Boa Constrictor Bite to the Eye

Ocular injury to the eye from snake bite is extremely rare with few cases being reported in the literature. We report the case of man who sustained a penetrating injury to the eye from a snake bite.

Report of a Case. An 18-year-old man was bathing his pet snake, a 6-ft-long North Brazilian boa constrictor (Boa constrictor), when it attacked him and bit him on the right eye. The snake had infectious stomatitis, a bacterial infection in the mouth. When the snake struck, the patient partially blocked the attack with his right hand; however, the snake was able to engage the patient’s right eye with its lower teeth, and his hand with its upper teeth. It would not release its bite and tried to wrap around the patient’s neck. The patient managed to telephone a neighbor, who dialed 911, and the police arrived. The policeman who answered the call, however, was ophidiophobic (fear of snakes) and was unable to lend assistance. The fire department arrived shortly thereafter, and a fireman, using a large knife, cut the snake’s head from its body. The multiple small recurved teeth could not be disengaged, so the patient was transferred to a nearby emergency department with the snake’s head attached to his eye. The attending physician removed the snake’s head from the patient, and diagnosed a ruptured globe. Photographs were obtained before the snake’s head was removed (Figure 1). After receiving 1 g of the ampicillin and sulbactam sodium combination drug (Unasyn) intravenously, the patient was transferred to our institution with a shield over his right eye while awaiting definitive treatment. The head of the snake was sent in a specimen bag (Figure 2).

When the patient arrived, he was in minimal discomfort. His eyelids were mildly swollen on the right side. There was a puncture wound in the right upper eyelid. Visual acuity was 20/50 OD. Three small puncture wounds were noted in the cornea; 2 were Seidel positive. A small conjunctival puncture wound was also noted in the inferonasal quadrant. The anterior chamber was deep and fibrin was adherent to the internal surface of the wound. The retina and vitreous humor appeared normal. Initial management consisted of a bandage contact lens and topical and intravenous antibiotics; ofloxacin was applied topically every hour, and standard doses of intravenous vancomycin hydrochloride, cefazidine, and clindamycin were given. The next day, the chamber had shallowed, and 1 wound remained Seidel positive. Glue was applied, but over the next 48 hours the wound failed to seal, so the patient was taken to the operating room where 2 No. 11-0 nylon sutures were sewn in place. The patient was discharged from the hospital after receiving a 72-hour course of prophylactic antibiotics with no signs of infection. Three months postoperatively, the patient’s best-corrected visual acuity is 20/25 OD (Figure 3).

On arrival at the hospital, cultures were made from the teeth of the snake, and numerous species of gram-negative rods were identified. No further attempt to classify the bacteria was made, as the patient did not develop an infection.

Comment. In the United States, approximately 50 000 people per year are bitten by snakes, most of which are nonvenomous.1 We reviewed MEDLINE from 1966 to the present and found only 2 cases of snake-bites to the eye.2,3 Both patients were bitten by venomous snakes and both were children. One patient eventually required enucleation and the other recovered. The patient who recovered was bitten on the medial canthus and did not suffer a penetrating ocular injury.

Infectious stomatitis is a relatively common infection in captive snakes.4 This disease is known to occur when snakes are stressed environmentally by poor husbandry. The most common predisposing cause is not providing the snake its preferred optimal temperature zone, which decreases the effectiveness of the animal’s immune system and allows opportunistic pathogens to cause disease. Gram-negative organisms such as Pseudomonas, Salmonella, Klebsiella, and Peromonas species are frequently implicated. A culture made from the boa constrictor’s mouth from this case yielded multiple organisms consistent with bacterial stomatitis.
Most snakebites probably do not need prophylactic antibiotics. In studies on snakebites, fewer than 5% of patients had resultant infections.1 Nevertheless, we treated our patient with a 72-hour course of prophylactic antibiotics that was initiated almost immediately. The concern for infection in this case was considerable, because the snake had bacterial stomatitis, the patient’s cornea was punctured, and endophthalmitis is potentially devastating to vision. We believe that prophylactic antibiotics may be indicated for snakebites when the development of an infection would have very serious consequences, such as with a bite to the eye.

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Amebic Keratitis Due to Vahlkampfia Infection Following Corneal Trauma

Acanthamoeba keratitis occurs in association with contact lens use, minor corneal trauma, or contact of the eye with contaminated water. It is a relatively uncommon but potentially sight-threatening keratitis. In mild to moderate cases, medical treatment alone can eradicate the infection; however, in severe cases surgical treatment is usually required.1 Other members of the ameba family rarely cause ocular infection. A case of contact lens-related amebic keratitis due to a mixed infection of Vahlkampfia and Hartmannella was recently reported.2 Both patients were contact lens wearers.

We report herein the second case of amebic keratitis secondary to Vahlkampfia infection. To our knowledge, this is the first case of amebic keratitis following minor corneal trauma in a patient who did not wear contact lenses, and the first report of a non-Acanthamoeba amebic keratitis in the United States.

Report of a Case. A 30-year-old man came to our institution with severe pain and irritation in the right eye. One month previously he had sustained corneal abrasion to the right eye from fiberglass while working on his boat and subsequently self-irrigated the eye with tap water. He was then seen by his private ophthalmologist and was treated with a combination ointment of neomycin sulfate, polymyxin B sulfate, and bacitracin zinc (Neosporin, Glaxo Wellcome Inc, Research Triangle Park, NC) after yields from a corneal culture were examined. No organism was isolated from the initial corneal cultures. No notable improvement of his symptoms and corneal findings was noted after 1 month. He was referred to our institution for further evaluation. On ophthalmic examination, his best-corrected visual acuity was 20/50 OD and 20/20 OS. Intraocular pressure was normal in both eyes. Slitlamp examination of the right eye revealed a midstromal infiltrate with an overlying epithelial defect and localized corneal edema (Figure 1). No other ocular abnormality was noted. Corneal scrapings were obtained for culture and the patient was given empiric treatment with ciprofloxacin under the assumption that he had bacterial keratitis. The patient continued to have persistent in-
traocular inflammation with stromal infiltrates. Two weeks later, a second set of scrapings was obtained, and the patient’s treatment was switched to fortified vancomycin hydrochloride and ceftazidime. Yields from routine culturing of both sets of corneal scrapings were negative for organisms. Despite intensive antibiotic treatment, he remained symptomatic. One week later, yields from an agar-agar culture obtained from a third set of corneal scrapings revealed a moderate amount of cysts and trophozoites. The organisms were also identified by Giemsa stain (Figure 2) in both scraping and culture yields. They appeared smaller and morphologically distinct from *Acanthamoeba* and were identified as *Vahlkampfia*. The patient subsequently began receiving routine antiamoebic treatment that included propamidine; polyhexamethyl biguanide (Bacquacil); the combination ointment of neomycin sulfate, polymyxin B sulfate, and bacitracin zinc; and clotrimazole every 2 hours with rapid resolution of symptoms. He was maintained on a regimen of propamidine (Brolene) and Bacquacil for a month. His final visual acuity was 20/20 with complete resolution of the stromal infiltrate.

Comment. *Vahlkampfia* is a free-living ameba found in water and soil, and it belongs to the same family as *Naegleria*. Two cases of encephalitis presumably due to *Vahlkampfia* have been reported. The first case of *Vahlkampfia* keratitis was in a 24-year-old contact lens wearer from whom amebas were isolated from the corneal biopsy tissue, contact lens, case, and home water supply. He was treated with amphotericin B; the combination ointment of neomycin sulfate, polymyxin B sulfate, and bacitracin zinc, and propamidine. However, eventual penetrating keratoplasty secondary to central corneal scarring was required. In our case, corneal abrasion and self-irrigation with tap water predisposed the patient to such an unusual infection. The *Vahlkampfia* was finally isolated only after repeated corneal scrapings, emphasizing the difficulty in culturing these organisms. Given the difficulty growing and identifying these amebic organisms, it is impossible to know the prevalence of non-*Acanthamoeba* amebic keratitis. Special techniques such as the polymerase chain reaction or organism-specific immunohistochemistry may facilitate identifying this kind of unusual amebic keratitis. Our patient’s rapid response to the standard treatment for *Acanthamoeba* keratitis indicates that *Vahlkampfia* is also sensitive to these medications. To the best of our knowledge, this case is the first report in the United States of keratitis due to an ameba other than *Acanthamoeba*, and the first case not associated with contact lens usage.

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**Postoperative Streptococcus pneumoniae Endophthalmitis Complicated by Meningitis**

The most common causes of bacterial endophthalmitis are ocular surgery, usually cataract extraction, and...
Endogenous spread from other infections such as meningitis, abdominal infection, endocarditis, and urinary tract infection. The clinical course is highly variable and depends on the virulence of the infecting organism, the quickness of diagnosis and administration of antibiotics, and the patient's underlying medical condition. According to the Endophthalmitis Vitrectomy Study² conducted between 1990 and 1994, which studied the role of immediate vitrectomy and of intravenous antibiotics in the management of postoperative bacterial endophthalmitis, on post hoc data analysis, there was no difference in visual outcome whether or not an immediate vitrectomy was performed, except in a selected subgroup of patients. Furthermore, no ocular benefit was derived from the administration of systemic antibiotics. We reviewed the medical record of a patient who developed meningitis following a postoperative case of Streptococcus pneumoniae endophthalmitis. Since the advent of antibiotics, no other cases have been reported of systemic spread from a primary exogenous bacterial endophthalmitis, to our knowledge.

Report of a Case. An 81-year-old woman with Fuch corneal dystrophy complained of severe pain in her left eye 1 day following a penetrating keratoplasty and cataract extraction. The donor cornea was from a 3-year-old child who had drowned. On examination, she had hand motions/light perception only visual acuity, an intraocular pressure of 49 mm Hg, conjunctival chemosis, keratic precipitates on the corneal graft, a heavy cellular reaction in her anterior chamber, and a +4 vitreous reaction that obscured retinal details in her left eye. The results of the remainder of her physical examination were normal. At this time, it was thought that she had postoperative endophthalmitis, and after consultation with physicians from both the retina and uveitis services, she was admitted for a vitreous tap and intravitreal injection as well as being started on a regimen of fortified topical antibiotics. She was treated with an intravitreal injection of vancomycin hydrochloride (1 mg), gentamicin sulfate (0.4 mg), and dexamethasone phosphate (0.4 mg), as well as topical vancomycin (50 mg/mL), tobramycin sulfate (14 mg/mL), and 1% prednisolone acetate. No systemic antibiotics were given. Ocular and orbital ultrasonography were performed and showed that no retinal detachment had occurred and no obvious extraocular inflammation was present. Cultures from the donor corneal rim and the vitreous humour both grew S pneumoniae sensitive to penicillin, clindamycin, and erythromycin.

On the seventh hospital day, the patient suddenly became confused and agitated and was found to have a stiff neck, a temperature of 38.9°C, and to be tachycardic. Her white blood cell count was 18.6 × 10⁹/L. A lumbar puncture was performed that showed pink, turbid cerebrospinal fluid containing high levels of protein and abundant neutrophils and erythrocytes, as well as gram-positive diplococci. A diagnosis of meningitis secondary to endophthalmitis was made and a regimen of intravenous penicillin was started. Blood cultures obtained at that time later confirmed the presence of S pneumoniae sensitive to penicillin and chloramphenicol. Cerebrospinal fluid cultures yielded no bacterial growth. The patient recovered quickly once the intravenous antibiotics were given and was in her normal neurological state the next day.

During the patient’s course in the hospital, the vision in her left eye did not improve and her intraocular pressure continued to rise despite maximal therapy. Magnetic resonance imaging of the head was performed and ruled out any further intraorbital or intracranial abscesses. A repeated orbital ultrasonogram showed dense vitreous opacities, vitreous membrane formation, and partial posterior vitreous detachment. After she was pronounced medically stable with her meningitis resolving uneventfully, a vitrectomy was performed, although her visual acuity never improved.

Comment. Most cases of bacterial endophthalmitis are caused by gram-positive organisms, notably Staphylococcus epidermidis, Staphylococcus aureus, and various Streptococcus species, including S pneumoniae.³,⁴ Endophthalmitis resulting from gram-negative organisms and fungi generally predisposes to an unfavorable clinical outcome. When compared with endophthalmitis due to other gram-positive organisms, infection by nonviridans Streptococcus species appears to result in the worst clinical outcome by a significant margin.⁴ This relatively poor outcome may be due to the greater degree of inflammatory response evoked by streptococcal exotoxins and enzymes.

In addition, there may be increasing reports of Streptococcus bacteremia and secondary endophthalmitis, particularly in patients with underlying medical conditions such as diabetes mellitus, malignant neoplasms, and human immunodeficiency virus infection.³ While the Endophthalmitis Vitrectomy Study² clearly showed no ocular benefit from systemic antibiotics overall, there may have been specific causal subgroups that would derive benefit from intravenous antibiotics; however, data were insufficient for drawing statistical inferences. Furthermore, the study was designed to determine if there were any ocular, not systemic, benefits to using intravenous antibiotics.

Patients with bacterial endophthalmitis should be observed closely for signs or symptoms of metastatic spread. Although postoperative bacterial endophthalmitis is typically confined to the eye, this case report indicates that it is possible for the infection to spread to the central nervous system as well as other areas of the body. Aggressive treatment of endophthalmitis, possibly including intravenous antibiotics, may be considered in cases of particularly virulent pathogens and in patients with high-risk medical conditions.

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Miliary Tuberculosis

A 31-year-old male-to-female transsexual prostitute, a recent immigrant from Mexico, came to the emergency department disoriented and with an elevated temperature. Medical history was notable for recent purified protein derivative positivity on skin testing. A chest x-ray film at examination showed fine miliary opacities involving all lung fields (Figure 1). A computed tomographic scan of the head revealed prominent meningeal vascularity and multiple supratentorial and infratentorial enhancing lesions (Figure 2). A lumbar puncture specimen contained 618 white blood cells, of which 0.84 were neutrophils; 0.12, monocytes; and 0.04, lymphocytes. The diagnosis was presumed Mycobacterium tuberculosis infection and the patient was admitted for therapy with 4 drugs that included isoniazide, rifampin, ethambutol hydrochloride, and pyrazinamide. On the second hospital day, the ophthalmology service was asked to see the patient because of blurred vision of 2 months' duration. The patient was lethargic, with a best-corrected visual acuity of 20/40 in each eye. No afferent pupillary defect was present. External and anterior segment examination findings were normal. Fundus examination findings revealed multiple choroidal infiltrates involving the posterior pole in each eye (Figure 3, A, B). Serial fluorescein angiography showed early blockage and late staining of these lesions (Figure 3, C-G). Cultures from sputum and cerebrospinal fluid (Figure 4) grew M tuberculosis. The patient showed slow resolution of the multifocal choroiditis and improvement of mental status and visual acuity with continued treatment.

Mycobacterium tuberculosis is the most common infectious cause of death worldwide, accounting for almost 10 million fatalities per year.\(^1\) Recent immigrants to the United States appear to be at particularly high risk of infection.\(^2\) Tuberculous multifocal choroiditis, although uncommon, is well recognized,\(^3\) and can support the diagnosis of miliary, or disseminated, disease as was observed in our patient.

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Figure 3. Color fundus photographs of the right (A) and left (B) eyes show bilateral, multifocal choroiditis (arrowheads). Serial fluorescein angiographic photographs (C-F) show early blocking hypofluorescence and late-staining hyperfluorescence corresponding to areas of choroidal infiltrate, as well as mild, late leakage from the optic nerve heads in each eye.

Figure 4. High-power, brightfield photomicrograph shows typical cording of Mycobacterium tuberculosis organisms grown in culture from cerebrospinal fluid (Kinyoun acid-fast stain, original magnification ×400).
The oculocardiac reflex is a triad of bradycardia, nausea, and syncope. The ocular causes are numerous.\(^1\) Orbital causes also exist.\(^2\) The ophthalmic division of the trigeminal nerve is the afferent limb. The impulses pass through the reticular formation to the vagus nerve’s visceral motor nuclei. The efferent limb message is carried by the vagus nerve to the heart and stomach.

We report 3 cases of orbital floor fractures that entrapped the inferior rectus muscle and/or the orbital connective tissue to immediately produce the oculocardiac reflex in 1 case and was highly suggestive in 2 others. To our knowledge, there have been no previous reports of oculocardiac reflex caused by incarceration of orbital soft tissue in an orbital trapdoor fracture.

**Report of Cases.** *Case 1.* An 8-year-old boy who was pushed and hit the right side of his face, immediately developed diplopia, nausea, and vomiting. In the emergency department, his pulse rate was 58/min with a blood pressure of 111/56 mm Hg. The results of ocular examination were normal except for periorbital ecchymosis and limited vertical gaze that was worse with depression. Motility measurements revealed a 3-prism diopter (PD) right hypertropia (Figure 1, top). The computed tomographic scan revealed a non-displaced right orbital floor fracture with incarcerated orbital soft tissue in the maxillary sinus and entrapment of the inferior rectus muscle.

The patient was taken to the operating room 2 days later where an abnormal forced duction was confirmed. During surgery the orbital connective tissue septae and fat were freed from the fracture. The inferior rectus muscle was not directly visualized. The fracture site was repaired with a shave of cranial bone. The forced duction test was repeated at the completion of the surgery and results were normal.

Postoperatively, his pulse rate was 86/min with a blood pressure of 121/63 mm Hg. The nausea and vomiting subsided. Initially there was mild limitation with depression. Thirteen months after surgery the patient was orthophoric (Figure 1, bottom).

*Case 2.* A 12-year-old boy was hit with a brick in the right periorbital region, which caused binocular vertical diplopia, nausea, and vomiting. In the emergency department his pulse rate was 36/min with a blood pressure of 115/70 mm Hg. Results from an electrocardiogram revealed bradycardia along with some QRS complexes missing P waves. The ocular examination findings were normal except for ecchymosis on the right cheek, a 9-PD right hypertropia with limitation in upgaze, and abnormal vertical forced duction test results. The maxillary nerve sensation was symmetrically intact. The computed tomographic scan revealed a nondisplaced right orbital floor fracture with the right inferior rectus muscle entrapped in the maxillary sinus (Figure 2).

The patient was taken to the operating room 7 hours later. Intravenous atropine sulfate was given prior to induction. His pulse rate ranged from 70 to 110/min. A portion of the orbital floor was removed to allow extraction of the inferior rectus muscle. The inferior rectus muscle appeared ischemic, but after delivery from the maxillary sinus, it became perfused. The floor defect was repaired with a porous polyethylene sheet. Findings from repeated forced duction tests revealed no restriction.

Postoperatively, his pulse rate remained at 60 to 70/min and all QRS complexes were associated with P waves. The nausea and vomiting subsided. The extraocular motility examination findings revealed moderate inability to depress the right globe. He was discharged and the diplopia resolved.

*Case 3.* A 20-year-old man walked into a metal pipe striking his left cheek, which caused immediate pain, nausea, and vertical binocular double vision. His ocular examination findings were normal except for limitation of both upgaze and downgaze in the left eye.
In primary gaze, he demonstrated a 6-PD left hypotropia. The computed tomographic scan revealed a nondisplaced left orbital floor fracture with soft tissue herniation into the maxillary sinus that caused entrapment of the inferior rectus muscle.

Three days later, the patient was taken to the operating room. His preoperative pulse rate was 58/min; and his blood pressure, 132/72 mm Hg. An abnormal forced duction was noted on testing. Entrapped fatty connective tissue as well as muscular tissue were released from the fracture line. The muscle contained a hematoma with signs of ischemic necrosis. The fracture was repaired with a Silastic sheet. Forced duction test results were normal. His postoperative pulse rate was 76/min with a blood pressure of 144/93 mm Hg. The nausea resolved.

Seven months after surgery, diplopia of more than 20° from primary persisted in extreme upgaze and downgaze.

**Comment.** The association of an oculocardiac reflex with an orbital fracture is rare, but prompt identification and treatment are important. The risk of a fatal cardiac arrhythmia exists (1:3500) with the oculocardiac reflex.1 In addition, the rhythmia exists (1:3500) with the trigeminal nerve triggers the oculocardiac reflex. A patient experiencing the triad of bradycardia, nausea, and syncope following orbital injury should immediately undergo computed tomography with coronal sections. If a trapdoor fracture with incarceration of soft tissue is identified, the fracture should be repaired the same day. Timely fracture reduction and release of the muscle and/or connective tissue protect against not only life-threatening cardiac arrhythmias but also ischemic necrosis of the muscle that may lead to fibrosis and permanent restrictive strabismus.

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**Identical Twins No Longer Discordant for Leber’s Hereditary Optic Neuropathy**

In 1993, Johns and colleagues1 described 2 monozygous twin brothers who had remained discordant for the development of Leber’s hereditary optic neuropathy (LHON) for 6.5 years. Both brothers were found to harbor the identical homoplasmic 4216, 13708, and 11778 mitochondrial DNA mutations. Twin A developed bilateral optic neuropathy at the age of 34 years and had a visual acuity of 1/200 OU. At the time of the report, twin B was 41 years old and still had a visual acuity of 20/15 OU with no signs or symptoms of LHON. In this current report, updated information on twin B is provided and indicates that twin B has subsequently developed bilateral LHON.

**Report of a Case.** Twin B, now 45 years old, had remained healthy and visually asymptomatic until he noted painless, blurred vision while driving on the morning of September 23, 1997. The visual loss first occurred in the left eye and then in the right eye 1 hour later. The patient reported no trauma, recent illness, weight change, or any unusual environmental exposure. He has continued to smoke 1 pack of cigarettes daily and to drink 24 beers weekly for the past 25 years. He was a firefighter but had not worked in this capacity for 2 years. He has been a part-time painter and has used latex paint for many years. His only medication was Ex-Lax twice a month for constipation since age 38. He has experienced much stress in the past year due to an ongoing litigation against his former employer for a nonphysically related matter. His daily activity has remained unchanged except for starting an exercise regimen 1 month prior to the visual loss. His typical routine was a 1-mile light run, 45 sit-ups, and 30 push-ups twice a week.

On examination, best visual acuity was count fingers at 4 inches OU. Pupillary reaction in both eyes was sluggish to light with no relative afferent pupillary defect. Goldmann perimetry demonstrated large central scotomas in both eyes. Intraocular pressures were 17 mm Hg OU. The optic nerves were hyperemic with a cup-disc ratio of 0.5 and no peripapillary vascular telangiectasia. The maculae and retinal vasculature appeared normal.
Comment. Updated information indicates that the discordance for LHON reported previously for a pair of monozygous twin brothers is temporal and not absolute. The discordance of the onset of LHON is 11 years, and the phenotypic findings are similar.

Potential mitigating factors such as excessive tobacco and alcohol use have been suggested in the phenotypic expression of LHON. In this case, both twins smoked 1 pack of cigarettes daily. However, twin A drank 6 to 12 beers weekly, which is less than half of the amount consumed by twin B who in fact had a much later onset of LHON. Both twins were firefighters, and twin A experienced visual loss 2 weeks after exposure to smoke. In contrast, twin B had not worked as a firefighter for 2 years and reported no recent exposure to smoke. The only recent change in his routine was starting a light exercise regimen 1 month prior to the visual loss. He did, however, report experiencing much stress for more than a year due to ongoing litigation. Interestingly, both twins noted the onset of visual loss while driving. However, this is a common activity and has not been associated with the onset of LHON.

In short, no common identifiable epigenetic factor for the clinical expression of LHON is apparent for these 2 monozygous twins who had a discordance for the onset of LHON of 11 years. This suggests that potential epigenetic factors for the clinical expression of LHON are variable and may be numerous.

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