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## Acute corneal hydrops of presumed traumatic origin: An uncontrolled case series (three horses)

Louise M. O'Leary  
*Iowa State University*, [lolarey@iastate.edu](mailto:lolarey@iastate.edu)

Lionel Sebbag  
*Iowa State University*

L. M. Moody  
*University of Georgia and University of Tennessee*

K. A. Diehl  
*University of Georgia*

Rachel A. Allbaugh  
*Iowa State University*, [allbaugh@iastate.edu](mailto:allbaugh@iastate.edu)

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### Abstract

This case series describes acute corneal hydrops in three young horses. Due to similarities in the clinical appearance and progression of the disease with that which is reported in humans and cats with acute corneal hydrops, traumatic Descemet's membrane rupture was suspected to be the underlying aetiology in these equine cases. The horses presented with acute severe corneal oedema with intrastromal bullae formation and anterior bulging of the corneal contour. Focal posterior corneal changes were also seen in two of three cases. Mild anterior uveitis was also present. Other causes of corneal oedema (e.g. glaucoma) were ruled out based on presentation and clinical examination. Treatment approaches were medical and included various combinations of prophylactic topical antimicrobial therapy in case of secondary corneal ulceration, anti-inflammatory therapy for uveitis, targeted oedema therapy with topical hypertonic saline and corneal cross-linking, and placement of a temporary partial tarsorrhaphy for corneal tamponade. The outcome was excellent in all cases, with rapid resolution of the ocular changes. Acute corneal hydrops of presumed traumatic Descemet's membrane rupture origin should be considered in cases of young horses presenting with acute corneal oedema. However, further studies are warranted to better characterise the disease and to try to confirm the suspected aetiology.

### Keywords

horse, bullous keratopathy, keratoconus, corneal oedema, ocular trauma, Descemet's membrane rupture

### Disciplines

Large or Food Animal and Equine Medicine | Ophthalmology

### Comments

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## Case Report

**Acute corneal hydrops of presumed traumatic origin:  
An uncontrolled case series (three horses)**L. M. O'Leary<sup>†\*</sup> , L. Sebbag<sup>‡</sup>, L. M. Moody<sup>‡</sup>, K. A. Diehl<sup>‡</sup> and R. A. Allbaugh<sup>†</sup><sup>†</sup>Department of Veterinary Clinical Sciences, College of Veterinary Medicine, Iowa State University, Ames, Iowa,; and <sup>‡</sup>Department of Small Animal Medicine and Surgery, College of Veterinary Medicine, University of Georgia, Athens, Georgia, USA

\*Corresponding author email: loleary@iastate.edu

L. M. Moody's present address: Department of Small Animal Clinical Sciences, College of Veterinary Medicine, University of Tennessee, Knoxville, Tennessee, USA

**Keywords:** horse; bullous keratopathy; keratoconus; corneal oedema; ocular trauma; Descemet's membrane rupture**Summary**

**This case series describes acute corneal hydrops in three young horses. Due to similarities in the clinical appearance and progression of the disease with that which is reported in humans and cats with acute corneal hydrops, traumatic Descemet's membrane rupture was suspected to be the underlying aetiology in these equine cases. The horses presented with acute severe corneal oedema with intrastromal bullae formation and anterior bulging of the corneal contour. Focal posterior corneal changes were also seen in two of three cases. Mild anterior uveitis was also present. Other causes of corneal oedema (e.g. glaucoma) were ruled out based on presentation and clinical examination. Treatment approaches were medical and included various combinations of prophylactic topical antimicrobial therapy in case of secondary corneal ulceration, anti-inflammatory therapy for uveitis, targeted oedema therapy with topical hypertonic saline and corneal cross-linking, and placement of a temporary partial tarsorrhaphy for corneal tamponade. The outcome was excellent in all cases, with rapid resolution of the ocular changes. Acute corneal hydrops of presumed traumatic Descemet's membrane rupture origin should be considered in cases of young horses presenting with acute corneal oedema. However, further studies are warranted to better characterise the disease and to try to confirm the suspected aetiology.**

**Introduction**

Acute corneal hydrops is a severe manifestation of corneal oedema where rapid, large accumulation of fluid results in gross thickening of the cornea and bullae formation. The condition results in keratoconus, or anterior bulging of the cornea and can be vision- or globe-threatening if not managed appropriately. Acute corneal hydrops is reported in humans and cats and is caused by localised rupture of Descemet's membrane (Dubielzig *et al.* 2010; Fan Gaskin *et al.* 2013; Barsam *et al.* 2017; Schlesener *et al.* 2018), allowing ingress of aqueous humour with rapid and progressive corneal hydration. Risk factors have been suggested in humans (pre-existing ectatic corneal disease, trauma) (Grewal *et al.* 2000; Barsam *et al.* 2017) and cats (systemic cyclosporine administration) (Pierce *et al.* 2016), but

the disease is still poorly understood. Males and younger individuals tend to be predisposed in both species (Fan Gaskin *et al.* 2013; Pedersen *et al.* 2016). To the best of the authors' knowledge, acute corneal hydrops has not been formally described in the horse although its existence has been alluded to in veterinary textbooks without further characterisation (Dubielzig *et al.* 2010; Brooks *et al.* 2017a). The purpose of this case series is to describe the clinical findings, progression, treatment and outcome of three horses with acute corneal hydrops, presumed to be secondary to traumatic Descemet's membrane rupture.

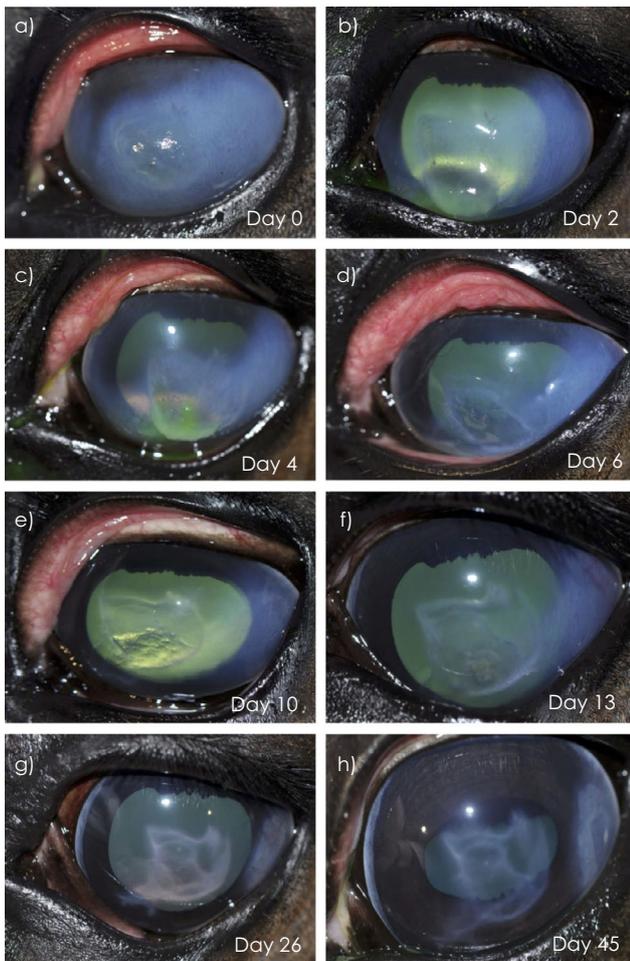
**Materials and methods**

A retrospective review of the medical records from two veterinary teaching hospitals was performed from January 2005 to July 2020. Search terms included 'corneal hydrops', 'bullous keratopathy', 'corneal oedema', and 'keratoconus'. Cases were excluded if clinical signs were present for more than 2 weeks prior to presentation, if records were incomplete, if infectious keratitis was present (based on corneal cellular infiltrates and cytological or culture results), or if other notable changes were present on ophthalmic examination or ocular histopathology (eg severe uveitis, glaucoma and retinal detachment). All cases were evaluated by a board-certified veterinary ophthalmologist. Data included age, breed, sex, presenting clinical signs, treatment and outcome. Nine cases met the inclusion criteria, but six of nine cases were excluded based on >1 aforementioned exclusion criteria.

**Case details****Case 1**

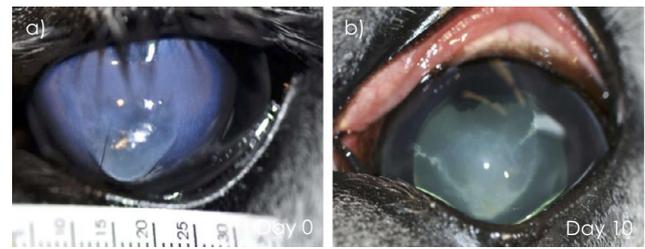
A 2.5-year-old Paint gelding presented with severe corneal oedema of *oculus sinister* (OS) noted by the owner following a reported ocular trauma several days prior at the trainer's facility for which no treatments were administered.

Abnormalities observed OS on presentation included mild blepharospasm, marked diffuse corneal oedema with large intrastromal bullae axially and keratoconus (**Fig 1a**), and mild anterior uveitis characterised by 1+ flare, miosis and decreased intraocular pressure (8 mmHg vs 22 mmHg in



**Fig 1:** Case 1. Clinical photographs of the affected OS showing the progression of corneal hydrops. **a)** At the initial examination, note the severe diffuse corneal oedema and large axial intrastromal bulla formation leading to keratoconus. **b)** At Day 2, the oedema has markedly improved but the bulla has become progressively more flaccid and migrated ventrally. **c)** By Day 4, the bulla had superficial fluorescein stain uptake. The corneal epithelium was debrided to allow corneal cross-linking (photograph was taken prior to cross-linking). **d)** The flaccid corneal bulla has been trimmed on Day 6, leaving behind an irregular topography corneal defect. **e)** Day 10, the corneal oedema has continued to improve with an axial irregularly-surfaced, ulcerated area of the cornea (where the flaccid cornea was trimmed) and residual lateral corneal oedema with mild perilimbal neovascularisation. **f)** Day 13, fibrosis of the corneal defect is seen as a grey haze outlining the area of previous corneal excision. The temporal oedema remains, and there is increased neovascularisation temporally. **g)** Day 26, fibrosis of the site of previous large intrastromal bulla formation/corneal excision has progressed and is moderate. The lateral oedema has receded further with a residual area of focal oedema, fibrosis and neovascularisation covering the site of peripheral iridal separation (iridodialysis). **h)** Day 45, the corneal fibrosis axially and temporal oedema are static to mildly improved.

oculus dexter (OD)). A small linear endothelial white opacity in the region of the large bullae was also noted OS. Fluorescein uptake was negative oculus uterque (OU).

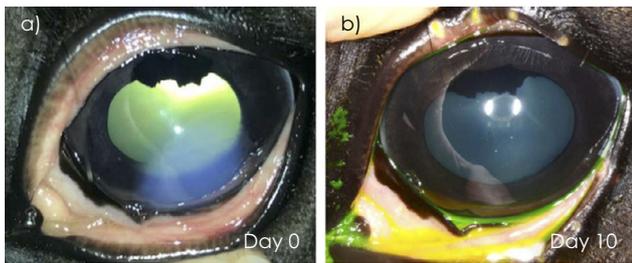


**Fig 2:** Case 2: Clinical photographs of the affected OS. **a)** At initial examination, note the severe corneal oedema and axial bullae with keratoconus. **b)** By Day 10, there has been marked improvement of the corneal oedema with resolution of the keratoconus and bullae. Mild oedema and fibrosis remain axially. The pupil is pharmacologically dilated.

The patient was hospitalised with a sub-palpebral lavage system. Medical therapy OS consisted of atropine sulfate<sup>1</sup> 1% q. 8 h, ofloxacin<sup>2</sup> 0.3% q. 6 h, diclofenac<sup>3</sup> 0.1% q. 6 h, 5% hypertonic saline (Muro 128)<sup>1</sup> q. 6 h and oral flunixin meglumine (Banamine)<sup>4</sup> 1.1 mg/kg q. 12 h. After 48 h of therapy, corneal oedema improved but the large bulla continued to progress (**Fig 1b**) and became associated with a superficial corneal ulcer another 48 h later. At that time, the corneal epithelium was debrided with sterile cotton-tipped applicators and modified accelerated corneal cross-linking was performed under sedation (45 mW/cm<sup>2</sup> for 2 min) (Famose 2014). The corneal hydrops continued to enlarge and cover the ventral cornea despite cross-linking, becoming progressively more flaccid in appearance (**Fig 1c**). Two days after cross-linking, the flaccid cornea contacting the lower eyelid was trimmed with Westcott scissors under topical anaesthesia and sedation (**Fig 1d**). Over the successive 10 days of hospitalisation, the corneal oedema and ulcer slowly resolved OS (**Fig 1e**), which revealed localised disinsertion of the iris base laterally (iridodialysis). The patient was discharged from the hospital with continued medical therapy consisting of a tapering course of flunixin meglumine per os, ofloxacin OS q. 8 h, serum OS q. 8 h, hypertonic saline OS q. 8 h and atropine OS q. 24 h. Recheck examination 2 weeks later showed axial superficial fibrosis and faint corneal oedema at the site of the corneal hydrops, as well as lateral perilimbal endothelial fibrosis, mild oedema and thin neovascularisation of the cornea associated with the site of iridodialysis (**Fig 1f**). The patient was discharged with a tapering course of topical prednisolone acetate<sup>5</sup> to reduce corneal fibrosis (**Fig 1g**), and no recurrence of disease was noted at the final recheck 6 weeks later (**Fig 1h**).

## Case 2

A 1-year-old Arabian filly presented with severe corneal oedema OS. The lesion was first noted 4 days prior to referral, was positive to fluorescein stain uptake, and progressed rapidly despite management with oral flunixin meglumine per os q. 12 h (unknown dose) and topical oxytetracycline/polymyxin B (Terramycin)<sup>6</sup> OS q. 12 h. Abnormalities observed on presentation included mild blepharospasm, marked diffuse corneal oedema with an 8 mm × 10 mm large intrastromal bulla axially and keratoconus OS (**Fig 2a**), as well as mild anterior uveitis characterised by trace flare and miosis. Intraocular pressures were 12 mmHg OS and 15 mmHg



**Fig 3: Case 3: Clinical photographs of the affected OS. a) At presentation with focal severe corneal oedema resulting in early keratoconus. The pupil has been pharmacologically dilated. b) At recheck 10 days later, note the resolution of the corneal oedema and keratoconus with very mild residual fibrosis.**

OD. No corneal infiltrates were seen. Fluorescein uptake was negative. Corneal cytology and aerobic bacterial culture were taken. The cytology revealed mild neutrophilic inflammation with no infectious organisms and the culture was negative, suggestive of a sterile process.

Though corneal sampling did not yield infectious organisms, the patient was hospitalised with a sub-palpebral lavage system, and aggressive antimicrobial therapy was initiated OS based on disease severity and inability to definitively rule out infection: ofloxacin q. 4 h, chloramphenicol<sup>7</sup> 0.5% ophthalmic solution q. 4 h, voriconazole<sup>7</sup> 1% ophthalmic solution q. 4 h, serum q. 4 h, atropine q. 12 h and flunixin meglumine 1.1 mg/kg per os q. 12 h. Improvement in corneal hydrops was rapid, with a notable decrease in the size of the large bulla to 5 mm × 7 mm by the next day. On Day 4 of hospitalisation, topical antimicrobials and serum were decreased in frequency to three times daily, and atropine and flunixin meglumine were tapered due to the continued marked improvement. Within 10 days of presentation, all medications were discontinued, the bulla had resolved and the residual oedema was minimal. A focal area of fibrosis was present where the bulla and hydrops had been present (Fig 2b). The patient was discharged without any medications, and no recurrence of disease was reported by the owner (phone update at one year).

### Case 3

A 1-year-old Warmblood colt presented with acute corneal oedema OS of less than 24 h duration. The eye was diagnosed with a corneal ulcer and corneal oedema by the referring veterinarian, who prescribed atropine q. 24 h OS and neomycin-polymyxin-bacitracin<sup>1</sup> OS q. 8 h and administered a single 1 mg/kg dose of intravenous flunixin meglumine. The patient was referred 12 h later due to significant lesion progression. Abnormalities observed on presentation OS included mild blepharospasm and marked focal corneal oedema with mild keratoconus (Fig 3a). The left pupil was pharmacologically dilated from atropine administration. No corneal infiltrates were seen. Pin-point fluorescein uptake was present over the corneal bullae. A small amount of fibrin was noted to be adherent to the corneal endothelium in the region of the bullae, and trace flare was present. The patient also had several small cuts and

abrasions to the forehead and jaw-line, along with a small lip laceration.

The horse was sedated, and a partial temporary tarsorrhaphy was placed with two interrupted horizontal mattress sutures 4-0 Nylon (Ethilon)<sup>8</sup> to provide tamponade to the corneal hydrops. Medical treatment was initiated OS with neomycin-polymyxin-bacitracin q. 12 h, atropine q. 48 h, hypertonic saline q. 4–6 h, voriconazole 2% ophthalmic ointment<sup>9</sup> q. 12 h and oral flunixin meglumine on a tapering schedule. At recheck one week later, the tarsorrhaphy sutures were removed revealing resolution of the corneal hydrops and fibrin. Minimal residual corneal oedema was present (Fig 3b). All medications were discontinued at that time, and no recurrence of disease was reported by the owner (phone update at 9 months).

### Discussion

The present case series highlights the clinical presentation, therapeutic plan and clinical outcomes of three young horses diagnosed with acute corneal hydrops of presumed traumatic origin. Early recognition and prompt management of this uncommon condition can result in excellent clinical outcomes, as described herein.

Several features suggest a traumatic aetiology with resulting Descemet's membrane rupture and development of acute corneal hydrops. First, each young horse was likely 'green', two of the three horses were in active training, and all three horses had their OS affected, consistent with handlers typically working with horses from their left side. An injury to the eye of Case 1 was specifically reported by the trainer, but no details were provided; however, the disinsertion of the lateral iris base in Case 1 is consistent with blunt trauma (Brooks *et al.* 2017b). Further, focal endothelial opacities were detected in two horses, consistent with traumatic Descemet's membrane rupture, suggested by a linear endothelial lesion (Brooks *et al.* 2017a, 2017b; Rodriguez Galarza and McMullen 2020) in Case 1 and fibrin adhered to the endothelial opacity in Case 3 (Ledbetter and Gilger 2013), with concomitant worse oedema compared to the surrounding cornea without endothelial changes.

Second, the severity, progressive and peracute nature of the oedema was consistent with Descemet's membrane rupture, as reported in human and feline cases of acute corneal hydrops (Grewal *et al.* 2000; Pederson *et al.* 2016). Although blunt trauma is not considered to play a role in feline acute corneal hydrops (Pederson *et al.* 2016), it is recognised as an inciting event for acute corneal hydrops in humans (Grewal *et al.* 2000; Fan Gaskin *et al.* 2013).

Third, other differentials for severe corneal oedema in horses (eg uveitis, glaucoma, endotheliitis, heterochromic iridocyclitis with secondary keratitis and corneal infection) (Pinto *et al.* 2015; Brooks *et al.* 2017a; Slenter *et al.* 2020) were ruled out based on the history, comprehensive ophthalmic examination and response to therapy in each case.

Taken together, rupture of Descemet's membrane is the most plausible explanation for the corneal hydrops in the cases described in this report, although this suspicion was not confirmed with advanced diagnostics. Optical coherence tomography and ultrasound biomicroscopy are two imaging modalities used to detect Descemet's membrane rupture in humans, cats and horses (Grewal *et al.* 2000; Matas Riera

*et al.* 2015; Schlesener *et al.* 2018; Rodriguez Galarza and McMullen 2020); unfortunately, such imaging modalities were not available to the authors at the time of each case management. Conventional ocular ultrasound can be considered for cases with large Descemet's membrane separations in horses (Matas Riera *et al.* 2015; Slenter *et al.* 2020), although this imaging tool may not be sensitive enough to detect a focal Descemet's membrane rupture. Attempts to confirm the suspicion of Descemet's membrane rupture using optical coherence tomography or ultrasound biomicroscopy are suggested in future cases of equine acute corneal hydrops.

Acute corneal hydrops requires rapid and targeted therapy to avoid vision- or globe-threatening complications such as corneal rupture, as described in human and feline patients (Grewal *et al.* 2000; Pederson *et al.* 2016). In fact, three of nine horses originally identified in the medical record search (excluded from further consideration for reasons as above) were enucleated due to painful corneal hydrops complications including recurrent corneal ulceration from bullae rupture and corneal perforation. It is likely that early and aggressive management of those cases would have achieved a positive outcome. In contrast, the three horses described in this case series had excellent outcomes despite diverse treatment approaches, utilising medical management ± corneal cross-linking or temporary tarsorrhaphy. Although corneal cross-linking was performed in Case 1, it did not appear to reverse the corneal changes or halt progression, possibly due to limited penetration of riboflavin and subsequent UV-A stimulus in the thickened oedematous equine cornea (Gallhoefer *et al.* 2016). Aggressive medical management was initiated with antimicrobials for Case 2 pending culture results; although bacterial and fungal cultures were negative, the possibility of previous infection could not be entirely ruled out given the use of broad-spectrum antibiotics prior to presentation and corneal sampling. Case 3 was treated with a temporary tarsorrhaphy to provide tamponade to the bulging cornea, similar to the most effective treatment reported in cats with acute corneal hydrops (Pederson *et al.* 2016). This patient's disease was mild in comparison to the other two cases described in this report, suspected to be due to the prompt referral (the patient presented within 24 h) and tamponade of the developing corneal hydrops. The condition resolved within 7 days in this patient. The authors advise clinicians to consider temporary tarsorrhaphy or a third eyelid flap (Pederson *et al.* 2016) as adjunctive tools in the management of acute corneal hydrops in horses, although additional cases and prospective case studies are required to confirm the potential benefits of these procedures. Other options such as corneal welding may be considered in nonresponsive or chronic cases (Rodriguez Galarza and McMullen 2020).

## Conclusions

Acute corneal hydrops is an uncommon condition affecting horses. Presumptive trauma causing Descemet's membrane rupture was suspected in the horses in this case series given the predilection for young horses and those in training. Clinical outcome is expected to be excellent with early recognition and therapeutic intervention. Although medical

management alone may suffice, the use of a temporary tarsorrhaphy to tamponade the corneal hydrops could accelerate healing. Additional work is required to better characterise this condition in horses and optimise clinical management.

## Authors' declaration of interests

No conflicts of interest have been declared.

## Ethical animal research

Case report therefore no ethical approval needed.

## Source of funding

Owner funded clinical cases. No funding provided.

## Authorship

L. O'Leary as the primary author contributed to the case collection, case series design, data analysis and preparation of the manuscript. K. Diehl, L. Sebbag and R. Allbaugh contributed to case management and assisted in study design and manuscript preparation. L. Moody contributed to case collection, data analysis, and assisted in study design and preparation of the manuscript. All authors gave the manuscript their final approval.

## Manufacturers' addresses

<sup>1</sup>Bausch and Lomb, Rochester, New York, USA.

<sup>2</sup>Hi-Tech Pharmacal, Amityville, New York, USA.

<sup>3</sup>Akorn Inc., Lake Forest, Illinois, USA.

<sup>4</sup>Schering-Plough Animal Health, Kenilworth, New Jersey, USA.

<sup>5</sup>Sandoz, Holzkirchen, Germany.

<sup>6</sup>Zoetis, Florham Park, New Jersey, USA.

<sup>7</sup>Compounded from Iowa State University College of Veterinary Medicine's pharmacy, Ames, Iowa, USA.

<sup>8</sup>Ethicon, Somerville, New Jersey, USA.

<sup>9</sup>Compounded from University of Georgia College of Veterinary Medicine's pharmacy, Athens, Georgia, USA.

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