

Ocular envenomation by the equatorial spitting cobra: a case report and review of venom ophthalmia associated with *Naja* species

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Abstract

Snake venom ophthalmia is a rare and potentially blinding condition caused by the defensive projection of venom into the eyes by certain elapids, such as *Naja* species. While primarily a public health concern in tropical regions, its incidence is rising in Western countries due to the increasing popularity of exotic pet ownership and risks inherent in animal research. Despite the well-characterized systemic effects of *Naja* bites, significant gaps remain in the literature regarding the clinical course, optimal management strategies, and long-term visual outcomes of venom ophthalmia. We report a paediatric case of venom ophthalmia following direct ocular exposure to venom projected by the Equatorial spitting cobra (*Naja sumatrana*), accompanied by a brief review of the existing literature.

Key words: venom ophthalmia, ocular envenomation, *Naja* species, Equatorial Spitting Cobra, *Naja sumatrana*.

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Introduction

Snakes represent a remarkably diverse group of carnivorous reptiles, utilizing both mechanical and toxicological strategies for predation. While venomous snake species are taxonomically distributed across primary families such as *Elapidae*, *Viperidae*, and *Natricidae* – their methods of delivery vary significantly, ranging from direct injection to venom projection.¹ Within the family *Elapidae*, some species defensively projected venom from the venom gland toward the eyes of potential predators when provoked or threatened, resulting in a condition known as venom-induced ophthalmia.¹ The estimated annual incidence of venom ophthalmia is approximately 6 – 8 cases per 100,000 population.²

Globally, this condition is most frequently associated with *Naja* species such as *Naja sumatrana* in Southeast Asia,³ *Naja atra* in East Asia,^{4,5} *Naja pallida* and *Naja nigricollis* in Africa.^{2,6,7} In the United States, incidents involving *Naja nigricollis* are also noted, typically occurring in the context of captive or exotic animal handling.⁸ As the primary point of contact for projected venom, the ocular surface is particularly vulnerable to acute inflammatory injury. While most cases recover fully with prompt irrigation and topical treatment, delayed or inadequate management may result in serious complications such as corneal ulceration or perforation, glaucoma, or even permanent vision loss.¹

Despite the known systemic effects of *Naja* bites, a significant literature gap exists regarding the clinical progression, optimal management and long-term visual outcomes of venom ophthalmia. Although underreported, this condition is an endemic concern in tropical regions and is increasingly encountered in Western coun-

tries due to the rise in private collections of exotic spitting elapids.⁸ We report a case of venom-induced ophthalmia caused by the Equatorial spitting cobra (*Naja sumatrana*), accompanied by a focused review of the existing clinical literature.

Case Report

A 12-year-old female presented to the Emergency Department (ED) at Seri Iskandar Hospital approximately two hours after being sprayed in her left eye by the venom of an Equatorial spitting cobra (*Naja sumatrana*) at a distance of less than one meter, while at her residence. Following the exposure, the patient experienced immediate, debilitating ocular pain, blurred vision, and profuse epiphora, resulting in blepharospasm (Figure 1) The patient admitted to rubbing the affected eye prior to arrival.

Clinical examination demonstrated a Best-Corrected Visual Acuity (BCVA) of 6/18 in the left eye and 6/9 in the right eye, with no evidence of a Relative Afferent Pupillary Defect (RAPD). The patient reported a pain score of 7/10, accompanied by epiphora. There was no eyelid oedema. Anterior segment evaluation of the left eye revealed conjunctival hyperaemia without limbal ischaemia. Corneal assessment showed left eye corneal epithelial defect (Figure 2) The anterior chamber was deep, and the pupil was round and reactive to light. Intraocular pressure measured 14 mmHg. The crystalline lens was clear, and fundoscopic examination findings were unremarkable.

Initial irrigation began at a nearby clinic using 0.9% normal saline. In the ED, topical proparacaine was administered to the

affected eye for analgesia, followed by intensive re-irrigation with two litres of 0.9% normal saline to mitigate further ocular damage. Following ophthalmological evaluation, the patient initiated a tapering course of topical moxifloxacin 0.5%. The regimen consisted of bi-hourly administration for three days, followed by dosing every four hours for an additional four days, totalling a seven-day treatment course. Following this regimen, the patient demonstrated a complete clinical recovery at the one-week follow-up, with no remaining ocular sequelae. We obtained written parental consent from the mother.



Figure 1. Left eye of patient with blepharospasm and epiphora.

Discussion

The geographic distribution of *Naja* spitting incidents is broad, posing a notable public health concern across both Africa and Asia. In Africa, most reported cases are attributed to *Naja nigricollis* and *Naja pallida*.^{2,6,7} In contrast, Asian literature highlights *Naja sumatrana* as a predominant cause in Malaysia-Singapore peninsula and Indonesia³, *Naja atra* in China and Taiwan^{1,5}, and *Naja siamensis* in Laos.⁹ Notably, the present paediatric case differs from the typ-



Figure 2. Fluorescent staining showed left eye corneal epithelial defect.

Table 1. Venom ophthalmia caused by *Naja* species.

Study	Years of publication	<i>Naja</i> species	Country of origin	Age	Sex	Clinical presentation	First aid treatment	Treatment	Outcomes
L.J. Ang <i>et al.</i> ³	2014	<i>Naja sumatrana</i>	Singapore	28	Male	RE reduced VA, corneal epithelial erosions, conjunctivitis, chemosis, and corneal stromal oedema	Tap water and normal saline 0.9% irrigation	Chloramphenicol eye drops	No ocular sequelae
				23	Male	Corneal epithelial erosion and conjunctivitis	Tap water	Chloramphenicol eye drops	No ocular sequelae
				41	Male	LE conjunctivitis and corneal epithelial erosion	Tap water and normal saline 0.9% irrigation	Chloramphenicol eye drops	No ocular sequelae
Zhang Z <i>et al.</i> ⁴	2023	<i>Naja atra</i>	China	67	Female	LE pain, blurring of vision, epiphora, photophobia, conjunctival congestion, and epithelial punctate defects	Normal saline irrigation	Levofloxacin eye drops	No ocular sequelae
				21	Male	LE pain, blurring of vision, epiphora, conjunctivitis, and corneal epithelial defect	Normal saline irrigation	Levofloxacin hydrochloride eye gel	No ocular sequelae
Fung HT <i>et al.</i> ⁵	2010	<i>Naja atra</i>	China	83	Female	Bilateral eye pain, conjunctivitis, and corneal abrasions	1 litre normal saline irrigation	1% amethocaine eye drops, topical diluted <i>Naja naja</i> antivenom, 0.5% chloramphenicol eye drops	No ocular sequelae
Lanzetta M.A. <i>et al.</i> ⁶	2017	<i>Naja nigricollis</i>	Ghana	65	Male	LE reduced VA, corneal epithelial erosion, conjunctivitis, and chemosis	Tap water	Vitamin A ophthalmic ointment	No ocular sequelae
Handford C ⁷	2018	<i>Naja pallida</i>	Africa		Female	RE pain and conjunctivitis	0.5 litre clean water	Tetracaine eye drops, antibiotic eye drops, cyclopentolate eye drops	No ocular sequelae

LE, left eye; RE, right eye; VA, visual acuity.

ical epidemiological profile, as venom ophthalmia most commonly affects adult males between 22 and 83 years of age.²⁻¹⁰ Although most exposures occur accidentally, rare cases have also been documented in laboratory settings and among individuals involved in exotic animal husbandry.⁸⁻⁹ In addition to direct venom projection, venom ophthalmia may also occur through indirect exposure, such as rubbing the eyes after handling venom (e.g., among snake handlers or laboratory personnel)⁹ (Table 1 and 2).

Venom ophthalmia results from direct epithelial and stromal toxicity, where cardiotoxins, cytotoxins, and enzymatic components (proteases, collagenases, phospholipases) disrupt cellular membranes and extracellular matrix, leading to epithelial necrosis, corneal oedema, erosions, and possible ulceration.¹ Additionally, inflammatory mediators and neurotoxins induce conjunctival inflammation, uveitis, increased vascular permeability, and pupillary or cranial nerve dysfunction, with occasional immune-mediated keratoconjunctivitis contributing to the clinical picture.¹ The extent of tissue damage is largely dependent on the duration of ocular exposure. Although prolonged contact theoretically raises concerns regarding local necrosis or systemic absorption, current evidence indicates that systemic toxicity has not been reported following isolated spitting injuries.⁹

Clinically, the acute presentation of venom ophthalmia closely resembles that of a severe chemical burn. Patients typically experience immediate and severe symptoms, including intense ocular

pain, blurred vision, and blepharospasm. Examination frequently reveals marked conjunctival inflammation, corneal oedema, and diffuse punctate epithelial staining or defects.^{1,9} While the overall prognosis is favourable, with most patients achieving complete recovery, delayed or inadequate management may result in serious complications. These include iritis, hypopyon, corneal necrosis or perforation, and, in extreme cases, the need for enucleation.⁹

Immediate and copious ocular irrigation remains the cornerstone of management. While tap water is acceptable as an initial first-aid measure, clinical management should prioritize isotonic crystalloid solutions such as 0.9% normal saline or Ringer's lactate. Although the volume and duration of irrigation should be guided by the severity of exposure, a minimum of two litres is recommended to ensure adequate dilution and removal of venom. In this case, ocular irrigation was performed under topical anaesthesia with the patient supine, using normal saline delivered via a standard infusion set, with a kidney dish for effluent collection. Although this manual method proved effective, some clinicians advocate the use of a Morgan Lens, citing improved patient tolerance and continuous irrigation efficiency.³

To manage intense pain and facilitate effective irrigation and examination, the use of temporary topical agents is recommended. While mild vasoconstrictors with weak mydriatic activity such as epinephrine can help reduce blepharospasm, clinical practice often relies on the targeted use of local anaesthetics, including tetracaine

Table 2. Venom ophthalmia caused by Naja species.

Study	Years of publication	Naja species	Country of origin	Age	Sex	Clinical presentation	First aid treatment	Treatment	Outcomes
Goldman D.R. <i>et al.</i> ⁸	2010	<i>Naja nigricollis</i>	United States of America	30	Male	LE pain, LE reduced VA, eyelid oedema, conjunctivitis, and corneal epithelial erosion	Tap water, 2 litres normal saline 0.9% irrigation	Ciprofloxacin, topical cycloplegic Topical prednisolone acetate 1%	Corneal epithelial defect, reduced LE visual acuity
				22	Male	LE pain, LE reduced VA, conjunctivitis, pseudo membrane, and corneal epithelial defect	3 litres fluid	Ciprofloxacin hydrochloride eye drops, cyclopentolate eye drops, topical prednisolone acetate 1%	No ocular sequelae
Chu E.R. <i>et al.</i> ⁹	2010	<i>Naja nigricollis</i>	Nigeria	20	Male	RE burning pain, conjunctivitis, blepharitis, and discharge	Water and milk irrigation		No ocular sequelae
		<i>Naja pallida</i>			Male	RE pain, blurring of vision, blepharitis, blepharospasm, conjunctivitis, and discharge	Tap water irrigation		No ocular sequelae
		<i>Naja atra</i>			Male	RE corneal ulcer	Water irrigation		No ocular sequelae
		<i>Naja pallida</i>			Male	RE pain	Tap water irrigation		No ocular sequelae
		<i>Naja nigricollis</i>		40	Female	LE pain, conjunctivitis, reduced LE VA, corneal ulcer, and diffuse superficial keratitis	Milk and saline irrigation	Topical tetracycline, atropine drops, topical tetracortil	No ocular sequelae
		<i>Naja nigricollis</i>		Middle-aged	Male	Bilateral eyes pain, conjunctivitis, and eyelid oedema	Tap water irrigation		No ocular sequelae
		<i>Naja siamensis</i>		45	Male	Bilateral eyes pain and conjunctivitis		Topical epinephrine 1:10,000 drops	No ocular sequelae
		<i>Naja naja</i>		Middle-aged	Male	LE pain, conjunctivitis, eyelid oedema, left lower motor neuron facial nerve palsy	Tap water irrigation		No ocular and neurological sequelae
Sharma V.K. & Baranwal V.K. ¹⁰	2015		India	28		LE pain and redness. Reduce LE VA, conjunctival congestion, corneal epithelial defect	Normal saline 0.9% irrigation	Moxifloxacin 0.5% eye drops, homatropine 2% eye drops, topical heparin (2500 U/ml)	No ocular sequelae

LE, left eye; RE, right eye; VA, visual acuity.

hydrochloride or proparacaine, as utilized in this case.^{9,11,12} However, the administration of these agents must be strictly limited; prolonged use is associated with direct corneal toxicity and secondary complications. These include delayed epithelial healing, increased susceptibility to bacterial superinfection, chronic keratitis, and, in severe cases, corneal perforation or permanent scarring.⁹ Cycloplegic agents, including atropine or homatropine, are recommended to relieve ciliary spasm and prevent posterior synechiae, though clinicians should remain vigilant for the potential induction of acute angle-closure glaucoma in susceptible individuals.⁹

Prophylactic topical antibiotics are essential, particularly in the presence of corneal epithelial defects, to prevent secondary bacterial infection such as endophthalmitis.^{1,9,11,12} Commonly used agents include chloramphenicol, 0.5% moxifloxacin, and combination preparations containing polymyxin, neomycin, and gramicidin.^{1,3,9,11} Emerging evidence has also explored the use of topical heparin as a venom-neutralizing adjunct. Animal studies involving *Naja sumatrana* have demonstrated improved ocular outcomes following topical heparin administration, a benefit that is likely attributable to its acidic, negatively charged properties, which facilitate binding to and neutralization of cardiotoxins.¹³ Although human data remain limited, one study reported enhanced recovery when topical heparin was used alongside antibiotics and homatropine.¹⁰

Topical corticosteroids are generally contraindicated in venom ophthalmia due to their potential to precipitate herpes simplex keratitis and interfere with corneal healing. By altering calcium mobilization, steroids may enhance collagenase activity, resulting in corneal oedema and opacification.^{9,11,12} Our literature review identified three cases in which topical prednisolone was used^{5,8}; notably, one patient developed persistent, unhealed corneal epithelial defects.⁸ Although causality cannot be definitively established, these findings highlight the need for extreme caution when considering steroid therapy in this setting.

Similarly, the use of diluted topical antivenom is not recommended. We identified one study conducted in China in which diluted topical *Naja* antivenom was administered to manage severe ocular pain.⁵ However, this approach is inconsistent with the recommendations of the World Health Organization, as it has been associated with local ocular irritation and has not demonstrated additional clinical benefit.¹¹ Residual venom is more effectively eliminated through irrigation. Given the absence of documented systemic envenomation following isolated ocular exposure, intravenous antivenom is not indicated and unnecessarily exposes patients to risks such as anaphylaxis and other adverse reactions, without clear therapeutic advantage.

Conclusions

Snake venom ophthalmia resulting from the spitting of *Naja* species is an uncommon condition that typically presents as painful conjunctivitis accompanied by minimal to diffuse corneal

epithelial erosions. Prompt and thorough ocular irrigation, along with appropriate topical analgesics, vasoconstrictors, mydriatics, and antibiotics, is associated with improved clinical outcomes. In contrast, the use of topical or systemic antivenom and topical corticosteroids is contraindicated.

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