



Purpose

To describe a case of corneal sequestrum in a dog.

Methods

A 14 year old castrated male Cairn terrier, with a remote history of KCS in the left eye (OS) only occasionally treated with topical cyclosporine (CsA) 0.2% was presented for severe blepharospasm and a black plaque involving OS of 6 weeks duration. The condition had progressively got worse despite more accurate treatment with triple antibiotic ointment and CsA 0.2%. In the last few days, the plaque had doubled in size according to owner's provided information.

Complete ophthalmic examination revealed severe blepharospasm OS, mild ocular discharge in both eyes (OU) that appeared to be enophthalmic because of a severe generalized atrophy of masticatory muscles. The left eye presented a superficial black plaque (8x4mm) in the central cornea. The plaque was surrounded by granulation tissue around 270 degrees of its perimeter. The rest of the cornea showed moderate edema. Range of motion of the temporomandibular joints was normal. Menace was normal in the right eye (OD) and absent OS, PLRs and dazzle reflexes were normal OU. Blinking reflexes were normal OU. Shirmer Tear test (STT) was 12mm/60" OS and 15mm/30" OD. Applanation tonometry results were 20 and 18 mmHg OS and OD respectively. Pulverulent nuclear cataract and nuclear sclerosis were seen OD while ocular examination was further impossible OS. Fundic examination OD was unremarkable.

Results

Complete physical examination, CBC, chemistry panel, urinalysis and chest X-rays were performed, which were unremarkable, aside from ophthalmic and masticatory muscles abnormalities. A superficial keratectomy was performed under general anesthesia and a hood conjunctival graft was applied to the corneal defect. The graft was trimmed after 2 weeks. Post operative therapy included topical tobramycin, atropine, CsA 0.2% and hyaluronic acid. Oral carprofen and oral doxyciclyne were administered for 4 and 10 days respectively. Topical tobramycin was changed with topical erythromicyn once the culture and sensitivity tests were reported.

Histologically, the corneal superficial layer was degenerated and necrotic, and its deep zone was expanded by large numbers of neutrophils that extended into the subjacent stroma along with admixed erythrocytes and macrophages, some of which had hemosiderin-laden cytoplasm. The stroma was extensively fibrotic with numerous embedded small blood vessels lined by plump endothelial cells (neovascularization). No infectious agents or neoplastic cells are seen. Von Kossa staining was negative for mineralization and PAS was negative for fungal organisms. Gram staining failed to identify any bacterial involvement.

Low growth (+1) of Streptococcus β -hemoliticus was cultured and considered a secondary infection.

TEM was processed on glutharaldeide 2% embedded specimen and revealed degenerated keratocytes, macrophagic and neutrophilic presence and denaturated collagen. No infectious agents were found.

Standard canine virus isolation (Madin-Darby K9 kidney cells) and canine herpesvirus specific PCR performed on the excised sample were negative.

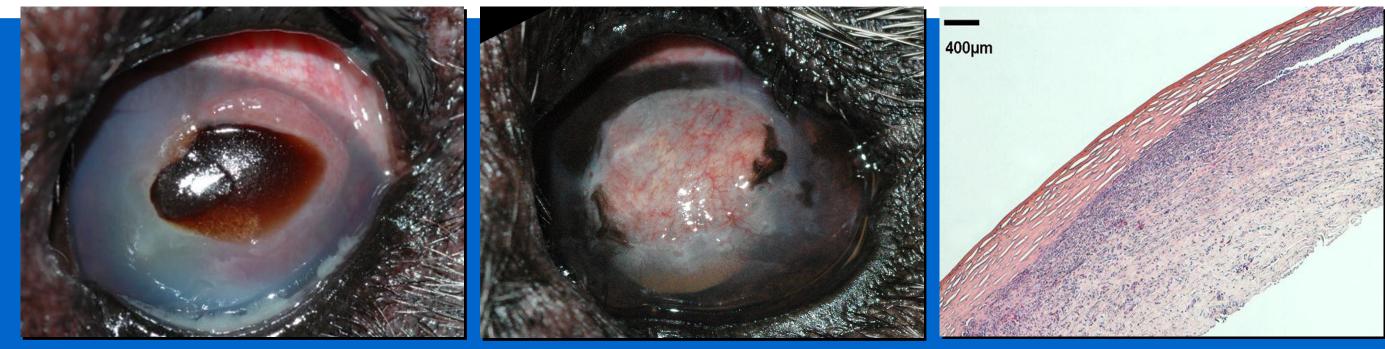


Fig 1 - Clinical picture of the left eye of a 14 year old Cairn terrier on presentation. The central cornea showed a brownish/black plaque surrounded by granulation tissue and diffuse corneal edema. Mucopurulent discharge was also present.



Fig. 4 - Photomicrograph of the keratectomy sample Fig. 5 - Photomicrograph of the keratectomy sample Fig. 6 - Photomicrograph of the keratectomy sample. showing collagen denaturation and the underlying dense inflammatory infiltrate with angiogenesis. (H&E, original magnification, x 10) original magnification, x20)



Corneal Sequestrum in a Dog

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Fig. 2 – Clinical picture of the healed graft 4 months Fig. 3 - Photomicrograph of the keratectomy sample after the surgical procedure. Although the axial cornea was covered by the graft tissue, the surrounding cornea was transparent and menace response was present.

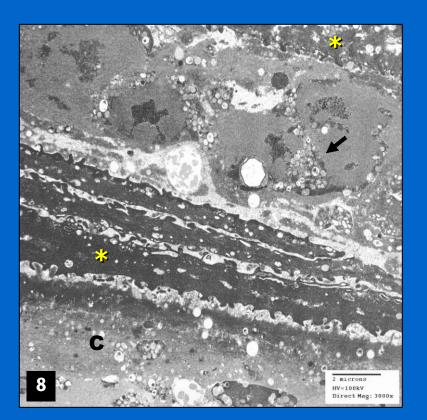
showing a superficial plaque with cellular necrosis and collagen denaturation. The stroma underlying the plaque displays a severe pyogranulomatous infiltrate and neovascularization. (H&E, original magnification, x5)

showing absence of mineral deposits (Von Kossa, No micro-organisms could be identified in the examined specimens. (Gram stain, original magnification, x10)

Electron micrographs of the keratectomy sample.

Fig. 7 - Invading macrophages (arrows) surrounded by denaturated collagen and cellular remnants (asterisks). Apparently preserved stromal lamellae can be seen (L)

Fig. 8 - Vacuolated cytoplasm of clustered keratocytes (asterisks) are interspersed between denaturating collagen (C) and vacuolated cellular residuals (arrows)



Conclusions

Corneal sequestrum is for a long time a well recognized clinical corneal entity in cats.^{1,2} The sequestrum is represented by a brownish/black plaque of degenerated corneal tissue with cellular apoptosis and collagen denaturation.

Causes are unknown and several conditions have been reported to be associated risk factors (entropion, ulcerative keratitis, trauma, corneal exposure, local or systemic metabolic defects, primary stromal dystrophy).² The role of FHV-1 infection seems controversial.^{3,4}

Surgical treatment is advocated as the gold-standard treatment, even though spontaneous healing may eventually occur in some cases treated medically.² Corneal sequestrum has also been reported in horses .^{5,6}

Stromal denaturation may occur in patients with superficial corneal erosions⁶⁻⁸ and the authors have noticed central corneal sequestrum-like lesions in dogs undergoing intrascleral prosthesis or in lagophthalmic cases (unpublished data). In these conditions, the corneal plaque may eventually heal with appropriate treatment of the predisposing causes.

This is the first description⁹ of a corneal sequestrum in a canine patient with identical clinical, histological and ultrastructural findings as those described in cats.¹⁰

The pathogenesis of the disease is unknown. Corneal sequestration has been previously associated with KCS in a horse.⁵ In this case bilateral masticatory muscle atrophy was not investigated further and possible corneal sensitivity defects (aesthesiometry was not performed) related to hypothetical cranial nerve V disease should be included into differentials. The controlateral eye however did not show any similar signs.

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