaveric studies (5,25), likely contributing to the increased vulnerability of this portion to different forms of pathology.

Traumatic optic neuropathies have been reported in 0.5% to 5.2% of patients after TBI (7,23) with an estimated annual incidence of one case per million among children (11). Although sports-related injuries account for approximately 20% of traumatic optic neuropathies in children (11,13), the authors are not aware of any previously reported cases of coexisting traumatic optic neuropathy and SRC. Traumatic optic neuropathy can be classified on the basis of anatomical location or the mechanism of injury. Mechanistically, traumatic optic neuropathies are broadly classified as direct and indirect injuries. Direct injuries most typically result in laceration, contusion, or crush injury to the optic nerve from direct external penetrating trauma, nerve avulsion, or injury from fractured bony fragments of the orbit and skull base. Indirect injuries, on the other hand, occur as a result of the transmission of abnormal biomechanical forces to the optic nerve from other areas of the skull and globe preferentially affecting the intracanalicular segment that is fixed within the confinements of the optic canal making it vulnerable to traction, edema, compression, ischemia, and neuroinflammatory injury (16). Traumatic optic neuropathies can present with varying degrees of visual impairment ranging from mild deficits in color vision to complete visual loss and no light perception. Although most deficits manifest at the time of injury, a minority of patients may experience acute or stepwise deterioration in vision occurring hours or days later (17,19). In the setting of SRC, symptoms of traumatic optic neuropathy may be difficult to distinguish from those arising from vestibulo-ocular dysfunction, which can be present in up to 29% of pediatric acute SRC and 62% of PCS patients (8). In both cases, patients may report blurred or double vision, dizziness, and difficulty focusing or concentrating. Concussion symptom inventories that include subjective rating of nonspecific symptoms such as "visual disturbance" or "visual problems" are insufficient stand-alone screening tools for both traumatic optic neuropathy and vestibulo-ocular dysfunction. Children in particular may not have the vocabulary to express subtle changes in visual perception and must be asked directly about subtle visual symptoms, whether they are present in one or both eyes and what factors exacerbate these symptoms. For instance, vestibulo-ocular dysfunction is associated with binocular blurred vision or diplopia that is usually not present at rest and is only elicited by quick eye or head movements or prolonged periods of focusing or reading. In contrast, blurred vision associated with traumatic optic neuropathy is monocular, fixed, and can be associated with a visual field defect, color vision deficits, or abnormal visual obscurations. In the case presented here, symptoms of both vestibulo-ocular dysfunction and traumatic optic neuropathy were present.

The physical examination of SRC patients with visual disturbances must always follow a structured approach that includes interrogation of the visual, oculomotor, and vestibular systems. Whenever an optic neuropathy of any etiology is suspected, all patients should undergo urgent evaluation by an experienced neuro-ophthalmologist who has the training and resources to provide a comprehensive assessment of visual functioning. Physical examination findings that may be present in traumatic optic neuropathy include impairments in visual acuity, relative afferent papillary defect, visual field defect, or impairments in color vision. Fundoscopy also should be competed with clinical findings often dictated by the mechanism and extent of injury. Direct injuries may be associated with hemorrhage or contusion of the optic disc, whereas indirect injuries may be associated with mild pallor of the optic disc or papilledema depending on the timing of injury. Because optic nerve pallor does not develop until 4 to 6 wk after injury, fundoscopy may appear normal in the setting of acute injury (16). Similar to our patient, SRC patients with traumatic optic neuropathy may exhibit evidence of coexisting vestibulo-ocular dysfunction. Physical examination findings in these patients may include evidence of convergence insufficiency (a near point of convergence of less than 6 to 10 cm) as well as abnormalities in smooth pursuits, horizontal and vertical saccades, and the vestibulo-ocular reflex (8,16). Although these objective tests may be normal on inspection, testing alone may evoke vestibular symptoms, which not only can point to more subtle vestibulo-ocular dysfunction but also can be elicited in healthy subjects (21). Standardized balance tests such as the Balance Error Scoring System (BESS) can be helpful also to detect postural instability suggestive of vestibular dysfunction (14). Over the past few years, concussion tools such as the King-Devick test (12) and vestibulo-oculomotor screen tool (21) have been developed to screen for vestibular and oculomotor impairments in the setting of SRC. Although it may be helpful to incorporate these tools into the initial sideline screening assessment of patients with acute SRC, these tools are not designed to diagnose subtle abnormalities of the visual processing system such as traumatic optic neuropathy. This case illustrates the essential need of comprehensive assessment performed by physicians with training in TBI and neuro-ophthalmology in patients presenting with persistent visual disturbance after SRC. Additional tests that neuro-ophthalmologists can use to help clarify the extent of traumatic optic neuropathy include formal visual field testing to reliably evaluate peripheral vision, optical coherence tomography to look for a reduction in the nerve and ganglion cell layers (20) as well as visual evoked potential tests to examine the neurophysiologic integrity of the visual pathways (1). Once the diagnosis of traumatic optic neuropathy is confirmed by a neuro-ophthalmologist, further neuroimaging work-up and treatment can be considered (for summary, see the Table; for additional information on distinguishing features of PCS subtypes, see the study by Ellis et al. (9).

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Table.

Summary of clinical presentation, physical examination findings, diagnostic tests, prognosis, management, and return-to-play considerations in patients presenting with vestibulo-ocular dysfunction and traumatic optic neuropathy after SRC.

	Vestibulo-ocular Dysfunction	Traumatic Optic Neuropathy
Clinical complaints	• Visual disturbance	• Visual disturbance
	• Visual problems	• Visual problems
	• Blurred vision	• Blurred vision
	• Difficulty focusing/concentrating	• Difficulty focusing/concentrating
	• Diplopia	• Complete monocular vision loss (severe cases)
	• Dizziness	
Distinguishing features	• Blurred vision and/or diplopia often intermittent and associated with quick head movements or prolonged reading/focusing	• Blurred vision is fixed and monocular
	• Blurred vision and/or diplopia are binocular	• Can be associated with visual field defect, changes in color vision, and abnormal visual perceptions
Common physical examination findings	• Visual acuity (normal)	• Visual acuity (reduced in affected eye)
	• Color vision (normal)	• Color vision (reduced in affected eye)
	• Fundoscopy (normal)	• Fundoscopy (normal or abnormal depending on time and mechanism of injury)
	• Pupillary function (normal)	• Pupillary function (relative afferent pupillary defect in affected eye)
	• Visual fields (normal)	• Visual fields (abnormal in affected eye)
	• Smooth pursuits (abnormal)	• Smooth pursuits (normal)
	• Horizontal and vertical saccades (abnormal)	• Horizontal and vertical saccades (normal)
	• Near point of convergence (abnormal, >6 to 10 cm in children)	• Near point of convergence (normal, <6 to 10 cm in children)
	• Vestibulo-ocular reflex (abnormal)	• Vestibulo-ocular reflex (normal)
	• Balance testing (abnormal)	• Balance testing (normal)
Diagnostic tests	• Graded aerobic treadmill testing (to diagnose other coexisting PCS subtypes)	• Automated visual field testing
	• Formal neuropsychological testing (to confirm neurocognitive recovery)	• Optical coherence tomography
		• Visual evoked potentials
		• CT/MRI (depending on the mechanism and severity of injury)
Prognosis	• Good	• Dependent on the extent of visual impairment at the time of initial assessment
	• Majority of cases resolve with conservative management	
Management considerations	• Conservative management	• Conservative management
	• Targeted vestibular and vision physiotherapy	• Consider low- and high-dose corticosteroids, optic nerve decompression via intracranial and extracranial surgical approaches
Return-to-play considerations	• No contraindication to safe return-to-play	• Consider sport retirement because of risk of recurrent injury and associated disability

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Neuroimaging plays a limited role in the evaluation of patients with SRC but should be considered in SRC patients with focal, prolonged, or worsening visual disturbance and especially in patients with suspected traumatic optic neuropathy (10). Computerized tomography (CT) is useful for the evaluation of patients with suspected fractures of the orbit or skull base associated with direct traumatic optic neuropathy, but it must be used judiciously in children because of the potential long-term risks of radiation exposure. With its enhanced contrast resolution, conventional MRI may detect evidence of edema, hemorrhage, swelling, or atrophy, but in many patients, including the present case, these studies are normal (4). For this reason, recent studies have aimed to apply even more sophisticated neuroimaging techniques such as DTI to these patients, which is capable of examining the microstructural integrity of the white matter tracts within the optic nerves (4,18). Unfortunately as illustrated here, the small caliber of the optic nerve and artifact from eye movements and the nearby air-filled sinuses make routine application of these studies challenging.

The prognosis of visual disturbances after pediatric SRC is highly dependent on the etiology. Previous work indicates that pediatric acute SRC patients with subjective and objective evidence of vestibulo-ocular dysfunction take significantly longer to recover compared with those without this clinical feature (22 d vs 41 d) and are four times more likely to develop PCS even after controlling for other predictors of PCS (8). Nonetheless, the vast majority of pediatric SRC patients with vestibulo-ocular dysfunction achieve a complete neurological recovery and are successfully returned to sport activities. On the other hand, the prognosis of traumatic optic neuropathy is highly dependent on the extent of visual impairment at the time of initial assessment. Improvement in visual acuity has been observed in 33% to 44% of patients in published pediatric studies (11,13,19); however, children and adolescents with no light perception at the time of initial assessment rarely experience significant visual improvement (13).

The management of traumatic optic neuropathy remains controversial. There have been no completed randomized controlled trials conducted in this population and no convincing evidence that any therapeutic intervention improves patient outcomes (16,17). Management options include low- and high-dose corticosteroids, optic nerve decompression via intracranial and extracranial surgical approaches, and observation (17,24). Emerging evidence suggests that TBI and SRC patients with vestibulo-ocular dysfunction may benefit from rehabilitation programs that target specific objective oculomotor and vestibular deficits. One randomized controlled trial demonstrated enhanced recovery in SRC patients treated with cervicovestibular therapy (22); however, future studies are needed to evaluate the effects of targeted vestibular therapy on the objective findings mediating symptoms in patients with isolated vestibulo-ocular dysfunction, such as the patient presented here. Accumulating evidence also suggests that some patients with PCS have symptoms attributable to an ongoing metabolic brain injury that can be diagnosed with graded aerobic treadmill testing. Specifically, patients who achieve a symptom-limiting threshold on graded aerobic exercise testing can be diagnosed with overlapping physiologic PCD (9). Published studies demonstrate that patients with physiologic PCD, including adolescents, demonstrate a high rate of symptom improvement after tailored submaximal exercise prescription (3). Accordingly, the patient presented here was managed using a multidisciplinary approach to address the pathophysiologic mechanisms responsible for individual persistent symptoms. This management plan included conservative management of the traumatic optic neuropathy, targeted vision therapy for convergence insufficiency, and submaximal exercise prescription for the symptoms of physiologic PCD. At 2 months of follow-up, symptoms of traumatic optic neuropathy and vestibulo-ocular dysfunction were resolved. There was no objective evidence of convergence insufficiency or vestibulo-ocular dysfunction. However, a slight relative afferent papillary defect, slight pallor of the optic nerve head, and reduced neurofibrillary layer inferiorly in the left eye on OCT pointed to permanent residual optic nerve injury. At 6 months, the patient reported complete resolution of all concussion symptoms at rest and during exercise but endorsed intermittent difficulty with visual memory. Performance on objective measures of neurocognitive functioning was not suggestive of persistent cognitive deficits secondary to concussion. In the absence of evidence-based guidelines directing return-to-play decision-making in cases of traumatic optic neuropathy, the patient was medically cleared for return to competitive running and swimming but advised to avoid future contact and collision sports.

In conclusion, visual disturbances after SRC can arise from injury to the visual processing pathways or functional impairments of the vestibulo-ocular system. Distinguishing between these important causes of visual disturbance in TBI and SRC patients requires comprehensive assessment by physicians with clinical training and experience in TBI and neuro-ophthalmology. Multidisciplinary management of pediatric SRC and PCS should focus on medical and rehabilitative strategies that target the pathophysiologic mechanisms and objective clinical examination findings responsible for individual concussion symptoms.

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