

REVIEWS ON OPHTHALMIC CONDITIONS AFFECTING DOMESTIC ANIMALS

I - A KNOW – HOW OF MELTING CORNEAL ULCERS IN RUMINANT AND ITS MEDICAL MANAGEMENT

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“Melting corneal ulcer”, scientifically known as “keratomalacia”, is a progressive destruction of the corneal stroma, leading to liquefactive necrosis and loss of keratocytes (Brooks, 2004). These pathological changes are the consequence of an imbalance in proteinases and proteinase inhibitors activity on the corneal surface (Ollivier *et al.*, 2007). The over expression of certain proteinases of microbial, leukocyte, tear film and corneal cell origins, combined with reduction in the antiproteinase activity of naturally occurring inhibitors present in tear film and corneal tissues lead to liquefaction of the corneal stroma. Generally, melting corneal ulcers are infrequently diagnosed in ruminants compared with some monogastric animals (dogs and horses). This is considered due to the presence of lactoferrins in the tear film of some ruminants, ultra structural differences in the cornea, or other unknown factors (Estrada *et al.*, 2013).

The matrix metalloproteinases (MMP-2 and MMP-9) and serine proteinases (neutrophil elastase) seems to be abundant in the tear film of an ulcerated eye. Bacteria, fungi, epithelial cells, keratocytes, and polymorphonuclear cells together produces the tear film proteinases including the collagenases and elastases (Strubbe *et al.*, 2000) . These corneal proteinases routinely maintains the corneal stromal collagen and extra cellular matrix components, helps in leukocyte chemotaxis and activation during inflammatory keratopathies, pathogen destruction after corneal infection, corneal epithelial migration after corneal ulceration, and corneal angiogenesis (Ollivier *et al.*, 2003) . These enzymes normally exist in balance with proteinase inhibitors. Inhibition of these proteolytic enzymes through various pathways is effective in preventing the progress of corneal melting (Ollivier, 2005).

Clinically, the corneal melting in early stages can be observed as grey gelatinous corneal opacity. In advanced stages the cornea will be more gelatinous and protruding, with generalised oedema. Finally, in later stages severe hyperaemia and well marked chemosis along with deep corneal vascularisation, fibrin and hypopyon can be noticed.

The condition can be medically managed by topical application of EDTA solution, autogenous serum fortified with amikacin, atropine eye drops and 5% hypertonic saline as eye drops. All the medications can be compounded, in-house, in a sterile manner. Autogenous serum and EDTA is used as anti proteolytic agents. Autogenous serum contains α 2-macroglobulin and α 1-proteinase inhibitors which inhibits serine proteinases. EDTA chelates calcium and zinc ions which are essential for the MMPs to cause collagenolysis, thereby inhibiting action of MMPs. The most common bacteria isolated from the collagenolysis sites in cornea are, *Pseudomonas* spp. Aminoglycosides are usually the antibiotics of choice for *Pseudomonas*. So autogenous serum can be fortified with amikacin and administered. Atropine is used to reduce ocular discomfort due to ciliary muscle spasm by the relaxation of the ciliary muscles and to prevent synechiae formation. The anti-oedematous properties of hypertonic saline is also employed in managing the corneal melting. This multimodal approach does have a promising significant therapeutic outcome in the treatment of melting corneal ulcers in animals.

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Melting corneal ulcer of a cow -1st day



Improvement in corneal clarity after 1 week of medication



Return of transparency of the cornea after 3 weeks of treatment



A goat with stromal abscess and cornea with melting areas- first day



Reduction in the abscessation and oedema and reduced areas of melting ulcer-5th day



Reduced melting areas and corneal clarity regained in the peripheral cornea-2 weeks



Complete corneal clarity-3 weeks



Melting ulcer of a goat-first day



Complete clarity of cornea after 2 weeks of medication