



<https://doi.org/10.1016/j.jemermed.2021.11.014>

Clinical Communications: Pediatric

Total Isolated Monocular Vision Loss in a Patient Who Suffered Closed Head Injury

Derek Chi Fung Tam,* and Matthew P. Murray†

*University of California (UC) San Diego Department of Pediatrics, Rady Children's Hospital of San Diego, San Diego, California, and †UC San Diego Department of Emergency Medicine, Department of Pediatric Emergency Medicine, Rady Children's Hospital of San Diego, San Diego, California

Corresponding author: Derek Tam, Department of Pediatric Emergency Medicine, Rady Children's Hospital of San Diego, 3020 Children's Way, MC 5075, San Diego, CA 92123

Abstract—Background: Head injuries are an important cause of morbidity and mortality in children and young adults. There are multiple sight-threatening complications of head injury, even in closed head injury without visible violation of the globe or orbits. One such entity is traumatic optic neuropathy. **Case Report:** Herein we describe a case of traumatic optic neuropathy in an otherwise healthy teenage patient who suffered total monocular vision loss after a fall and without any other injuries on examination. Unfortunately, the prognosis for this condition is relatively poor in terms of visual recovery. Though much research has been conducted attempting to treat this condition, to date there have been no studies showing a clear benefit of medical or surgical intervention. *Why Should an Emergency Physician Be Aware of This?* Although there is no proven treatment for traumatic optic neuropathy, emergency physicians may encounter this in their practice while caring for both pediatric and adult patients presenting with head injury. Having more background knowledge on this condition will enhance emergency physicians' ability to consult with subspecialist providers as well as to educate patients and their families on their condition and prognosis. © 2021 Published by Elsevier Inc.

Keywords—trauma; head trauma; closed head injury; vision loss; blindness; traumatic optic neuropathy; pediatric

Introduction

Head trauma is a major cause of morbidity and mortality in children, with thousands of emergency department (ED) encounters occurring each year. Ocular injuries and sequelae are similarly common, with over 58,000 hospital admissions over a 6-year period in one study (1). There are multiple sight-threatening conditions that can occur as a result of head trauma, even if the globe itself is intact. We present a case of a rare but severe consequence of closed head injury in a pediatric patient.

Case Report

The patient is a 13-year-old boy with no significant past medical history presenting with head trauma from a fall while riding his bicycle. The patient was wearing a helmet and fell forward from his bike, hitting the ground with his right shoulder and right side of his head. The fall was not witnessed, but the patient was unaware of any loss of consciousness and reported full recall of the event. A bystander who arrived after the fall summoned Emergency Medical Services, who brought the patient to another ED prior to transfer to our institution. Upon arrival to our facility, he was protecting his airway, breathing spontaneously, and was hemodynamically stable. His Glasgow

Abbreviations: TON, Traumatic Optic Neuropathy.

RECEIVED: 24 August 2021; FINAL SUBMISSION RECEIVED: 1 November 2021;
 ACCEPTED: 27 November 2021

Coma Scale score remained 15 throughout his stay in the ED. His vital signs on arrival were: temperature 36.5°C, heart rate 75 beats/min, blood pressure 98/44 mm Hg, and respiratory rate 22 breaths/min.

His physical examination was notable for superficial abrasions to the right side of the face. His cranial nerve examination was significant for an unreactive right pupil fixed at 5 mm; the left pupil was appropriately reactive to light. The patient reported total vision loss in his right eye without pain, saying that he could “only see black” out of his right eye. Visual acuity in the left eye was intact. There were no other focal neurologic findings on examination. In addition to mild pain at the sites of the abrasions, the patient reported mild nausea and had one episode of emesis shortly after arrival.

A non-contrast computed tomography study of the head performed by the referring institution was negative for any fracture, intracranial bleeding, or other abnormality. On discussion with the trauma and neurosurgical consultants, we obtained computed tomography angiography of the head and neck to assess for arterial dissection as a cause of his vision loss. These studies were negative for vascular injuries or other serious abnormalities. Both globes were intact on imaging and there was a right periorbital and frontal scalp hematoma.

On discussion with the consultant ophthalmologist, the most likely diagnosis was determined to be traumatic optic neuropathy (TON). The team also considered the diagnosis of retinal detachment, however, this was felt to be unlikely given the history, as traumatic retinal detachment is often seen with more severe mechanisms of injury than the fall reported in this patient. Ophthalmology examined the patient in the ED and confirmed the diagnosis of TON, given that the fundoscopic and ophthalmologic examinations were normal aside from absent light perception in the right eye, as well as the pupillary defect, which is commonly seen in TON (2).

The patient was admitted in stable condition to the pediatric intensive care unit, where his neurological condition and cardiorespiratory status were monitored closely. He had improvement but not complete resolution in his headache and nausea; these symptoms were attributed to a mild concussion. On the second hospital day, the patient was discharged home. No improvement in the patient's vision occurred by the second hospital day.

The consulting ophthalmologist followed up with the patient in the outpatient clinic approximately 3.5 weeks after discharge. At that visit the patient was again found to have no light perception in the right eye, with normal visual acuity in the left. Intraocular pressures were normal bilaterally. The patient and his family were counseled at this visit and instructed to follow up again in 6 months.

Discussion

Traumatic optic neuropathy is a relatively rare complication of head trauma, but its effects on eyesight can be devastating. Better described in the ophthalmologic literature, TON has been a subject of study for decades, as an effective treatment for the condition has remained elusive (3). The pathophysiology of this condition is similarly disputed, but loss of eyesight is thought to occur due to disruption of blood vessels supplying the nerve by shear forces, leading to localized hyperemia (2). Due to the fixed volume of the tracts through which the optic nerve courses, the increased pressure caused by fluid accumulation leads to ischemia and cell death, similar to the pathophysiology of compartment syndrome (2). Another proposed mechanism involves direct shear forces leading to nerve damage, similar to that of diffuse axonal injury (2).

The reported prevalence of TON varies, depending upon the study and population. One of the largest studies of traumatic ocular and optic injuries (a review of data from the National Trauma Data Bank from 2008 through 2014) reported “visual pathway” injuries in children aged younger than 21 years in 1.3% of non-accidental injuries and 2.7% of injuries related to assault (1). However, this study does not differentiate the different subtypes of visual pathway injury, so other injuries such as optic nerve avulsion, transection, hematoma, or others are likely included in this category. Another series reviewed by Steinsapir and Goldberg reported an incidence of TON in 0.5–5% of cases of closed head injury (4).

The differential diagnosis for TON can be approached anatomically. Starting with the globe itself, vision loss or impairment in the setting of trauma should always prompt investigation for an open globe injury. A large hyphema or vitreous hemorrhage can also lead to vision loss (5). Retinal detachment or other injury can be assessed using ocular point-of-care ultrasound, provided that an open globe can be reasonably excluded. Application of the ultrasound probe to the ruptured globe can cause or worsen extrusion of intraocular contents. Direct transection or impingement on the optic nerve can also be seen in trauma, such as in fractures of the posterior orbit or basilar aspects of the skull. The blood supply to the optic nerve can be compromised in cases of carotid dissection (5). Finally, damage to the intracerebral optic pathways, including the optic chiasm and the brain regions responsible for visual perception, can lead to vision loss, though damage to these areas is less likely to be isolated to those areas alone in significant head trauma.

One of the earliest case series on TON (published in 1982) documented seven cases in children and adults who

suffered blunt frontal head trauma (6). The average patient age in that series was 18 years, and most of those patients (6 of 7) received high-dose steroids. Because some of those patients (3 of the 6) had some recovery of vision after steroids, steroids became a common treatment for TON. Mechanistically, because TON is thought to involve activation of an inflammatory cascade initiated by mechanical trauma, steroids were used as primary treatment for many years (2). Other studies from the neurosurgical world showing a benefit of steroids in spinal cord injury were also used to support the rationale of using steroids in TON. More recent retrospective and prospective studies, however, have failed to show a benefit of steroids in recovery of vision (7,8). A 2016 study by Sosin et al. reviewed 109 patients with TON and found a wide variety of treatment regimens, with no benefit of steroids over observation alone (7). In that series, 47.6% of patients had some degree of visual improvement. Animal models of optic nerve injury further cloud the picture, as some have shown an increase in axonal loss after steroid administration (9).

Another method used in the treatment of TON is surgical decompression of the optic nerve, with the proposed mechanism of action being that relief of pressure on an optic nerve swollen from trauma can allow return of blood flow to the nerve, thereby preserving or restoring vision. Animal models of TON have demonstrated axonal shearing as well as hyperemia and vasospasm. Given the narrow compartment through which the optic nerve travels, it was thought that blood flow could be restored through surgical decompression of the nerve (10). Although the practice is still used by some practitioners and institutions, there has not been convincing prospective evidence demonstrating the benefit of surgery in terms of visual recovery (7,11,12). Case reports supporting the use of surgery, like those supporting the use of steroids, involve quite small and heterogeneous groups of patients: some received steroids alone, some surgery alone, and still more, both modalities (6).

Perhaps the most convincing prospective comparison of TON treatment outcomes is the International Optic Nerve Trauma Study reported by Levin et al., in which 133 patients with TON were prospectively followed for at least 1 month after injury (12). In this study, the authors found no significant difference between patients treated with steroids, surgery, or simply observed without intervention. Specifically, 32% of surgically managed patients had improvement in visual acuity, whereas 52% of those treated with steroids and 57% of those managed with observation alone had improvement in acuity. There were no statistically significant differences in outcomes between any of the groups.

More recently, experimental treatments including administration of erythropoietin, hyperbaric oxygen, or

minocycline have proven successful in some case reports (13). However, at least for erythropoietin, a recent multicenter clinical trial failed to demonstrate benefit of erythropoietin over steroids or observation alone (14). Hyperbaric oxygen therapy has yet to be formally studied for this purpose, but its proposed utility in treating TON is thought to result from the anti-apoptotic, anti-inflammatory, and pro-neurogenic effects that have been demonstrated in some cases of traumatic brain injury (13,15).

Considering the lack of convincing evidence for a known effective intervention to potentially reverse this patient's vision loss, no steroids or other interventions were given either during the inpatient admission nor during outpatient follow-up.

Why Should an Emergency Physician Be Aware of This?

The total loss of vision in one or both eyes after trauma is alarming to physicians as well as patients and their families. From the emergency physician's standpoint, it remains critical to rule out possibly reversible or treatable causes of vision loss, such as stroke, arterial dissection, or direct injury to the optic nerve or globe. However, emergency physicians should be aware of the possibility that their patient with new onset of vision loss may be suffering from TON if other causes have been sufficiently ruled out. Consultation with a neurosurgeon and ophthalmologist is important in cases of traumatic vision loss. With the many ways vision loss can occur after trauma, consulting physicians may recommend a variety of treatments or observation alone. If the treating team diagnoses TON, it is unlikely that the patient will benefit from any pharmacological or surgical interventions, at least based upon the current understanding of the condition. It will be useful for patients and families to understand that there is no known effective treatment, and that experimental treatments can involve a significant amount of risk (e.g., the side effects of high-dose steroids). Patients and their families should be counseled that the prognosis is guarded, with half of patients being expected to have no improvement in visual perception, and with the other half experiencing some degree of, but not necessarily complete, improvement.

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