CLINICAL CASE REPORT

Acute direct traumatic optic neuropathy treated with steroids, minocycline and hyperbaric oxygen: a case report

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ABSTRACT

We describe the emergency management of a man who experienced acute vision loss diagnosed as direct traumatic optic neuropathy (TON) in his right eye (no light perception) after falling from a height. TON is caused by a high-impact mechanism of injury. Clinical findings include acute vision loss, which is typically immediate, afferent pupillary defect, decreased color vision, and visual field defects. Treatment is controversial because of the lack of strong evidence supporting intervention over observation. In this case report, our treatment strategy comprised immediate hyperbaric oxygen (HBO_2) and daily high doses of a steroid. On the second day, minocycline was added to the treatment regimen for its neuroprotective effects. The patient was discharged after receiving six HBO_2 treatments and six days of intravenous solumedrol transitioned to oral prednisone. After the third HBO_2 treatment, his vision improved to 20/100; after the fourth treatment, it was 20/40 and plateaued. At the time of discharge, it was 20/40. At two-month follow-up, his corrected visual acuity was 20/60+2 in the affected eye. Immediate HBO_2 for ischemic and mechanical injury to the optic nerve following trauma is a therapeutic option.

INTRODUCTION

Traumatic optic neuropathy (TON) is a potentially blinding condition that results from a high-energy impact. The most frequent mechanism is motor vehicle collision, particularly crashes involving motorcycle drivers [1]. In a recent large retrospective study, the prevalence of vision loss after facial trauma was 0.3% [2]. TON is typically described as indirect (caused by shearing force) or direct (caused by compression by a bone fragment or foreign body). The clinical finding associated with TON is vision loss, which is usually immediate after injury in direct TON and can progress in delayed fashion in indirect TON. On exam, afferent pupillary defect (APD), decreased color vision, and visual field defects that localize to the optic nerve, commonly with altitudinal defects, will be present.

Half of patients with indirect TON recover some amount of vision spontaneously [3, 4]. Optic nerve exam with fundoscopy is typically normal at presentation because most injuries occur posterior to the globe, at the optic canal. Optic atrophy or pallor often emerges in the months following injury.

Treatment for TON is controversial due to a lack of strong evidence to support intervention over observation [5]. Although there is anecdotal evidence of improvement of direct and indirect TON cases with hyperbaric oxygen (HBO_2) therapy, few case reports about this treatment modality have been published [6, 7]. We report a positive response to emergency HBO_2 combined with medical treatment in a patient with direct TON.

CASE REPORT

A 27-year-old man with no significant past medical or ocular history was transported to a trauma center after falling 20 feet from a ladder, with resultant loss of consciousness. Unarousable for 10 minutes, he regained consciousness with amnesia for the event and right-sided headache. In addition to orthopedic injuries, primary and secondary surveys revealed a non-displaced right orbital floor fracture and an orbital roof fracture that extended to the right optic canal (Figure 1). A skull base fracture was also noted, encompassing the middle cranial fossa through the sphenoid body and lesser wing of the sphenoid. Globes appeared intact both clinically and

KEYWORDS: ocular trauma; acute vision loss; hyperbaric oxygen therapy; ophthalmologic trauma

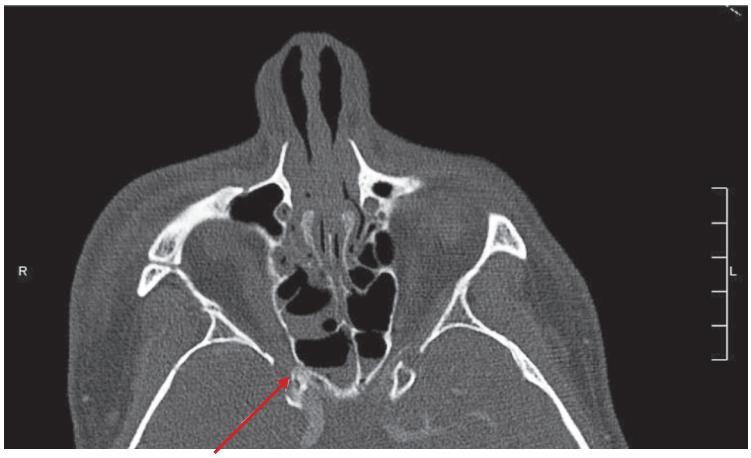

FIGURE 1: Axial computed tomography scan showing fracture of the right optic canal (red arrow).

radiologically. There were no other intracranial findings. The patient did not have eye pain or pain on eye movement, flashes, or floaters, but he described total loss of vision on the right. Initial ophthalmic examination revealed no light perception vision in the right eye, a 4.5-mm non-reactive pupil with 3+ APD, normal intraocular pressure, full motility, mild right periorbital edema and ecchymosis, and a grossly normal anterior segment and dilated fundoscopic examination. He had 20/20 vision in his left eye.

Given the severity of the TON, the morbidity associated with no light perception, and the patient's young age, we initiated high-dose steroid therapy with solumedrol, 1 gm intravenously daily, and HBO_2 therapy. The patient was treated emergently according to U.S. Navy Dive Table 5 (our institution's protocol for central retinal artery occlusion) on the day of presentation within six hours of presentation. We treated him quickly because the nervous system tissue was at risk and we were concerned about permanent damage to his vision.

On day two, the patient received two HBO_2 treatments at 2.4 ATA for 90 minutes. After the third treatment, his vision had improved significantly to 20/100 on the right with a 1+ APD and associated inferior hemifield defect with markedly diminished color saturation and brightness. He was treated with three additional daily HBO_2 sessions (2.4 ATA for 90 minutes) and daily high-dose steroids.

The patient's vision continued to improve, with the improvement plateauing to 20/40 after the fourth treatment. On treatment day two, we began administration of minocycline, 100 mg orally twice daily for its potential neuroprotective effects [7]. The patient was discharged after receiving six HBO_2 treatments and six days of solumedrol (1 gm IV) that was transitioned to oral prednisone (1 mg/kg) and tapered appropriately.

At his 2-month clinic follow-up, the patient's best corrected visual acuity was 20/60+2 on the right. Humphrey visual field testing confirmed a dense inferior hemifield defect (Figure 2). Optical coherence tomography and retinal nerve fiber layer analysis indicated significant thinning of the temporal quadrant and mild superior quadrant thinning (Figure 3).

DISCUSSION

HBO_2 therapy use in ophthalmology has generally been confined to the treatment of ischemic injury from central retinal artery occlusion and radiation retinopathy; however, there are limited case reports on its use in humans in the treatment of direct TON [8, 9]. Researchers have proposed that the mechanism of HBO_2 might be based on its neuroprotective effects against hypoxic or ischemic insults by down-regulating pro-apoptotic factors and up-regulating endogenous antioxidative enzymes and brain-derived neurotropic factors, resulting in ischemic tolerance. Using animal models of optic nerve crush injuries, investigators have shown reduction of retinal ganglion cell loss and decreased pro-apoptotic gene expression with use of HBO_2 [7, 10].

The current report features concurrent use of HBO_2 and additional medical treatments with steroids and minocycline. Steroid treatment for optic neuropathies has been widely studied but remains controversial. The significant risks of steroids are well known and include decreased immunity, poor glycemic control, and ulcerative gastrointestinal disease. Given our patient's profound vision loss on presentation, the lack of additional systemic injuries, and younger age, we concluded that steroid benefit was greater than the risk of medication complications.

In addition, we chose to use minocycline to optimize the care of this otherwise healthy patient. Minocycline is a tetracycline antibiotic with a few unique features. It is a broad-spectrum antimicrobial, with anti-inflammatory properties, excellent tissue distribution, and the ability to enter the central nervous system. It has been studied for its positive effects on microgli-

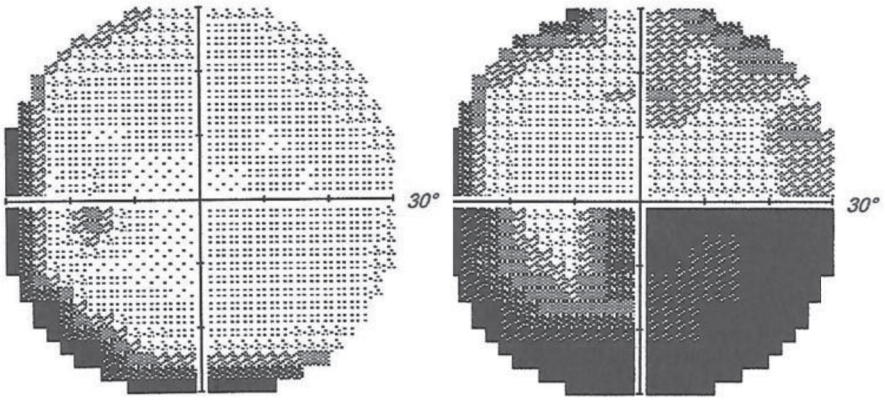

FIGURE 2: Humphrey visual fields, 30-2, obtained two months after injury
(by convention, with the left eye field presented on the left).
Demonstrates a rim artifact in each eye and an inferior altitudinal defect of the right eye.

Acknowledgment: Photography courtesy of Karen Klima, COT.

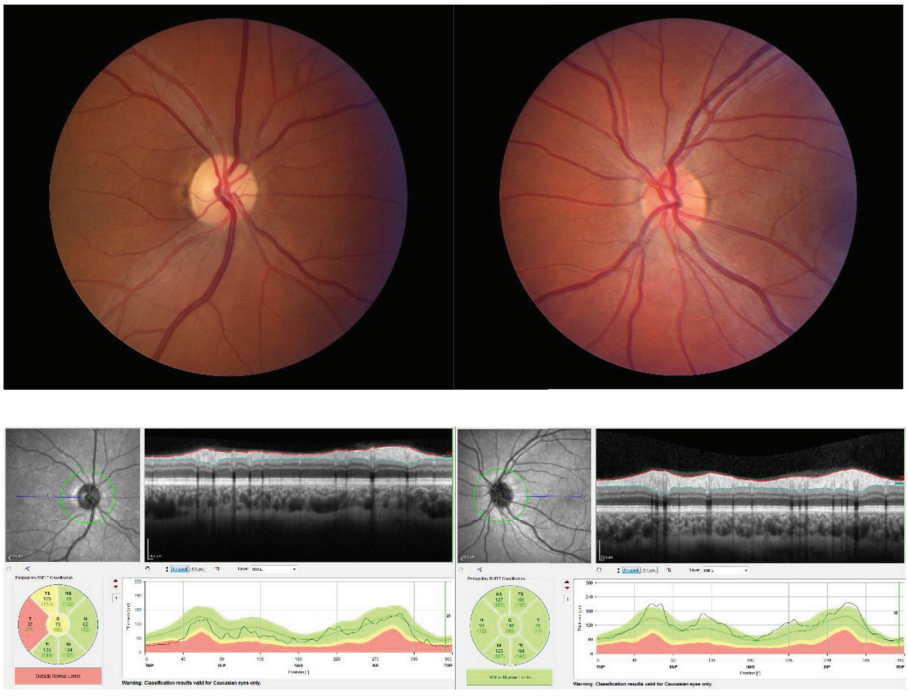

FIGURE 3: Optic nerves and optical coherence tomography of the retinal nerve fiber layer
two months after injury. These images demonstrate pallor of the right optic nerve
and thinning of the nerve fiber layer in the right eye.

al activation, matrix metalloproteinases, nitric oxide production, and apoptosis. It has shown neuroprotective effects in rodent models of spinal cord injury, stroke, multiple sclerosis, and Parkinson's disease. Minocycline effects have been studied in ophthalmic diseases, including retinitis pigmentosa and retinal vein occlusion [11-13]. The risks and benefits of each intervention were discussed with our patient, and he elected to proceed.

LIMITATIONS

Much of our management of this patient was based on our prior experience with acute conditions affecting the optic nerve. We treated him quickly because of the risk of ischemia to the optic nerve and vision loss. The addition of minocycline and steroids was suggested by the neuro-ophthalmology consultant because of animal studies of its neuroprotective effects. There are no large human studies that support this practice. Although unlikely because of the direct traumatic event, it is possible that the patient's vision would have improved spontaneously.

CONCLUSION

While there is no accepted standardized treatment protocol for TON, and as the use of high-dose steroids and optic nerve decompression is considered controversial in some instances, we believe that emergently treating patients with HBO_2 for ischemic and mechanical injury to the optic nerve following trauma is an under-studied and under-utilized therapeutic option that deserves further investigation.

Conflict of interest statement
The authors have declared that no conflict of interest exists with this submission.

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