Choroidal Rupture Secondary to Treble Fishhook Ocular Injury

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ABSTRACT

Introduction: Choroidal rupture is a vision-threatening complication of blunt ocular trauma but is rarely reported in association with fishing-related injuries. We describe a case of choroidal rupture following ocular trauma from a fishing lure.

Case presentation: A 9-year-old boy presented with a penetrating fishhook right eye injury from a treble hook lure. Ophthalmic examination and imaging confirmed choroidal rupture. Initial visual acuity was 20/200 in the right eye and improved to 20/60 at 2 months postinjury. At 6 months, visual acuity declined to hand motions, and optical coherence tomography showed choroidal neovascularization (CNV). The patient underwent anti-vascular endothelial growth factor (VEGF) injection under general anesthesia. At most recent follow-up visual acuity was 20/50.

Discussion: Fishing injuries typically cause vision loss through penetrating or perforating ocular trauma. Although blunt trauma is a recognized cause of choroidal rupture and vision loss, this mechanism from a fishing lure is rarely described. Close monitoring for secondary CNV is essential, and anti-VEGF therapy can effectively reduce subretinal and intraretinal fluid, resolve hemorrhage, and treat choroidal neovascularization.

Conclusions: This case highlights the potential for blunt ocular trauma from fishing lures to cause choroidal rupture and vision loss. Preventive strategies, including eye protection and adult supervision, are critical to reduce the risk of similar injuries.

INTRODUCTION

Ocular injuries are a significant cause of visual impairment in the United States. According to data from 2008 in the Nationwide Emergency Department Sample, ocular injuries result in an average of over 1700 emergency department (ED) visits per day, and males are almost twice as likely to be affected as females. These injuries usually occur as a result of work- or sports-related accidents, with the majority occurring before age 44.[1] Multiple databases consistently show that ocular injuries occur more frequently in young males than in other groups.[1,2] Additionally, ocular trauma disproportionately affects rural communities, with ED visits for such injuries occurring 5.4 times more often in rural areas than in urban areas.[1] The long-term morbidity of these injuries is especially concerning, as data from the United States Eye Injury Registry (1988–2003) indicate that fewer than two-thirds of patients with severe eye trauma regain visual acuity better than 20/200 after treatment.[2]

Although rare overall, fishhook injuries represent a substantial portion of sports-related eye traumas and typically are categorized as penetrating or perforating injuries.[3] Complications such as infection, intraocular hemorrhage, traumatic cataract, corneal scarring, and retinal detachment have previously been reported in cases of penetrating fishhooks injuries;[4] however, we report a case of blunt trauma from a fishhook ocular injury that resulted in choroidal rupture and vision loss.

CASE PRESENTATION

A 9-year-old boy presented to a rural ED following penetrating ocular trauma caused by a treble hook lure. On arrival, a towel was attached to the fishhook, as the family had attempted to cover the eye. Per interview, the patient had been practicing casting with a fishing pole and treble hook lure in his driveway when he reeled back and the lure struck his right eye.

External examination revealed one hook embedded in the right upper eyelid, one free hook, and one deeply embedded hook not

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Figure 1. Initial Penetrating Injury

Penetrating treble hook injury with blunt force trauma to right upper eyelid and globe from fishing lure.

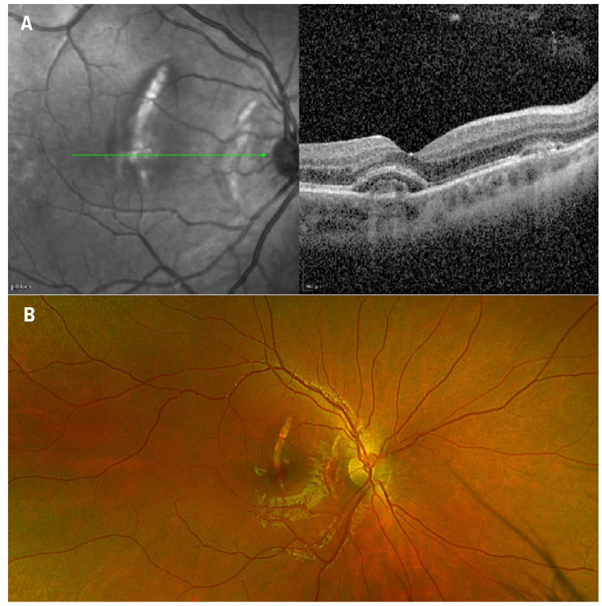
fully visualized. At the outside ED, an ED physician sedated the patient with ketamine and cut the lure from the hook. The hook in the eyebrow was extracted using the 18-gauge needle-over-the-barb technique. However, the final hook was found embedded in the sclera. A paper cup was placed over the eye for protection, tetanus status was reviewed, cefazolin was administered, and ophthalmology was then consulted. Ocular pressures were not checked initially due to concerns about an open globe injury.

The patient was transferred to a tertiary care hospital for specialized ophthalmologic management. Initial presentation showed a fishhook embedded in the right upper eyelid and eye (Figure 1). Orbital computed tomography (OCT) confirmed a right globe injury with a retained metallic fishhook spanning the lateral corneal limbus. Initial visual acuity was 20/200 in the right eye and 20/25 in the left eye based on a standard Snellen distance chart. He was given intravenous moxifloxacin as prophylaxis and brought to the operating room for emergent open globe repair and examination under anesthesia.

Under anesthesia, the fishhook was visualized perforating the right upper eyelid and penetrating the superolateral sclera 3 mm posterior to the limbus. The barb tip from one hook was found to penetrate the conjunctiva, Tenon's capsule, and sclera. Fortunately, the proximal barbed portion did not fully penetrate the sclera, allowing removal in the same trajectory as the injury without further damage to the underlying uveoscleral tissue. The hook was grasped with a locking hemostat and removed. The sharp end of the hook was rotated away from the globe, and a wire cutter was used to cut the barb, which was then discarded. The remaining hook was then removed from the eyelid without resistance.

Examination of the globe using the operating microscope

Figure 2. Images Three Days Postinjury



A. Initial orbital computed tomography 3 days after injury and open globe repair shows crescent-shaped subfoveal choroidal rupture.
B. Initial fundus photography shows subfoveal choroidal rupture 3 days after injury and open globe repair.

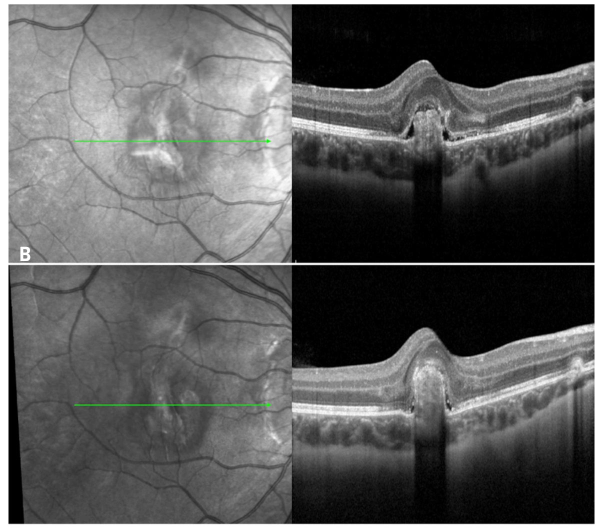
revealed a 1.5-mm scleral laceration with exposed pigmented uveal tissue. The globe was well-formed, including the anterior chamber. The uveal tissue was excised, and a single 9-0 nylon scleral suture was placed. Three 8-0 polyglactin 910 sutures were placed to close the conjunctiva. The wound was Seidel negative. Subconjunctival injections of cefazolin and betamethasone were administered.

The right upper eyelid laceration was repaired using 6-0 gut suture. Drops of phenylephrine 2.5% and cyclopentolate 1% were used to dilate both eyes prior to a dilated eye exam under anesthesia. The affected eye showed a clear lens without subluxation or evidence of traumatic cataract, and the optic nerve appeared normal. The macula exhibited two crescentic white bands, consistent with choroidal rupture. No retinal or vitreous hemorrhage was noted. Following the examination, the eye was patched and shielded for protection, and the patient was extubated.

Postoperatively, the patient continued to experience blurry vision. Visual acuity was 20/200 in the right eye and 20/20 in the left eye. Visual fields were full, and no afferent pupillary defect was noted. A handheld electronic tonometer was used to measure intraocular pressures, which were within normal limits. Retina specialist evaluation included a detailed fundus examination using a Haag-Streit slit lamp, and imaging (ultra-widefield fundus photography, OCT of the macula, and OCT angiography of the retina) confirmed choroidal rupture without choroidal neovascularization (CNV) (Figure 2).

The patient's vision improved to 20/125 at 1 month and 20/60 at 2 months postinjury. Repeat OCT and fundus photography showed resolved subretinal hemorrhage and scar formation. Notably, in the early postoperative period, the patient did not develop endophthalmitis, traumatic cataract, or retinal detachment, and there was no evidence of CNV. His visual impairment was attributed to the choroidal rupture from the blunt trauma. However, at 6 months postinjury, he developed blurred vision and presented urgently to the retina clinic. Examination and imaging demonstrated visual acuity of hand motions and a choroidal neovascular membrane (CNVM) with associated subretinal fluid in the affected eye. He underwent anti-vascular endothelial growth factor (VEGF) treatment under general anesthesia, and 1 month later, visual acuity improved to 20/60. At the most recent follow-up, 2 months posttreatment, visual acuity was 20/50 with involuted CNV and no subretinal fluid or hemorrhage (Figure 3).

Figure 3. Images at Follow-up and After Anti-Vascular Endothelial Growth Factor Injection



A. Orbital computed tomography (OCT) 6 months after injury and open globe repair shows development of choroidal neovascularization with associated subretinal fluid with visual acuity hand motions.

B. OCT approximately 2 months after anti-vascular endothelial growth factor injection with near complete resolution of subretinal fluid with improvement of visual acuity from hand motions to 20/50.

DISCUSSION

Ocular injuries are a common sports-related eye injury and cause of visual impairment. These injuries account for more than 630 000 ED visits annually, many of which could be prevented with improved safety practices.[1] In this case, the patient was not wearing eye protection while practicing casting independently. While the Wisconsin Department of Natural Resources recommends adult supervision of children while fishing, no specific guidelines address eye protection. For beginners learning to cast, safety can be enhanced by practicing without a lure or hook. Establishing clear guidelines and raising awareness of ocular injuries are important to prevent future incidents.

When ocular injuries occur, timely and appropriate initial evaluation is critical. Most often, the first assessment is conducted by nonophthalmologist clinicians.[1] Therefore, providers in primary care and the ED should be well prepared to assess and manage these cases in the early stages–especially in rural settings where access to an ophthalmologist may be limited. The assessment of ocular trauma should begin with evaluation of any life-threatening injuries. Then, a thorough history should be taken to address the mechanism of injury, setting and timing of injury, ocular review of systems, and past ocular history, including the patient's visual acuity prior to the injury. Relevant exam includes assessment of visual acuity, relative afferent pupillary defect, and confrontation visual field testing. If an open globe is suspected, an eye shield should be placed for protection, and tonometry should be deferred to avoid extrusion of ocular contents. Manipulation of the eye and removal of any visible foreign body should be deferred to an ophthalmologist.[5]

Although perforating or penetrating ocular injuries from fishhooks are the most commonly reported mechanisms, as seen in our patient, it is possible for a combined mechanism of penetrating and blunt force trauma from a fishing lure to cause ocular injury and vision impairment as well. Therefore, thorough ocular examination and imaging at follow-up are required to evaluate and manage less common fishing-related eye injuries.

One possible consequence of blunt ocular trauma is traumatic choroidal rupture, which is caused by shear stress and occurs in approximately 5% to 8% of cases. Patients may experience decreased vision, scotoma, and visual field defects.[5] The degree of visual impairment immediately following choroidal rupture depends on the location of the rupture, the degree of associated choroidal or vitreous hemorrhage, and other associated ocular injuries. Patients who sustain subfoveal choroidal ruptures, as in our patient, generally present with the worst visual acuity. In early stages of evaluation, choroidal ruptures appear as white to yellow-red crescent-shaped lesions and are often associated with retinal edema and hemorrhage. Angioid streaks and lacquer cracks have similar appearances and can be mistaken for choroidal rupture. In the weeks following choroidal rupture, a fibrotic or gliotic-appearing scar forms over the crescentic lesion, followed by hyperpigmentation at the margins of the healing lesions.[6]

In large part, the growth of CNV determines the likelihood of visual recovery. As part of the expected healing process, CNV is a normal occurrence following choroidal rupture, with 5% to 12% of patients with choroidal rupture developing CNV and 80% of CNV presenting within the first year after injury.[6,7] Newly formed blood vessels often regress once structural repair of the rupture has occurred. However, in 5% to 25% of cases, CNV persists after this initial period of healing and can further exacerbate visual impairment by causing loss of central vision, decline in visual acuity, metamorphopsia, and scotoma.[8] During this time, careful observation and close patient follow-up is appropriate.

Most patients with a choroidal rupture do not recover a final visual acuity of 20/40 or better.[9] Long term, children may have improved visual outcomes compared with adults due to increased CNV in older ages. Thus, patients who sustain choroidal rupture injuries earlier in life may be more likely to regain visual acuity. From a histopathology standpoint, CNV in older eyes are thicker and more extensive, vascular, and cellular. The difference in CNV formation based on age is thought to be due to age-related decreased expression of angiogenic growth factors, loss of the proliferative capacity of aging endothelial cells, or loss of the expression of endothelial enzymes responsible for digesting the extracellular matrix.[10]

For some cases in which CNV persists beyond the initial stages of healing, anti-VEGF injections have been used to slow the disease process by inhibiting the progression of the CNVM. Clinical trials such as MINERVA demonstrate that anti-VEGF therapy is safe and effective for CNV treatment in adults.[11] However, there is hesitancy in using anti-VEGF in pediatric patients due to potential short-term adverse events, including endophthalmitis, other ocular inflammation, rhegmatogenous retinal detachment, ocular hypertension, cataracts, corneal opacification, and vitreous hemorrhage. Long term, anti-VEGF therapy may lead to abnormal retinal vasculature, shunt vessels, and refractive errors.[12] However, despite these potential side effects, anti-VEGF has been used in the setting of a choroidal rupture injury and posttraumatic CNV to safely provide improvement to visual acuity.[13,14] Additionally, intravitreal injection of anti-VEGF is utilized in infants with retinopathy of prematurity, Coats disease, retinoblastoma, CNV, and other advanced pediatric vitreoretinal diseases.[15] Future studies to evaluate long-term safety in pediatric populations are warranted.

CONCLUSIONS

This is the first reported case of choroidal rupture from a fishing lure injury. Given the common association of fishing injuries with globe penetration or perforation, awareness should be raised surrounding the possibility of blunt trauma and choroidal rupture, especially if a heavy lure or weight is used and strikes the eye. In these cases, thorough examination and follow-up are essential to optimize visual recovery and inform further treatment. In the event of secondary CNV, anti-VEGF injection may be warranted and requires special consideration in pediatric patients because of anesthesia requirements and limited long-term safety data.

Preventive strategies are critical. Improved guidelines that emphasize eye protection and adult supervision for children could reduce the risk of similar injuries in the future. Efforts are underway to advocate for enhanced safety recommendations through the Wisconsin Department of Natural Resources, including routine use of protective eyewear during recreational fishing.

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