Corneal Ulcers

Corneal ulcers or ulcerative keratitis involve disruption of the surface layer of the eye. In horses, corneal ulcers can deteriorate very quickly and possibly lead to blindness. Therefore, whenever tearing, swelling, and squinting are present in your horse, you should call your veterinarian.

The equine cornea is very thin (1mm) and consists of the tear film layer, epithelium (top cell layer), stroma, and endothelium (bottom cell layer, also known as Descemet’s membrane). The epithelium and the tear film serve as outer barriers to normal and pathogenic bacteria. In addition to this protective effect, the tear film lubricates, provides nutrients, enhances corneal defenses, and flushes away toxins and waste products. The tear film is comprised of three layers.

The lipid outer layer reduces evaporation and helps provide an even distribution of the tear layer. The middle layer is the aqueous layer. The inner mucin layer is essentially a gel that binds the aqueous layer to the corneal epithelial cells. The stroma is structurally arranged in a lamellar pattern that allows light to pass through it without scattering. The endothelium supports the stroma and prevents fluid from the inside of the eye from disrupting the stromal lamellar pattern. The cornea is innervated by nerve branches that enter mid stroma at the periphery and then become superficial as they head to the center of the eye. This superficial course/pathway of the nerves explains the intense pain associated with corneal disease.

In equine ulcerative keratitis (corneal ulcers), disruption of the tear film, epithelium, stroma, and sometimes endothelium occur. Clinical signs of corneal ulcers include squinting (blepharospasm), swelling (blepharoeDea), tearing (epiphora), redness (conjunctival hyperemia), hazy cornea (corneal Please see Corneal Ulcers: page 2
edema), and pain. Because the horse has strong eyelids, your veterinarian may perform a local nerve block with lidocaine to examine the eye. Your veterinarian may put fluorescein (a bright yellow dye) on the eye to highlight potential ulcers. A normal cornea will not take up the bright yellow stain. Your veterinarian may also take a sample of the ulcer and submit the sample for bacterial and fungal culture, however, results of those cultures may take several days to a week. In the short term, the veterinarian may also examine the cells from the sample to get hints on the potential cause of the problem.

The course of a superficial ulcer is often short as epithelial cell migration (i.e., normal healthy cells moving across to heal the surface of the ulcer) occurs at approximately 0.6mm/day. Thus, this often takes 5-7 days and usually results in minimal scarring and vision loss. Superficial corneal ulcers have limited cellular infiltrate and/or stromal loss. Deeper ulcers may become infected. Stromal abscesses can develop if the superficial epithelium heals and the infection is walled off within the stroma. Abscesses can cause severe anterior uveitis (inflammation in the front chamber of the eye) and may need surgical intervention to heal. Common infectious causes of stromal disease/abscess include, viral, fungal, and bacterial agents.

In the United States, the most common cause of viral keratitis is Equine Herpes Virus 2 (EHV-2). However, overall, viral keratitis is diagnosed less frequently than other infectious causes. EHV-2 usually causes multiple corneal defects. By contrast, primary fungal ulcers can be superficial or deep involving the stroma, and be either focal or multifocal. Infectious keratitis can also have a mixed bacterial and fungal population. Bacterial ulcerative keratitis can have marked edema (hazy cornea), progressive deepening of the ulcer bed, keratomalacia (melting), anterior uveitis, miosis (constricted pupil), and corneal vascularization.

The most common bacterial isolates are Streptococcus spp, Staphylococcus spp and Pseudomonas. These deeper ulcers require the stroma to respond with cellular infiltrates and slower healing ensues. The inflammatory process associated with deeper and more painful corneal injury signals new vessel growth or neovascularization. Normal corneas have a balance of enzymes (proteases) that remove tissue and new cell growth. In the presence of a corneal ulcer, inflammatory cells can incite an over production of proteases that cause keratomalacia (melting). Keratomalacia is often referred to as a “melting” ulcer because the stroma becomes soft and droopy looking.

Treatment of equine ulcerative keratitis is aimed at controlling infection, reducing protease activity, and controlling pain and reflex anterior uveitis (uveitis secondary to the keratitis). Antibiotic therapy is primarily topical. Topical therapy delivers a high concentration of drug at the site of infection without having to achieve a high systemic level, which would be necessary if systemic antibiotics were given. If the patient is difficult to treat, a sub-palpebral lavage system can be placed to facilitate treatment and deliver medication in a solution. Treatments may need to be done as often as every two hours. Preferably antibiotic choice should be directed based on the culture and sensitivity. Before results are obtained, therapy should be broad spectrum. A combination of neomycin, bacitracin, polymyxin B is a good choice. Chloramphenicol can penetrate the cornea and is often the first choice in stromal abscesses. Topical antiproteases such as serum can be used to combat the keratomalacia (melting). Atropine can be used to block pain associated with uveitis and help dilate the pupil. However, if atropine is used, the horse should be kept inside as the pupil cannot constrict if the horse goes out into sunlight. Oral non steroidal anti-inflammatory drugs (i.e., NSAIDS such as flunixin meglumine, [Banamine]) should also be given to control pain as well as to reduce pain reflex anterior uveitis.

Most ulcers, abscesses, and keratitis will respond to medical treatment if detected and treated early. Surgery may be needed if there is a large amount of stromal loss, delayed healing despite appropriate therapy, or rupture. Conjunctival grafts (a biological bandage) are commonly used to provide immediate corneal vascularization and support. The blood vessels can provide growth factors and antiproteases directly to the compromised cornea. In four to six weeks, the graft can be trimmed. Horses tend to recover well from this treatment, with minimal scarring present.

With the potential for poor prognosis, all corneal ulcerations should receive immediate treatment. Early treatment with topical antibiotics renders the best outcome and the quickest time to healing. The prognosis is good for ulcerative keratitis that is immediately managed and monitored until healing. Deeper and more extensive ulcers still have a good prognosis, but may require a much longer course of treatment. Surgery e.g., grafts may be needed on some horses, even if treatment is administered quickly. Untreated ulcerative keratitis can have a poor prognosis, including blindness or loss of the eye.
When Mia was born three weeks premature in early February, EFS was called out to examine her and make sure she was healthy. The EFS group recommends that foals be examined within approximately 24 hrs following birth to identify any problems, such as failure of passive transfer (not getting enough antibodies through the colostrum), congenital abnormalities, or infection. On examination, Mia had a number of congenital limb deformities, and she also had failure of passive transfer. Mia was given a liter of specially formulated equine plasma intravenously, which enhanced her overall immune system to help prevent infection. Her legs were then examined more closely. All four of her limbs had flexor tendon laxity, which caused her to walk on her heel bulbs with her toes off the ground. The left hind fetlock had a varus deformity (the pastern was angled inwards in respect to the cannon bone), and both of the knees of the front limbs had a valgus deformity (the cannon bone is angled outwards in respect to the radius). Normally, most foals have one or a few minor limb deformities, and as they age, straightening of the limbs occurs as the foals move around and use their limbs. In some cases, like Mia’s, the angulation is so severe that allowing her to continue to grow without intervention would result in worsening of the deformities. Foals’ legs grow at a rapid rate, and some of their growth plates close at a few months of age, so prompt therapy for a foal like Mia is very important. If a foal is allowed to continue to age with an excessive limb deformity, often these horses become chronically lame and unusable. Normal therapy for tendon laxity is to allow foals to exercise, causing the tendons and muscles to strengthen and resolve the laxity; however, because exercise could damage the growth plates, and therefore worsen the varus and valgus deformities, normal therapy for excessive varus and valgus deformities is strict stall rest. Thus, EFS recommended that Mia be kept confined to a stall with her mare. Over the next few weeks, her valgus and varus limb deformities would be monitored closely for straightening, but more aggressive therapies might be needed if the limbs did not straighten on their own.

Over the next few days, Mia’s flexor tendon laxity began to improve, with the front limbs becoming almost normal; however, her hind limb flexor tendon laxity persisted, and her valgus and varus deformities had failed to improve. In fact, Mia was now walking on the outside edge of her left hind foot because of the severity of her fetlock varus. EFS performed radiographic evaluation of her left hind fetlock and both of her knees. This allowed EFS clinicians to better understand exactly which bones in her legs were causing the angulations. The
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bones in her knees were rounded which is common in premature foals. The immaturity of the bones, combined with a mild angulation of the distal radius was causing the valgus deformities in the knees. Fetlock radiographs revealed the distal cannon bone of the left rear was moderately to severely angulated which cased the fetlock varus. Travis Burns, the VTH farrier, fashioned and glued small wooden shoes with heel extensions to Mia’s hind feet. These shoes were designed to prevent Mia’s toes from lifting up and hopefully allow her tendon laxity to resolve. The left hind shoe also had an extension to the outside to aid in correction of the fetlock varus. Other than keeping the front feet well balanced and restricting exercise, no additional therapy was needed for the front feet at this time. Three days later, Mia was rechecked, and although both shoes were found to still be in correct placement on the feet, the shoe on the right hind foot was not effectively keeping Mia’s toe off of the ground. In an attempt to remedy this problem, a shoe with a longer heel extension was glued to the foot.

Mia’s feet continued to be trimmed and have shoes applied as necessary to attempt to correct her tendon laxity. Her tendon laxity and front limb varus deformities were gradually improving, but conservative therapy was unsuccessful in improving the left hind fetlock varus. The decision was made to refer Mia into the Equine Surgery service at the Veterinary Teaching Hospital for surgical therapy for her limb deformities. So at approximately 3 weeks of age, Mia underwent a surgical procedure to stimulate bone growth. The surgical procedure removes a small amount of the soft tissue that covers the bone over the growth plate on the short side of the bone. This causes a mild degree of inflammation that stimulates the growth plate on that side of the limb thus allowing it to catch up to the other side. The main focus of Mia’s surgery was the left hind fetlock, but the knees were also addressed to stimulate resolution of these deformities. Mia recovered from her anesthesia without any complications, and over the next few weeks, EFS continued to monitor Mia’s growth and her incisions. During this time, her varus and valgus deformities improved significantly. She was gradually allowed increased exercise and, she and her mom are now out in a large pasture with the other mares and foals on the farm.

Mia has continued to improve, thanks to the diligent work of her owners, EFS clinicians and farrier, and the surgical staff at the VTH. All of us, as well as her owners, are hopeful that Mia will develop into a conformationally sound horse with no long term effects from these early health issues.

Please call EFS at 540-231-9042 or visit our website: http://www.vetmed.vt.edu/vth/la/equine.asp