Review Article

Equine recurrent uveitis: A review of clinical assessment and management

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Summary

Equine recurrent uveitis (ERU) is a vision-threatening ocular disease that practitioners must be able to identify and manage. Although not every case of acute uveitis will develop into ERU, if 2 or more episodes of uveitis are observed, a diagnosis of ERU can be made. Patient outcomes improve with early diagnosis, appropriate therapy and client education. Recent advances in surgical options and treatment of horses with ERU have improved success in managing this condition. New therapeutic strategies under investigation may further enhance results and reduce the development of complicating factors.

Introduction

The uveal tract is the vascular tissue in the eye, consisting of the iris, ciliary body and choroid. Inflammation of these structures is termed uveitis and is the most common cause worldwide of equine blindness (Schwink 1992; Gilger and Deeg 2011; Hollingsworth 2011). Causes of primary, acute uveitis include local and systemic infectious diseases, blunt or penetrating trauma, neoplasia and idiopathic or immune-mediated inflammation. The acute form of uveitis can affect any horse so it is important to distinguish primary, acute uveitis from the chronic, recurrent form and not assume that every case of uveitis in a horse is equine recurrent uveitis (ERU) as diagnostic testing, management and prognostic considerations differ (Gilger 2013).

Prevalence and aetiology

Equine recurrent uveitis, also known as moon blindness or periodic ophthalmia, has a prevalence of 2–25%, 8–10% and <1% in the United States (US), Europe and United Kingdom (UK), respectively. Differences are presumed to be due to genetic and environmental factors (Deeg *et al.* 2002a,b; Gilger and Deeg 2011; Mitchell 2014). It is characterised by episodes of persistent or recurrent intraocular inflammation that typically develop months after an initial uveitis episode subsides. A uveitis episode recurring within only a few weeks may be ERU, or may simply represent unresolved primary, acute uveitis if the underlying cause was not identified and managed appropriately, or if therapy was not continued for 2–4 weeks past the resolution of clinical signs.

The ERU condition is known to be a T-helper *type 1* immune-mediated disease that occurs when disruption of the blood-ocular barrier allows CD4+ T lymphocytes to enter the eye and their presence leads to repeated or persistent episodes of uveitis through recognition of self-antigens (Romeike *et al.* 1998; Gilger *et al.* 1999, 2008; Deeg *et al.* 2001, 2002a,b, 2006a,b, 2008; Deeg 2008; Regan *et al.* 2012; Malalana *et al.* 2015). Although leptospirosis is the most commonly implicated ERU initiator (Sillerud *et al.* 1987; Dwyer *et al.* 1995; Faber *et al.* 2000; Wollanke *et al.* 2001; Brandes *et al.* 2007) and numerous investigations have been performed to further elucidate its role in disease (Halliwell *et al.* 1985; Matthews *et al.* 1987; Brem *et al.* 1999; Rohrbach *et al.* 2005; Pearce *et al.* 2007; Gilger *et al.* 2008; Tömördy *et al.* 2010; Verma *et al.* 2010; Verma and Stevenson 2012; Polle *et al.* 2014), other infectious and noninfectious aetiologies may also trigger ERU including past ocular trauma (Gilger and Deeg 2011; Gerding and Gilger 2016) and Leptosporosis remains to be identified as a cause of ERU in the UK. Initiating or persisting infection alone does not sufficiently explain the clinical course or therapeutic response and recurrent episodes appear to result from epitope spreading (Deeg *et al.* 2006a; Deeg 2008). The primary uveitis cause, environmental influences and genetic make-up of the individual play a role in the development of this immune-mediated disease (Gilger and Deeg 2011).

Signalment and history

Appaloosas, Draught breeds, Warmbloods and European horses are predisposed to ERU; however, the disease could impact any breed with Quarter Horses and/or Thoroughbred lineage-breeds commonly affected due to population numbers. Genetic markers for ERU risk have been identified in Appaloosas and German Warmblood horses (Kulbrock *et al.* 2013a; Fritz *et al.* 2014). Coat colour within breeds is also a factor. The Leopard complex spotting locus is associated with insidious ERU in Appaloosas (Fritz *et al.* 2014) and brown or black Warmbloods are more often affected than those of other colours (Kulbrock *et al.* 2013b). No gender predilection has been proven, but 2 recent retrospective studies documented a significant overrepresentation of male horses (Kulbrock *et al.* 2013b; Gerding and Gilger 2016). The age of onset is variable, yet many cases present during mid-adult prime performance years (Dwyer *et al.* 1995; Gilger 2013; Gerding and Gilger 2016). Disease is unilateral in approximately 50% of horses, except for the Appaloosa breed with 80–87% bilateral ERU (Mitchell 2014; Gerding and Gilger 2016). Initial ocular signs such as intermittent squinting, tearing, or cloudiness may be noted by owners weeks, months, or years prior to eventual presentation due to persistent signs of ocular pain, a change in appearance to the eyes, or vision compromise.

Clinical appearance and classification

Clinical signs and findings are highly variable depending on ERU chronicity and clinical syndrome. The 3 stages of ERU are active, quiescent and end-stage. The active stage involves acute flare-ups of uveitis showing observable pain and blood-ocular barrier breakdown as evidenced by blepharospasm, epiphora, photophobia, corneal oedema, keratic precipitates, aqueous flare, hypopyon, iris hyperaemia, miosis, hypotony, vitreal cellular infiltrate and/or retinal inflammation and detachment (**Fig 1**). The quiescent stage occurs after an active flare-up resolves and may last for weeks, months or greater than a year. During this period, eyes appear comfortable and have no evidence of active uveitis. Indicators of previous inflammatory episodes such as posterior synechia, pigment rests on the anterior lens capsule (i.e. 'footprints of synechia'), iris hyperpigmentation, corpora nigra atrophy, lens subluxation, cataracts or inactive retinal lesions may be present (**Fig 2**). End-stage eyes are irreversibly blind and may manifest with phthisis bulbi, pupillary seclusion, blinding cataracts, luxated lenses and/or retinal detachments (**Figs 3–7** and **Supplementary Item 1**).

The disease is further classified by the clinical syndrome (classic, insidious, or posterior ERU) (Gilger and Deeg 2011; Gilger 2013). Classic ERU is the most common manifestation. It involves active observable inflammatory episodes followed by quiet periods, as described above and can occur in any horse. Recurrent episodes of classic ERU develop ocular changes that can lead to end-stage disease. Insidious ERU involves low-grade uveitis without apparent discomfort and is most common in Appaloosas and Draught breeds. Detailed veterinary examination is necessary to identify the subtle signs of this smouldering inflammation which may go unnoticed by owners until cataract, vision deficits or end-stage disease are present. Posterior ERU affects the vitreous and retina with little to no signs of anterior uveitis and most commonly affects Warmbloods, Draught breeds and European horses.

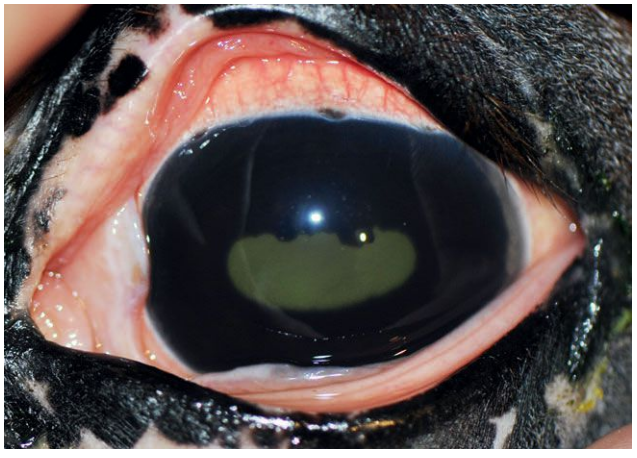

Fig 1: An active ERU episode presenting with ocular conjunctival and episcleral blood vessel congestion, mild diffuse corneal oedema and a relatively miotic pupil given the dim light examination setting. A mild degree of aqueous flare is also present that contributes to the generally hazy appearance of the eye. Reflections are present on the corneal surface that are artifactual and not associated with ocular disease.

Knowledge of ERU syndrome and stage classification impacts prognosis and treatment recommendations, as detailed below, but making an accurate diagnosis is imperative for appropriate therapy.

Examination and clinical aids

Ophthalmic examination should begin by assessing general eyelid and globe position, retroillumination, pupil size and symmetry, direct and consensual pupillary light reflexes and menace responses. If the menace response is not present a dazzle reflex can be performed in a dim light setting using a bright light source (e.g. LED light on most mobile phones) to assess for retina and optic nerve function. Intraocular pressure should ideally be documented with an applanation or rebound tonometer (**Fig 8**). Normal equine IOP is 15–30 mmHg, with uveitic eyes typically 5–12 mmHg and glaucomatous eyes above 35 mmHg (Miller *et al.* 1990; Knollinger *et al.* 2005). Sedation and periocular nerve blocks are used for examination of painful or uncooperative horses. Fluorescein staining is imperative to assess for corneal ulcers

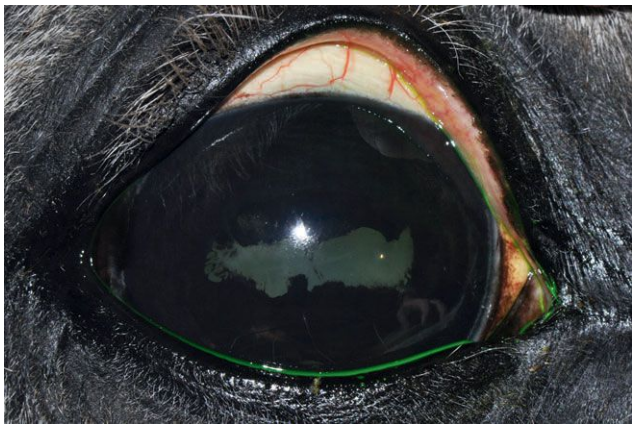

Fig 2: A comfortable quiescent ERU eye with posterior synechia causing dyscoria, degenerated corpora nigra and a diffusely hyperpigmented iris.

Fig 3: Chronic end-stage ERU in a phthisical eye with significant scleral show, a wrinkled fibrotic cornea, anterior synechia and an elevated third eyelid due to the shrunken globe.

Fig 4: Chronic end-stage ERU. The eye has a diffusely hyperpigmented iris, extensive posterior synechia and a complete cataract. The pupil aperture is greatly reduced by the adhesions and nearly secluded.

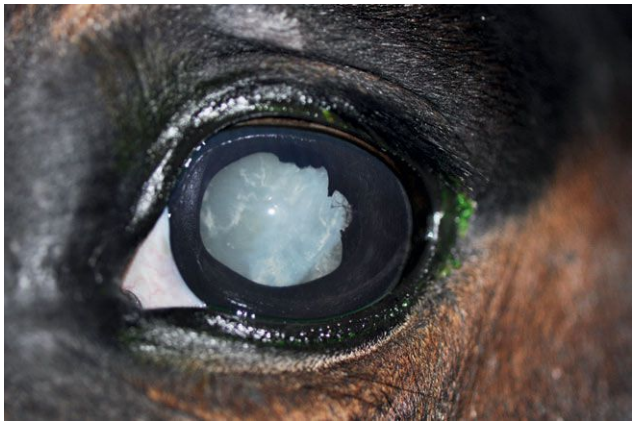

Fig 5: A horse presenting for cataract evaluation. The left eye had complete lens opacification, corpora nigra degeneration, lateral posterior synechia and diffuse iris hyperpigmentation. Recurrent bouts of previous inflammation were reported consistent with ERU and an ocular ultrasound revealed a retinal detachment.

and is especially warranted if topical steroid use will be initiated. Pupils should be dilated with tropicamide to allow for complete lens, vitreal and fundic examination. Active uveitis or posterior synechia may prevent full mydriasis; however, such impediments to dilation may reveal clinical findings supportive of ERU, reiterating the value of attempting pharmacological dilation.

A dark setting is necessary for identification of some clinical findings associated with ERU, such as aqueous flare, keratic precipitates, or faint cataracts. If an enclosed structure is not available for field examinations either work under a dense shade tree, place a large blanket over the heads of the horse and examiner, or schedule the examination at a dark time of the day. Aqueous flare is the hallmark of anterior uveitis. It is diagnosed by shining a bright focal light source (e.g. direct ophthalmoscope set on the smallest circle beam) into the eye held very close to the cornea (<1 cm away) while examining from a perpendicular angle. Aqueous flare is visualised as a beam of light

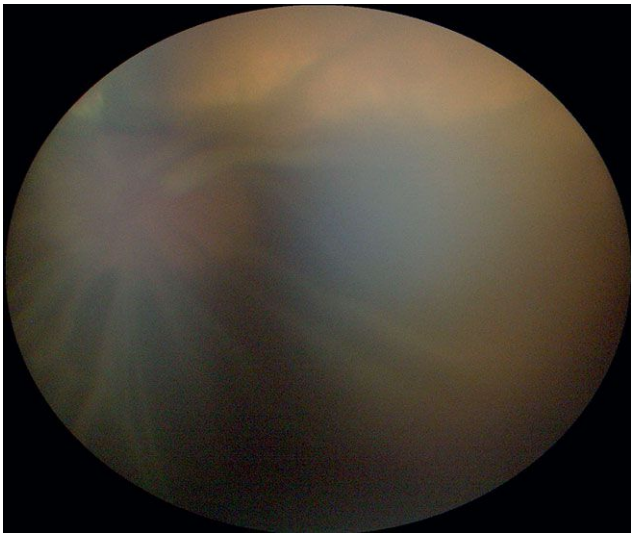

Fig 6: Fundic image showing the optic nerve on the left side of the picture with radiating white folds indicative of retinal detachment. The view is hazy due to unclear ocular media common with ERU. The eye was irreversibly blind but remained comfortable with medical management of the uveitis.

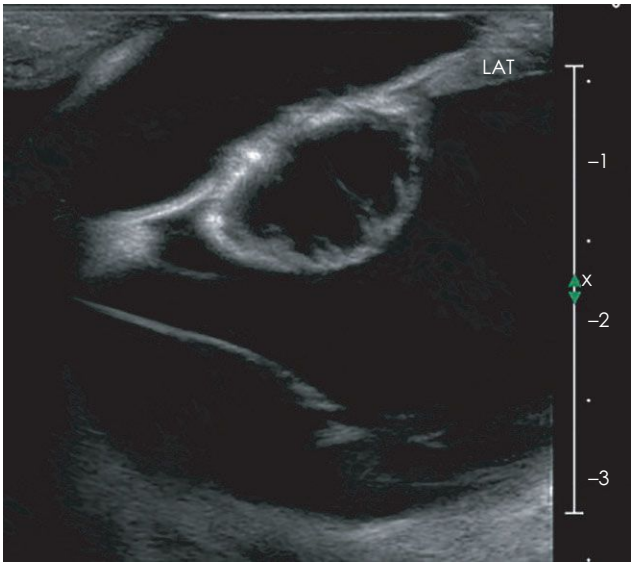

Fig 7: Ultrasound image of an eye with a detached retina due to ERU. The hyperechoic line visible in the vitreous is the neurosensory retina that has detached from the back of the eye but remains attached centrally at the optic nerve and peripherally at the ora.

continuing through the anterior chamber to connect the cornea and lens and is graded based on intensity (Hogan *et al.* 1959). This space should be clear in normal eyes and the technique can be practiced in healthy horses. Keratic precipitates may be found on the ventral corneal endothelium and can be viewed with direct illumination using magnification. Small cataracts associated with ERU are commonly located in the lens cortex. They may be associated with posterior synechia or pigment rests on the lens surface and will be more easily visualised after

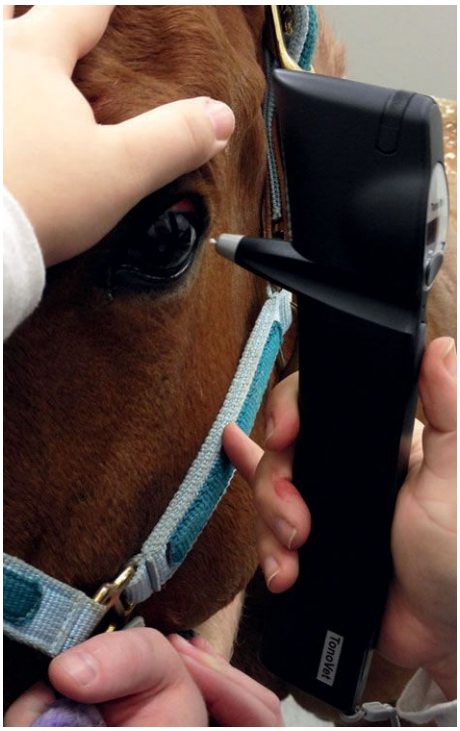

Fig 8: Intraocular pressure measurement using a TonoVet rebound tonometer.[1] The eyelid is gently held open with pressure on the orbital rim instead of the eyeball to avoid artifactual elevation of intraocular pressure and the tonometer probe oriented perpendicular to the ocular surface. The normal value for a horse is 15–30 mmHg.

pharmacological dilation. Anterior subcapsular lens opacities are noted with predominantly anterior uveitis, while primarily posterior changes are seen with posterior ERU and both locations may be affected with panuveitis. Vitreal inflammation (hyalitis) manifests as vitreal cellular infiltrate, vitreal debris and/or a yellow/green hue to the vitreous (**Fig 9**). Fundic evaluation may reveal active chorioretinitis lesions characterised by hazy retinal oedema, grey/white cellular infiltrates, haemorrhage, or retinal detachment. Inactive peripapillary ('butterfly') or multifocal chorioretinal ('bullet-hole') lesions present in eyes with other clinical findings of ERU may be associated with historic disease; however, depigmented punctate lesions or peripapillary scarring may also be considered incidental findings in the absence of other signs and are not pathognomonic for ERU (Matthews *et al.* 1990; Mathes *et al.* 2012).

Retroillumination is the technique of aligning an examiner's gaze with a light shone into the eyes of the patient from approximately 3 feet away to result in a fundic reflex. This can be done prior to dilation to help assess pupil size, symmetry and general optical axis clarity but should also be done after dilation to help identify keratic precipitates, cataracts, vitreal opacities, or other subtle imperfections which may be more easily highlighted. Eyes with a fundic reflex visible on retroillumination but lacking a menace response, PLR, or dazzle reflex likely have irreversible retinal or optic nerve damage. Detachment of the retina, whether diagnosed on ophthalmic examination or ocular ultrasound, is a grave prognosis for vision (see **Figs 6**, **7** and **Supplementary Item 1**) (Strobel *et al.* 2007).

Fig 9: The vitreous has a yellow-green appearance in this horse with uveitis as a result of serum pigments in the eye. Diffuse iris hyperpigmentation, corpora nigra atrophy dorsally, posterior synechia ventrally, dark pigment deposits on the ventral paracentral lens capsule and faint multifocal lens opacities are also present.

Phthisis bulbi is shrinking of the globe seen secondary to severe or prolonged uveitis and may be evidenced by prominent scleral show, corneal wrinkling, diffuse fibrosis and a lack of PLR or dazzle reflexes (see **Fig 3**). These changes are consistent with end-stage disease that may have occurred due to any form of ERU.

Differential diagnoses

Nonspecific signs of ocular pain include blepharospasm, epiphora and photophobia. These are symptoms clients may recognise with classic ERU, but they could also be due to conjunctival foreign bodies, corneal ulcers, or corneal stromal abscesses. Evaluation of the ocular surface during an examination can exclude these differential diagnoses. Clients should be instructed to seek immediate veterinary evaluation and not apply a topical corticosteroid if signs of ocular pain occur as it could exacerbate a newly formed corneal ulcer (**Fig 10**). When diffuse corneal oedema is present in a painful eye with or without corneal vascularisation, a cream-coloured corneal stromal abscess may be present and corticosteroids should not be administered. Diffuse corneal oedema associated with elevated IOP is diagnostic for glaucoma and commonly associated with ERU in horses, but requires additional management (Wilkie 2010; Utter and Brooks 2011; Annear *et al.* 2012).

Diagnostic testing

Fluorescein staining should be performed on every horse presenting with ocular complaints. Horses with ERU that are stain negative with no other signs of primary corneal disease can be treated with standard medical therapy as detailed below. Tonometry is useful to aid diagnosis and monitoring of ERU or secondary glaucoma. A physical examination should be performed to identify signs of general illness and ensure there are no contraindications with planned medical treatment.

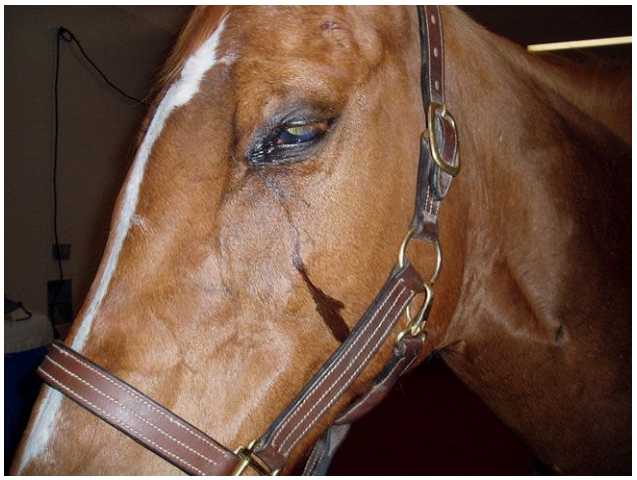

Fig 10: Epiphora and blepharospasm are present in this horse with historic ERU episodes. The client started empiric treatment 4 days prior with a topical steroid but signs did not resolve and a fungal corneal ulcer was diagnosed on veterinary examination as the cause of the ocular pain symptoms. This reiterates the importance of veterinary evaluation with the onset of clinical signs as corticosteroid use may be contraindicated.

Additional diagnostic tests to consider include a complete blood count (CBC), serum chemistry profile and testing for specific aetiological agents. The CBC and serum chemistry profile may help to identify concurrent or associated systemic disease or more commonly serve as a normal baseline prior to initiating therapy. Notably, plasma fibrinogen and serum amyloid A are not useful biomarkers for uveitis (Labelle *et al.* 2011). Leptospirosis serology can be performed on serum and ocular fluids by microscopic agglutination test (MAT). The most commonly tested serovars include *L. bratislava, L. canicola, L. grippotyphosa, L. hardjo, L. icterohaemorrhagiae* and *L. pomona*. Titres of 1:400 or greater are considered positive, but the value of a single positive serological specimen is limited so paired sera samples 2–3 weeks apart are preferred with a 4-fold rise being diagnostic (Verma *et al.* 2013). If both serum and aqueous humour or vitreous are tested, the ratio of ocular to serum antibody concentrations can be calculated (C value) for each serovar and a value greater than 4 is indicative of intraocular Leptospira antibody production (Gilger *et al.* 2008). Ocular fluids may also be tested with real-time PCR to detect the presence of leptospirosis DNA. Leptospira culture is not recommended for clinical cases due to the lack of sensitivity and length of culture (Polle *et al.* 2014). If an eye must be removed due to chronic pain and blindness, ocular histopathology can be performed to look for pathognomonic ERU lesions; however, aetiological agents are rarely identified (Cooley *et al.* 1990; Dubielzig *et al.* 1997).

Medical therapy

Active uveitis initially warrants aggressive treatment, with therapy slowly tapered and discontinued 2–4 weeks past the resolution of signs as confirmed by ophthalmic recheck examinations (**Fig 11**). The goals of therapy are to reduce pain and preserve vision. The most critical component of treatment is anti-inflammatory therapy. Topical prednisolone acetate 1% or ophthalmic dexamethasone 0.1% (as neomycin/polymyxin B/dexamethasone) should be given 4 times daily or even more frequently if needed to control the uveitis. Ophthalmic hydrocortisone and sodium phosphate steroid preparations are less effective due to poor intraocular penetration. Subconjunctival triamcinolone, which has effects over 2–3 weeks, is not effective as monotherapy and may predispose to bacterial infection or melting if a corneal ulcer develops. Concurrent topical nonsteroidal anti-inflammatory (NSAID) therapy with flurbiprofen 0.03%, diclofenac 0.1%, or bromfenac 0.09% 3–4 times daily may be needed in severe cases or can be utilised if corticosteroid therapy is contraindicated by corneal ulcers or calcific band keratopathy. Flunixin meglumine appears to be the most effective systemic NSAID (Gilger and Michau 2004) with initial treatment recommended at 1.1 mg/kg bwt twice daily for 3 days then once daily for one week or longer. Injectable 5% solution is inexpensive and may be given by mouth with good absorption and bioavailability (Pellegrini-Masini *et al.* 2004). An oral dosing syringe can be coated with a palatable substance to help mitigate the bitter taste of the injectable solution. Other systemic anti-inflammatories such as phenylbutazone, firocoxib, aspirin, prednisolone, or dexamethasone are either less effective or have greater risk of side effects, with systemic steroids reserved for severe, nonresponsive cases (Gilger and Michau 2004). Topical ophthalmic atropine 1% should be given once to twice daily to dilate the pupil (reducing the risk of blinding synechiae), alleviate ciliary muscle spasm and help stabilise the blood-aqueous barrier. Severe cases may require atropine treatment up to 4 times daily, but clients should closely monitor for inappetence, lethargy, reduced faecal output, or more profound signs of colic as ileus is an idiosyncratic reaction in some horses (Williams *et al.* 2000). Horses with frequently recurring ERU may benefit from long-term maintenance using a topical NSAID daily and/or topical atropine weekly.

If an active leptospiral infection is suspected, systemic antibiotics (doxycycline hyclate 10 mg/kg bwt *per os* twice daily for 4 weeks, minocycline hydrochloride 4 mg/kg bwt *per os* once daily for 4 weeks, or enrofloxacin 7.5 mg/kg bwt *per os* once daily for 3 weeks) may also be administered, but ocular penetration and efficacy is questionable (Gilmour *et al.* 2005; Divers *et al.* 2008; Popp *et al.* 2013). A recent study reported that systemic antibiotic therapy had minimal effect on visual outcome in horses with leptospirosis-associated ERU as a positive titre may not indicate active infection but rather prior exposure (Gerding and Gilger 2016). Vaccination against leptospirosis as adjunctive treatment for ERU is not supported at this time (Rohrbach *et al.* 2005).

Intravitreal injections and surgical treatment

Severe or frequently recurring ERU cases may require treatment by intravitreal injections, vitrectomy, or suprachoroidal cyclosporine implantation. The choice of advanced treatment will depend on the accessible options and ERU clinical syndrome.

Intravitreal injection is an inexpensive technique that can be performed with standing sedation and appropriate sterile preparation. Low dose gentamicin (4 mg intravitreally) has been previously reported (Gilger and Michau 2004) and a recent preliminary study showed promising results for use in

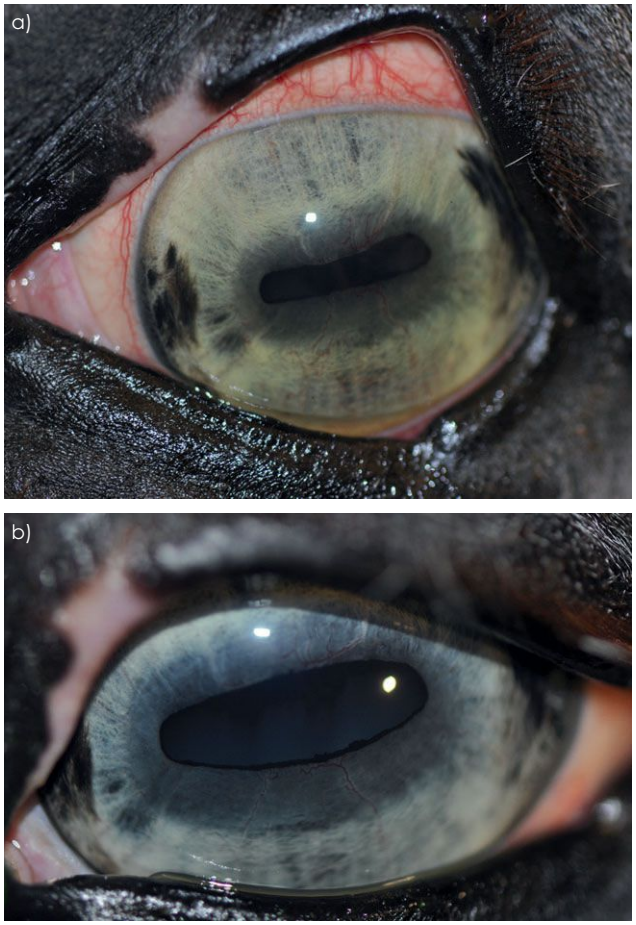

Fig 11: a) Active ERU episode in a blue-eyed horse with episcleral and conjunctival blood vessel injection, miosis, yellowing of the previously blue iris and engorged iris blood vessels ventrally and near the pupil margin dorsally. b) Same eye as shown in (a) one week following treatment with uveitis controlled and the iris a normal blue colour (dark areas medial and lateral are normal heterochromia irides colouration). Iris blood vessels remain visible and are likely within a preiridal fibrovascular membrane. Medications were gradually tapered over subsequent weeks following confirmation of inflammation control.

chronic or persistent posterior uveitis cases with notable clinical improvement within 1–3 days (McMullen 2015). Intravitreal dexamethasone (1–2 mg) or triamcinolone acetonide (5–10 mg) may also be considered; however, they may predispose to microbial infection (Yi *et al.* 2008; Gerding and Gilger 2016). Rapamycin (10 mg/eye) appears to be safe, but clinical studies are needed to establish efficacy in ERU (Douglas *et al.* 2008).

Pars plana vitrectomy, although of limited benefit in North America, is commonly performed in parts of mainland Europe, notably Germany, for horses with posterior uveitis to clear the visual axis of vitreal opacities and remove inflammatory mediators with 85% maintenance of vision reported (Frühauf *et al.* 1998). It is most beneficial for Leptospira-associated ERU and may not be recommended for horses testing negative for *L. interrogans* antibodies given significantly less clinical benefit (Tömördy *et al.* 2010). Despite success in relieving pain and preventing ERU recurrence, eyes

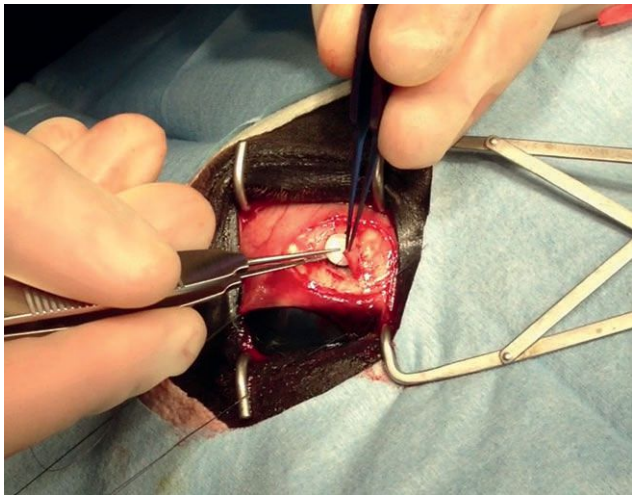

Fig 12: Surgical suprachoroidal cyclosporine implant placement dorsolaterally underneath a scleral flap in this left eye to reduce the frequency and severity of ERU episodes.

undergoing vitrectomy may become blind due to retinal detachment, or progressive cataract (Frühauf *et al.* 1998; von Borstel *et al.* 2005).

Given that ERU is a T cell mediated disease, the immunosuppressive drug cyclosporine has been investigated. Although topical therapy cannot penetrate the ocular surface, suprachoroidal cyclosporine implant placement allows achievable intraocular drug levels for over 3 years (Gilger *et al.* 2006). The implant is placed deep within the sclera in contact with the choroid (**Fig 12** and **Supplementary Item 2**). Horses that can be controlled with standard medical therapy, but have frequent recurrences or early relapse of active ERU after being gradually tapered off medications are considered ideal candidates (Gilger *et al.* 2010). In a review of 151 operated eyes, frequency of uveitic episodes was dramatically reduced and vision maintained in 70% of eyes 5 or more years after surgery (Gilger *et al.* 2010). Although surgery appears to reduce vision loss by approximately one half in all ERU horses, Appaloosa horses especially benefited as vision loss was only 12% in eyes that received the implant (Gilger *et al.* 2010).

Ocular diseases secondary to ERU

Eyes that present with additional changes secondary to ERU may need supplemental therapy or referral to a specialist.

Calcific band keratopathy

Calcific band keratopathy manifests as grey to white mineral depositions in the superficial cornea along the interpalpebral central region and may include fluorescein positive corneal ulcers (Rebhun *et al.* 1993; Brooks 2012). Topical steroid use has been associated with its development in both man and horses (Rebhun *et al.* 1993; Taravella *et al.* 1994; Schlötzer-Schrehardt *et al.* 1999; Brooks 2012). Treatment includes cessation of corticosteroid therapy and the addition of a topical calcium chelator such as dipotassium ethylenediaminetetraacetic acid (EDTA 1% ointment 3 times daily) to decrease tear film and tissue free calcium levels (Brooks 2008). Chelation with EDTA 3.75% following epithelial debridement may cause calcium dissolution (Najjar *et al.*

2004). When corneal ulcers occur due to the mineralisation, topical antibiotics, atropine and a systemic NSAID are indicated. In rare cases, diamond burr debridement or superficial keratectomy may be considered to facilitate healing (Rebhun *et al.* 1993; Brooks 2012; Lassaline-Utter *et al.* 2014). Topical NSAID and EDTA therapy should be prescribed for ERU-affected horses developing early signs of calcific band keratopathy while monitoring for worsening of mineralisation and ERU.

Cataracts

Small cataracts that develop with ERU may not impact vision but should be monitored for progression. Eyes with complete cataracts that prevent posterior segment examination may have ocular ultrasonography performed to assess the vitreous and retina. The presence of hyperechoic vitreal debris suggests hyalitis, while a hyperechoic line extending from the ora ciliaris retina to the optic nerve is diagnostic for retinal detachment (See **Fig 7** and **Supplementary Item 1**). It has been previously stated that horses with complete cataracts due to ERU are not good candidates for cataract surgery due to pre-existent inflammatory changes. A recent study confirmed ERU eyes that underwent cataract surgery were significantly less likely to be visual (25%) than horses without preoperative ERU (65%) at the last post operative follow-up (Edelmann *et al.* 2014). However, another report showed no difference in ERU and non-ERU eyes following cataract surgery with a similar visual outcome of 37% greater than 2 years post operatively (Brooks *et al.* 2014). Individual horse ocular status and surgeon preference will dictate if cataract surgery is an option in a patient with historic ERU.

Glaucoma

Horses with uveitis are at risk for glaucoma and ERU is the most common cause of equine glaucoma (Annear *et al.* 2012; Curto *et al.* 2014). Clinical examination of the equine iridocorneal angle is possible medially and laterally with some ERU eyes showing scarring or fibrosis that may predispose to glaucoma (**Fig 13**). Horses with secondary glaucoma (high IOP or inappropriately high-normal IOP during active uveitis) can be medically treated. The most common medical treatments include topical dorzolamide 2%, brinzolamide 1%, timolol 0.5%, or the combination dorzolamide 2% and timolol 0.5% product 2–3 times daily (van der Woerdt *et al.* 2000; Willis *et al.* 2001; Germann *et al.* 2008; Tofflemire *et al.* 2014). Although atropine stabilises the blood-aqueous barrier and may reduce IOP in some horses, its use is not recommended in equine glaucoma unless daily IOP monitoring is possible due to the risk of compromising aqueous humour drainage (Herring *et al.* 2000; Utter and Brooks 2011). Semiconductor diode laser transcleral cyclophotocoagulation may be used to help reduce IOP in affected eyes and maintain vision, but does not eliminate the need for medications (Annear *et al.* 2010). Chemical ciliary body ablation can be performed by intravitreal injection in blind eyes afflicted with glaucoma to reduce pressure using gentamicin (25–50 mg) and dexamethasone (1 mg) (König *et al.* 2003; Utter and Brooks 2011). Enucleation, evisceration, or other cosmetic globe surgeries could also be considered. A recent retrospective study reported that ERU horses with elevated IOP at initial presentation were at greater risk for blindness and/or enucleation than other horses despite attempted glaucoma therapy (Gerding and Gilger 2016).

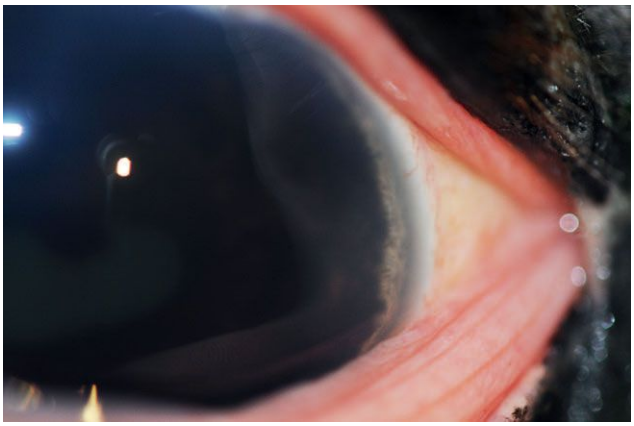

Fig 13: The iridocorneal angle or 'grey line' visible near the lateral limbus has become more fibrotic in appearance due to scarring of the trabecular meshwork. This change can be seen with ERU and is not pathognomic for glaucoma but does warrant intraocular pressure monitoring.

Prognosis and impact

Equine recurrent uveitis has a poor long-term prognosis; however, chances of retaining vision can be improved with prompt diagnosis of inflammatory episodes, appropriate treatment and diligent monitoring. Appaloosas have the poorest prognosis for vision when compared with other breeds and an 8 times greater predilection for ERU (Dwyer *et al.* 1995; Fritz *et al.* 2014), but suprachoroidal cyclosporine implant placement is especially beneficial in managing ERU in this breed (Gilger *et al.* 2010).

A recent retrospective study of 224 ERU horses (338 eyes) documented rates of vision loss (46.9% of eyes, including 28.4% of eyes on initial examination), globe loss (12.1%), decreased performance (60.8%), transfer of ownership (19.1%), or even euthanasia (14.9%) (Gerding and Gilger 2016). The financial impact of diagnosis and treatment ranged between $4000 and $8000 USD for most horses and nearly 85% of horse owners felt the value of their horse decreased as a result of ERU (Gerding and Gilger 2016). Although these numbers may be biased by the population being referred and a greater proportion of first line cases may be more effectively managed by veterinarians, the overall impact on individual horses, their owners and the equine industry as a whole is significant.

Client education

Clients should be informed about the potential for ERU whenever a horse presents with uveitis since inciting causes may be numerous; however, ERU should not be diagnosed until 2 or more episodes of uveitis are documented. Clients should also be told to contact a veterinarian for evaluation if ocular signs recur and should be cautioned against empiric treatment given the possibility of corneal ulceration, stromal abscessation, foreign body, or another condition presenting similarly. General health status should be monitored and a veterinarian contacted immediately if issues develop while receiving medical treatment for ERU as colic, colitis, gastrointestinal ulceration and other problems may be associated with some treatments.

Horses with ERU may have or develop visual deficits which owners should be informed of to allow for safe interactions and use. Breeding horses with ERU is not recommended given the potential genetic influences (Kulbrock *et al.* 2013a,b; Fritz *et al.* 2014). Genetic selection of individuals free of disease and not possessing risk markers may reduce the prevalence of ERU in the future.

Conclusion

Equine recurrent uveitis is the leading cause of vision loss in horses. Accurate diagnosis, appropriate treatment and close monitoring will help to improve outcomes and maintain vision. When ERU persists or worsens despite seemingly appropriate therapy, recurs frequently, or is simply beyond one's capability for treatment, referral should be offered as an intravitreal injection or surgical treatment may be warranted. Research is ongoing to determine more specific information about the pathogenesis of ERU and new therapeutic strategies are under investigation to aid clinical management.

Author's declaration of interests

No conflicts of interest have been declared.

Ethical animal research

Ethical review not applicable for this review article.

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Manufacturer's address

[1]Icare, Helsinki, Finland.

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Supporting information

Additional Supporting Information may be found in the online version of this article at the publisher's website:

Supplementary Item 1: Ultrasound clip of an eye with a detached retina due to ERU. The hyperechoic line visible in the vitreous is the neurosensory retina that has detached from the back of the eye but remains attached centrally at the optic nerve and peripherally at the ora.

Supplementary Item 2: Surgical suprachoroidal cyclosporine implant placement underneath a scleral flap to reduce the frequency and severity of ERU episodes. Abbreviated steps shown include scleral incision and dissection, implant placement, scleral closure and conjunctival closure. The horse is under general anesthesia in right lateral recumbency with the nose beyond the lower right-hand aspect of the video screen. The surgeon is sitting dorsal to the left eye and operating in the dorsolateral ocular quadrant.

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