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Equine corneal ulcers: medical treatment

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Medical therapy for equine corneal ulcers

Once a corneal ulcer is diagnosed, the therapy must be carefully considered to ensure comprehensive treatment. Medical therapy almost always comprises the initial major thrust in ulcer control, albeit tempered by judicious use of adjunctive surgical procedures. This intensive pharmacological attack should be modified according to its efficacy. Subpalpebral or nasolacrimal lavage treatment systems are employed to treat a fractious horse or one with a painful eye that needs frequent therapy.

The clarity of the cornea, the depth and size of the ulcer, the degree of corneal vascularization, the amount of tearing, the pupil size, and intensity of the anterior uveitis should be monitored. Serial fluorescein staining of the ulcer is indicated to assess healing. As the cornea heals the stimulus for the uveitis will diminish, and the pupil will dilate with minimal atropine therapy. Self-trauma should be reduced with hard or soft cup hoods.

Antibiotics

Bacterial and fungal growth must be halted and the microbes rendered non-viable. Broad-spectrum topical antibiotics are usually administered with culture and sensitivity tests aiding selection. Topical antibiotic solutions interfere with corneal epithelial healing less than ointments. Gentamicin should be used in ulcers with evidence of stromal melting only.

Topically applied antibiotics, such as bacitracin-neomycin-polymyxin B, gentamicin, ciprofloxacin, or tobramycin ophthalmic solutions may be utilized to treat bacterial ulcers. Frequency of medication varies from q2h to q8h. Cefazolin (55mg/ml), bacitracin, and carbenicillin are effective against beta hemolytic Streptococcus. Ciloxan (ciprofloxacin), amikacin (10 mg/ml), and polymyxin B (0.25% IV solution) may be used topically for gentamicin resistant Pseudomonas.

Collagenolysis prevention

Severe corneal inflammation secondary to bacterial (especially, Pseudomonas and beta hemolytic Streptococcus) or, much less commonly, fungal infection may result in sudden, rapid corneal liquefaction and perforation. Activation and/or production of proteolytic enzymes by corneal epithelial cells, leucocytes and microbial organisms are responsible for stromal collagenolysis or “melting”. Serum is biologically nontoxic and contains an alpha-2 macroglobulin with antiproteinase activity. Autogenous serum administered topically can reduce tear film and corneal protease activity in corneal ulcers in horses. The serum can be administered topically as often as possible, and should be replaced by new serum every five days. Five to 10 per cent acetylcysteine, and/or 0.05% sodium EDTA can be instilled hourly, in addition to the other indicated drugs, for antimelting effect until stromal liquefaction ceases. It may be necessary to use serum, EDTA, and acetylcysteine simultaneously in severe cases. Subconjunctival tetanus antitoxin contains macroglobulins with anticollagenase effects and can also slow corneal melting.
Treat the uveitis
Atropine sulfate is a common therapeutic agent for equine eye problems. Topically applied atropine (1%) is effective in stabilizing the blood-aqueous barrier, reducing vascular protein leakage, minimizing pain from ciliary muscle spasm, and reducing the chance of synchiae formation by causing pupillary dilatation. Atropine may be utilized topically q4h to q6h with the frequency of administration reduced as soon as the pupil dilates. Topical atropine has been shown to prolong intestinal transit time, reduce and abolish intestinal sounds, and diminish the normal myoelectric patterns in the small intestine and large colon of horses. Some horses appear more sensitive than others to these atropine effects, and may "respond" by displaying signs of colic and/or prolonged intestinal transit time. Horses receiving topically administered atropine should be monitored for signs of colic. Systemically administered NSAIDs such as phenylbutazone (1 gm BID PO) or flunixin meglumine (1 mg/kg BID, IV, IM or PO) can be used orally or parenterally, and are effective in reducing uveal exudation and relieving ocular discomfort from the anterior uveitis in horses with ulcers. Topical nonsteroidal antiinflammatory drugs (NSAIDs) such as profenol, flurbiprofen and diclofenamic acid (BID to TID) can also reduce the degree of uveitis. Horses with corneal ulcers and secondary uveitis should be stall-rested till the condition is healed. Intraocular hemorrhage and increased severity of uveitis are sequelae to overexertion.

Inappropriate therapy and ulcers
Topical corticosteroids may encourage growth of bacterial and fungal opportunists by interfering with non-specific inflammatory reactions and cellular immunity. Corticosteroid therapy by all routes is contraindicated in the management of corneal infections. Even topical corticosteroid instillation, to reduce the size of a corneal scar, may be disastrous if organisms remain indolent in the corneal stroma.

Fungal ulcers in horses
Fungi are normal inhabitants of the equine environment and conjunctival microflora, but can become pathogenic following corneal injury. Aspergillus, Fusarium, Cylindrocarpon, Curvularia, Penicillium, Cystodendron, yeasts, and molds are known causes of fungal ulceration in horses. Ulcerative keratomycosis is a serious, sight-threatening disease in the horse. Blindness can occur.

The most often proposed pathogenesis of ulcerative fungal keratitis in horses begins with slight to severe corneal trauma resulting in an epithelial defect, colonization of the defect by fungi normally present on the cornea, and subsequent stromal invasion. Seeding of fungi from a foreign body of plant origin is also possible. Some fungi may have the ability to invade the corneal epithelium following disruption of the tear film. Stromal destruction results from the release of proteinases and other enzymes from the fungi, tear film leukocytes and keratocytes. Fungi may produce antiangiogenic compounds that inhibit vascularization. Fungi appear to have an affinity for Descemet's membrane with hyphae frequently found deep in the equine cornea. Deeper corneal invasion can lead to sterile or infectious endophthalmitis. Saddlebreds appear to be prone to severe keratomycosis, while standardbreds are resistant.

Diagnostic tests should include fluorescein and rose bengal staining, corneal cytology, corneal culture with attempted growth on both fungal and aerobic plates, and biopsy if surgery is performed. Prompt diagnosis and aggressive medical therapy with topically administered antifungals, antibiotics and atropine, and systemically administered NSAIDs will positively influence visual outcome, and may negate the need for surgical treatment. Treatment must be directed against the fungi as well as against the iridocyclitis that occurs following fungal replication and fungal death. Therapy is quite prolonged and scarring of the cornea may be prominent. The fungi are overall more susceptible to antifungal drugs in this order: natamycin = miconazole > itraconazole > ketoconazole > fluconazole. Natamycin, miconazole, itraconazole/ DMSO, fluconazole, amphotericin B, betadine solution, chlorhexidine gluconate, posaconazole, voriconazole, and silver sulfadiazine can be utilized topically. Uveitis may be worse the day following initiation of antifungal therapy due to fungal death. Systemically administered itraconazole or fluconazole may be useful in recalcitrant cases.