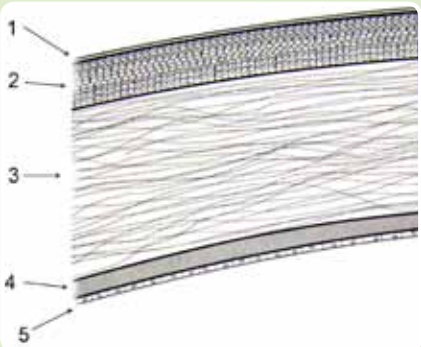


Εικ. 1. Σχηματική απεικόνιση των στιβάδων του κερατοειδή χιτώνα. 1. Προκεράτιος δακρυϊκή στιβάδα, 2. Επιθήλιο, 3. Ίδια ουσία (στρώμα), 4. Μεμβράνη του Descemet, 5. Ενδοθήλιο. (Τροποποίηση από: Gilger BC, 2007. Diseases and Surgery of the Canine Cornea and Sclera).

Fig. 1. Corneal layers. 1. Precorneal tear film, 2. Epithelium, 3. Stroma, 4. Descemet's membrane, 5. Endothelium. (Modified by: Gilger BC, 2007. Diseases and Surgery of the Canine Cornea and Sclera).

Εικ. 2. Μικροκερατοειδής. Σκύλος, φυλής Maltese, ηλικίας 7 ετών που πάσχει από μετατραυματική ραγοειδίτιδα στον δεξιό οφθαλμό. Μείωση του μεγέθους του ΚΧ εξαιτίας φθίσης του βολβού. Στον πάσχοντα οφθαλμό ο ΚΧ, σε αντίθεση με τον υγιή, δεν καταλαμβάνει ολόκληρη την επιφάνεια της βλεφαρικής σχισμής. Παρατηρείται επίσης οίδημα του ΚΧ, καθώς και υπεραιμία του βολβικού επιπεφυκότα συμπτώματα που αμφότερα συνοδεύουν την ραγοειδίτιδα.

Fig. 2. Microcornea. Dog, Maltese, 7 years old, suffering from post-traumatic uveitis in the right eye. Decrease in corneal size due to phthisis bulbi. In the diseased eye the cornea, in contraction to the healthy one, does not occupy the entire palpebral fissure. Corneal edema and hyperemia of bulbar conjunctiva are also noticed, and they are both symptoms accompanying uveitis.

Εικ. 3. Μεγαλοκερατοειδής. Σκύλος, μιγάς, 8 ετών που πάσχει από χρόνιο, τελικού σταδίου γλαύκωμα κλειστής γωνίας στον αριστερό οφθαλμό. Η αύξηση του μεγέθους του ΚΧ συνυπάρχει με τη γενικευμένη αύξηση του όγκου του βολβού του οφθαλμού (Βούφθαλμος). Παρατηρείται παρουσία οιδήματος του ΚΧ, καθώς και αρχόμενη εναπόθεση χρωστικής (βέλη).

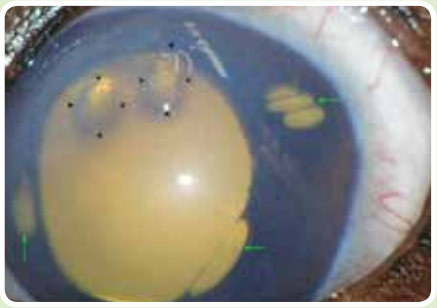
Fig. 3. Megalocornea. Dog, cross breed, 8 years old, suffering from chronic, end stage, close-angle glaucoma of the left eye. Increase in corneal size coexists with the generalized increase in globe's volume (buphthalmos). Corneal edema and incipient pigmentation (arrows) are noticed.

Εικ. 4. Κερατόκωνος. Σκύλος, μιγάς Pekingese, 10 ετών με ιστορικό κάκωσης του οφθαλμού από αμβλύ όργανο. Χρόνια κερατίτιδα με μείωση της αντοχής του στρώματος κεντρικά και αύξηση της ενδοτικότητάς του στην αντίστοιχη περιοχή. Παρατηρείται επίσης οίδημα του ΚΧ καθώς και ανάπτυξη περιφερικής εν τω βάθει νεοαγγείωσης (βέλη).

Fig. 4. Keratoconus. Dog, cross breed, Pekingese, 10 years old, with history of blunt eye trauma. Chronic keratitis, with decrease in corneal resistance centrally and increase of compliance in the same area. Corneal edema and deep peripheral neovascularisation are also noticed (arrows).

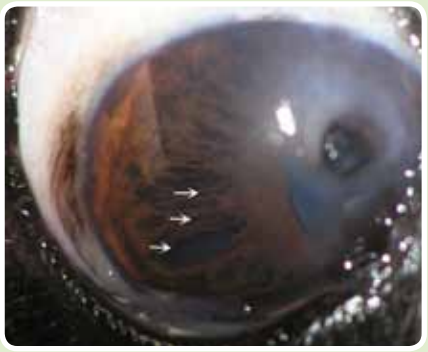
Εικ. 5. Επιθηλιακό οίδημα (λευκά βέλη) εξαιτίας επιφανειακού έλκους. Σκύλος, φυλής Γερμανικός Ποιμενικός, ηλικίας 10 ετών. Παρατηρείται ακόμα ατροφία της ίριδας λόγω προχωρημένης ηλικίας (πράσινο βέλος), καθώς και θόλωση του κρυσταλοειδούς φακού λόγω καταρράκτη (κόκκινο βέλος).

Fig. 5. Epithelial edema (white arrows) due to superficial ulcer. German Shepherd, 10 years old. Old aged iris atrophy is also observed (green arrow), as well as opacification of the lens due to cataract (red arrow).



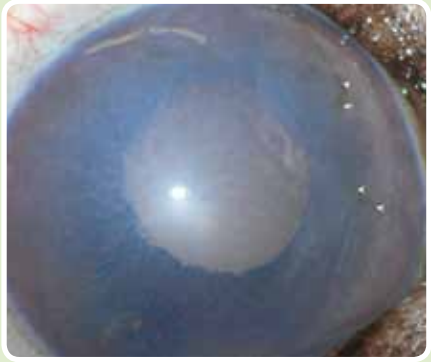
Εικ. 6. Φυσαλιδώδης κερατοπάθεια επί εδάφους εκφύλισης του ενδοθηλίου του ΚΧ. Σκύλος, μιγάς, ηλικίας 12 ετών. Ήπιο, διάχυτο, ενδοθηλιακής προέλευσης, οίδημα του ΚΧ. Παρουσία δυο μικροσταγόνων εντός του στρώματος (μαύρα βέλη). Παρατηρείται επίσης ατροφία, λόγω ηλικίας, της ίριδας (πράσινα βέλη)

Fig. 6. Bullous keratopathy due to degeneration of the endothelium. Dog, cross breed, 12 years old. Mild, diffuse, corneal edema of endothelial origin. Presence of two microdrops into the stroma (black arrows). Age-related iris atrophy is also present (green arrows).



Εικ. 7. Επιθηλιακής προέλευσης οίδημα του στρώματος του ΚΧ από βαθύ, προδεσκεμετικό έλκος. Σκύλος, φυλής Pekingese, ηλικίας 13 ετών. Παρατηρείται μεγάλη πυκνότητα του οιδήματος στην περιοχή του ΚΧ που γειτνιάζει με το έλκος και προοδευτική αραίωσή του, καθώς απομακρυνόμαστε από αυτό. Χαρακτηριστική και εδώ η ατροφία, λόγω της ηλικίας, της ίριδας (βέλη).

Fig. 7. Stromal edema of epithelial origin due to deep pre-descemetic ulcer. Dog, Pekignese, 13 years old. Dense edema is noticed around the ulcer which is gradually reduced while standing out from it. Age-related iris atrophy is also present (arrows).



Εικ. 8. Ενδοθηλιακής προέλευσης οίδημα του στρώματος του ΚΧ. Σκύλος, μιγάς ηλικίας 14 ετών, που πάσχει από εκφύλιση του ενδοθηλίου του ΚΧ. Παρατηρούνται πολλαπλές λευκωπές εστίες, με τη μορφή σύννεφων, εντός του στρώματος που τείνουν να συνενωθούν μεταξύ τους. Επιπλέον, σημειώνεται αρχόμενη εναπόθεση μελανίνης στον ΚΧ (βέλη).

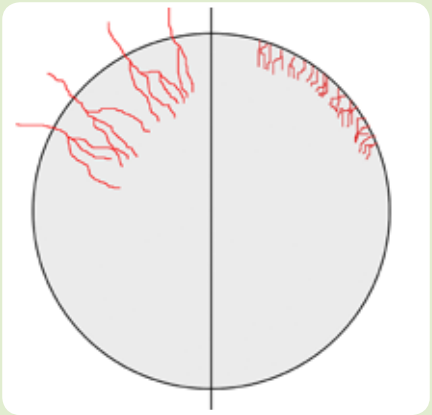
Fig. 8. Stromal edema of endothelial origin. Dog, cross breed, 14 years old, suffering from corneal endothelial degeneration. Multiple whitish, cloudy-like, spots are observed in the stroma tending to unify to each other. Moreover, incipient corneal pigmentation is noticed (arrows).

Εικ. 9. Μικτής προέλευσης οίδημα του στρώματος του ΚΧ. Σκύλος, φυλής Pekingese, 4 ετών που πάσχει από βαθύ έλκος του στρώματος του ΚΧ και πρόσθια ραγοειδίτιδα. Διάχυτο οίδημα του ΚΧ, το οποίο αυξάνεται στην περιοχή γύρω από το έλκος. Με τα λευκά βέλη σημειώνονται τα όρια της αποκόλλησης του επιθηλίου του ΚΧ και με τα πράσινα τα όρια του έλκους του στρώματος. Παρατηρείται συγκέντρωση φλεγμονώδους υλικού (υπόπυο) εντός του προσθίου θαλάμου (μπλέ βέλη), έντονη υπεραιμία του επιπεφυκότα, καθώς και ανάπτυξη επιφανειακής νεοαγγείωσης του ΚΧ.

Fig. 9. Mixed origin corneal edema. Dog, Pekignese, 4 years old, suffering from deep stromal corneal ulcer and anterior uveitis. The diffuse corneal edema is increasing around the ulcer. White arrows mark the epithelial detachment margins and green arrows the margins of the stromal ulcer. Inflammatory matrix concentration (hypopyon) is observed in the anterior chamber (blue arrows), as well as intense conjunctival hyperemia and superficial corneal neovascularisation.

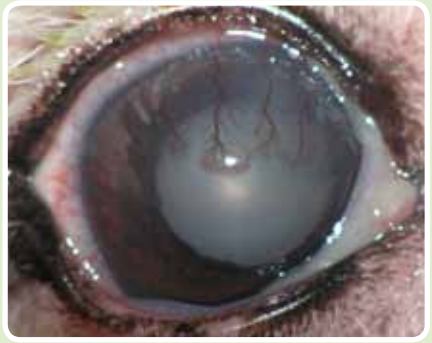
Εικ. 10. Διάμεσο οίδημα του στρώματος του ΚΧ . Σκύλος, μιγάς, ηλικίας 5 ετών που πάσχει από ξηρή κερατοεπιπεφυκίτιδα. Το οίδημα εντοπίζεται στο κέντρο του ΚΧ. Παρατηρείται επίσης ανάπτυξη επιφανειακής νεοαγγείωσης στον ΚΧ, καθώς και βλεννοπυώδες έκκριμα που χαρακτηρίζει τη νόσο και επικολλάται στα βλέφαρα.

Fig. 10. Interstitial stromal edema of cornea. Dog, cross breed, 5 years old, suffering from keratoconjunctivitis sicca. The edema is located in the center of the cornea. Superficial corneal neovascularisation and mucopurulent discharge is also present, which is typical of the disease and adheres to the eyelids.



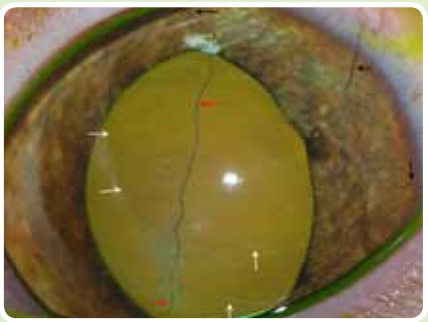
Εικ. 11. Σχηματική απεικόνιση των διαφορών μεταξύ επιφανειακής και εν τω βάθει νεοαγγείωσης του ΚΧ. Στην επιφανειακή (αριστερά), τα νεοαγγεία εμφανίζονται ως συνέχειες των αγγείων του επιπεφυκότα, διακλαδίζονται με τη μορφή κλαδιών δένδρου και έχουν ζωηρό κόκκινο χρώμα. Στην εν τω βάθει (δεξιά), τα αγγεία εμφανίζονται να εκκινούν από το σκληροκερατοειδές όριο και δεν αποτελούν συνέχειες των αγγείων του επιπεφυκότα, διατάσσονται παράλληλα μεταξύ τους και διακλαδίζονται ελάχιστα. Το χρώμα τους είναι βαθύ κόκκινο.

Fig. 11. Illustration of the differences between superficial and deep corneal neovascularisation. In superficial (left), new vessels are continuous with the conjunctival vessels, they are branching and are bright red in color. In deep (right), vessels arise from limbus and are not continuous with conjunctival vessels; they are parallel to each other and do not branch. Color is dark red.



Εικ. 12. Επιφανειακή νεοαγγείωση του ΚΧ. Σκύλος, φυλής Poodle, 9 ετών που πάσχει από ξηρά κερατοεπιπεφυκίτιδα. Τα νεοαγγεία εμφανίζονται σαν συνέχειες των αγγείων του επιπεφυκότα, διακλαδίζονται με τη μορφή κλαδιών δένδρου και έχουν ζωηρό κόκκινο χρώμα. Αξιοσημείωτη είναι επίσης η θόλωση του κρυσταλλοειδή φακού λόγω καταρράκτη.

Fig. 12. Superficial corneal neovascularisation. Dog, Poodle, 9 years old with keratoconjuctivitis sicca. New vessels appear to be continuous with the conjunctival vessels, branch, and are bright red in color. The lens opacification due to cataract formation is also remarkable.

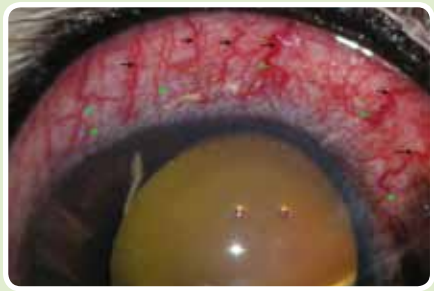


Εικ. 13. Επιφανειακή νεοαγγείωση του ΚΧ. Γάτα, φυλής Κοινής Ευρωπαϊκής, ηλικίας 2 ετών που πάσχει από χρόνια υποτροπιάζουσα κερατίτιδα λόγω μόλυνσης από τον feline herpesvirus-1 (FHV-1). Είναι χαρακτηριστική η ανάπτυξη επιφανειακής νεοαγγείωσης (μαύρα βέλη), καθώς και η ύπαρξη αγγείων σε στάδιο παλινδρόμησης (κόκκινα βέλη) και άλλων που έχουν ήδη υποστραφεί (λευκά βέλη) και έχει παραμείνει το ίχνος τους στον ΚΧ (φαντάσματα αγγείων).

Fig. 13. Superficial corneal neovascularisation. Cat, DSH, 2 years old with chronic recurrent keratitis due to feline herpesvirus-1 (FHV-1). Superficial corneal neovascularisation is typical (black arrows), as well as collapsed vessels (red arrows) and others already resolved (white arrows) with only their trace remaining in the cornea (ghost vessels).

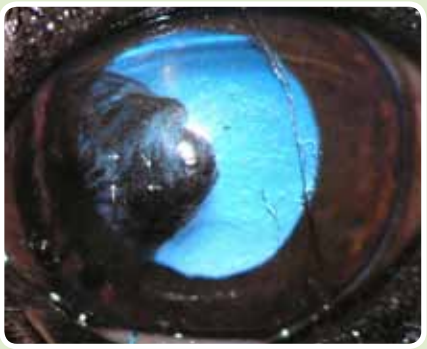
Εικ. 14. Εν τω βάθει νεοαγγείωση του ΚΧ. Σκύλος, φυλής Poodle, 12 ετών που πάσχει από πρόσθια ραγοειδίτιδα. Τα αγγεία εμφανίζονται να εκκινούν από το σκληροκερατοειδές όριο, διατάσσονται παράλληλα μεταξύ τους και διακλαδίζονται ελάχιστα. Παρατηρείται επίσης εναπόθεση φλεγμονώδους υλικού με τη μορφή κόκκων άμμου στην οπίσθια επιφάνεια του ΚΧ, εύρημα που συχνά συνοδεύει τις πρόσθιες ραγοειδίτιδες.

Fig. 14. Deep corneal neovascularisation Dog, Poodle, 12 years old with anterior uveitis. Vessels appear to begin from limbus, are parallel to each other and branch minimally. Focal accumulation of inflammatory precipitates resembling sand granules on the corneal endothelium, which is a common sign of anterior uveitis, are also observed.



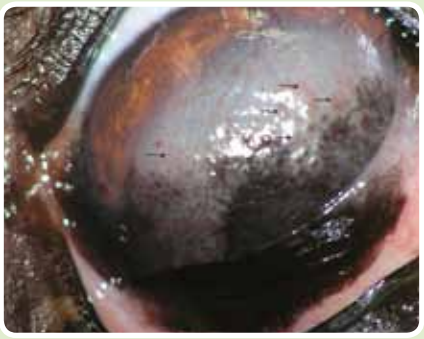
Εικ. 15. Εν τω βάθει νεοαγγείωση του ΚΧ. Σκύλος φυλής, West Highland White Terrier, που πάσχει από υπεξάρθρημα κρυσταλλοειδή φακού και γλαύκωμα. Παρατηρείται αλλαγή του σχήματος της κόρης, λόγω της εφίππευσης του κρυσταλλοειδούς φακού στην ίριδα, καθώς και έντονη διάταση των αγγείων του επιπεφυκότα (μαύρα βέλη) και του σκληρού χιτώνα (πράσινα βέλη).

Fig. 15. Deep corneal neovascularisation. Dog, West Highland White Terrier, with lens sublaxation and glaucoma. Alteration in the pupil's size is observed due to overriding of the lens on iris and intense enlargement of conjunctival (black arrows) and scleral vessels (green arrows).



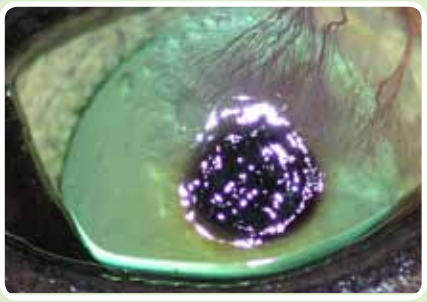
Εικ. 16. Επιφανειακή εναπόθεση μελανίνης στον ΚΧ. Σκύλος, φυλής Pug, ηλικίας 3 ετών που πάσχει από τριχίαση λόγω υπερβολικής ανάπτυξης των δερματικών πτυχών του προσώπου. Παρατηρείται επιφανειακή νεοαγγείωση (βέλη) που προηγείται συνήθως της εναπόθεσης μελανίνης.

Fig. 16. Superficial corneal melanin deposition. Dog, Pug, 3 years old, with trichiasis due to excessive facial skin folds. Superficial corneal neovascularisation is observed (arrows), which commonly precedes melanin deposition.



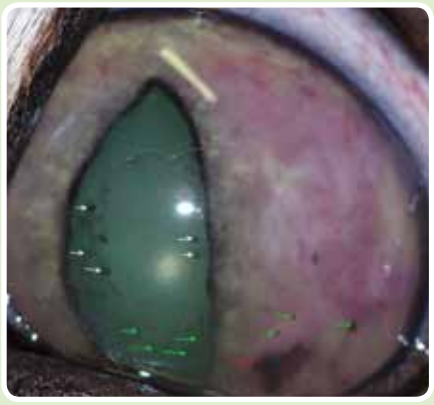
Εικ. 17. Επιφανειακή εναπόθεση μελανίνης στον ΚΧ. Σκύλος, φυλής Γερμανικός Ποιμενικός, 4 ετών που πάσχει από χρόνια επιφανειακή κερατίτιδα. Παρατηρείται διάμεσο οίδημα, επιφανειακή νεοαγγείωση και φλεγμονώδης διήθηση του ΚΧ (βέλη), αλλοιώσεις που χαρακτηρίζουν τη νόσο.

Fig. 17. Superficial corneal melanin deposition. German Shepherd, 4 years old, suffering from chronic superficial keratitis. Typical lesions of the disease as interstitial edema, superficial neovascularisation and corneal inflammatory infiltration (arrows) are observed.



Εικ. 18. Εναπόθεση μελανίνης στο στρώμα του ΚΧ. Γάτα, φυλής Persian, που πάσχει από νέκρωση του ΚΧ. Παρατηρείται έντονη ανάπτυξη επιφανειακής νεοαγγείωσης, καθώς και οίδημα του ΚΧ που εντοπίζεται ραχιαία της περιοχής της νέκρωσης.

Fig. 18. Corneal, stromal melanin deposition. Cat, Persian, suffering from corneal sequestrum. Marked superficial neovascularisation is observed, as well as corneal edema dorsally to sequestrum.



Εικ. 19. Εναπόθεση μελανίνης στο ενδοθήλιο του ΚΧ. Γάτα, φυλής Siamese, ηλικίας 2 ετών που πάσχει από πρόσθια ιδιοπαθή ραγοειδίτιδα. Λόγω της χρονιότητας της φλεγμονής, υπάρχει εναπόθεση μελανίνης στην οπίσθια επιφάνεια του ΚΧ (πράσινα βέλη). Παρατηρείται επίσης εναπόθεση μελανίνης στο πρόσθιο περιφάκιο (λευκά βέλη), και μελάγχρωση μιας περιοχής της ίριδας (κόκκινο βέλος), η οποία είναι παχυμένη και υπεραιμική.

Fig. 19. Corneal endothelial melanin deposition. Cat, Siamese, 2 years old, with anterior idiopathic uveitis. Melanin deposition occurs on to posterior corneal surface (green arrows), due to chronic inflammation. Melanin deposition is also noticed on the anterior lens capsule (white arrows) and iris regional pigmentation (red arrow), which is thickened and hyperemic.

Εικ. 20. Εναπόθεση μικροκρυστάλλων στον ΚΧ. Σκύλος, μιγάς φυλής Collie, ηλικίας 6 ετών, που πάσχει από δυστροφία του κερατοειδή.

Fig. 20. Microcrystal corneal deposition. Dog. Collie-cross, 6 years old with corneal dystrophy.

Εικ. 21. Εναπόθεση μικροκρυστάλλων χοληστερόλης στον ΚΧ. Σκύλος, μιγάς, 5 ετών που πάσχει από υποθυρεοειδισμό και εκφύλιση του ΚΧ. Παρατηρείται επίσης ανάπτυξη νεοαγγείωσης λόγω της χρονιότητας της αλλοίωσης.

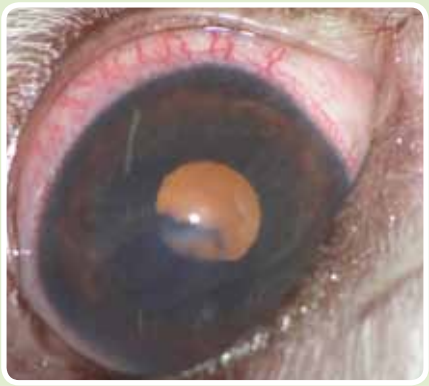
Fig. 21. Cholesterol microcrystal deposition on cornea. Dog. cross-breed, 5 years old with hypothyroidism and corneal degeneration. Neovascularisation is also present due to chronicity of the lesions.

a

b

Εικ. 22. Επιθηλιακό έλκος του ΚΧ. Σκύλος, μιγάς φυλής Γερμανικός Ποιμενικός, που πάσχει από χρόνιο υποτροπιάζον επιθηλιακό έλκος του ΚΧ a. Παρατηρείται ήπιο επιθηλιακής προέλευσης οίδημα του ΚΧ, καθώς και αποκόλληση του επιθηλίου στην περιφέρεια του έλκους (βέλη) b. Το έλκος μετά τη χρώση του με διάλυμα φλουορεσκεΐνης.

Fig. 22. Superficial epithelial corneal ulcer. German Shepherd with chronic corneal superficial recurrent ulcer. a. Mild edema of epithelial origin is apparent, as well as epithelial detachement around the ulcer (arrows) b. Fluorescein staining of the ulcer.

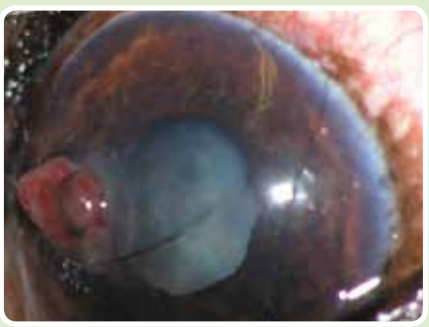


Εικ. 23. Έλκος στρώματος του ΚΧ. Σκύλος, φυλής Poodle, ηλικίας 6 ετών που πάσχει από οξύ, βαθύ (1/2 του πάχους του στρώματος) μη επιπλεγμένο έλκος. Παρατηρείται κλιμάκωση στο τοίχωμα του έλκους, το οποίο έχει τη μορφή κρατήρα. Σημειώνεται επίσης ήπιο οίδημα και απουσία νεοαγγείωσης λόγω της οξύτητας της βλάβης.

Fig. 23. Stromal corneal ulcer. Dog. Poodle, 6 years old with acute deep (1/2 of total stromal thickness) uninfected corneal ulcer. A crater shape escalated ulcer wall is observed. Mild edema and absence of vascularisation is noticed, due to acuteness of lesion.

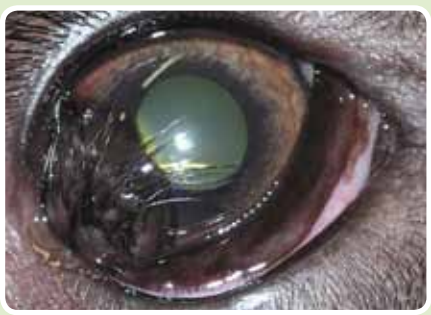
Εικ. 24. Προδεσκεμετικό έλκος του ΚΧ. Η ίδια περίπτωση της εικόνας 10 μετά τη χρώση με διάλυμα φλουορεσκεΐνης. Η στιβάδα του Descemet που αποτελεί τον πυθμένα του έλκους δεν συγκρατεί τη χρωστική. Το ίδιο και τα τοιχώματά του που εκτός από ένα μικρό τμήμα τους έχουν επιθηλιοποιηθεί στο μεγαλύτερο μέρος τους.

Fig. 24. Pre-descemetic corneal ulcer. Same case as in Figure 10 after fluorescein staining. The Descemet's membrane is not stained. The ulcer walls have already been re-epithelialised and also are not stained except from a small area.



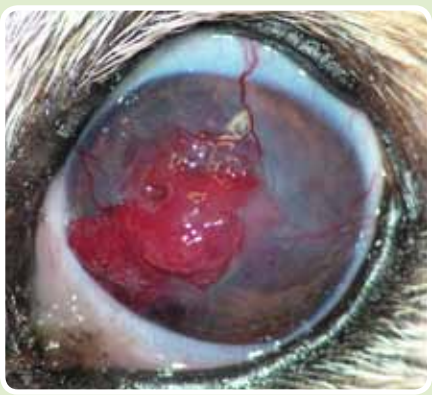
Εικ. 25. Διατιτραίνον έλκος του ΚΧ με πρόπτωση της ίριδας (σταφύλωμα). Σκύλος, μιγάς, 11 ετών.

Fig. 25. Perforating corneal ulcer with iris prolapse (staphyloma). Dog, cross breed, 11 years old.



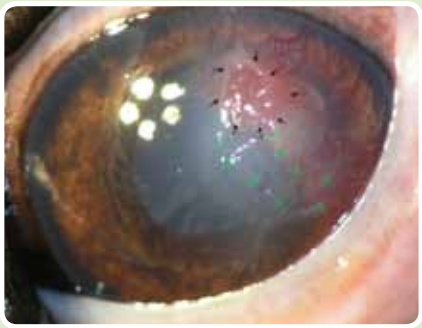
Εικ. 26. Δερμοειδής κύστη του ΚΧ. Σκύλος, φυλής Schnauzer, 7 μηνών.

Fig. 26. Corneal dermoid . Dog, Schnauzer, 7 months old.



Εικ. 27. Νεοπλασία του ΚΧ. Σκύλος, μιγάς Collie, ηλικίας 12 ετών που πάσχει από πρωτοπαθές αιμαγγειοσάρκωμα του ΚΧ. Παρατηρείται έντονη επιφανειακή νεοαγγείωση του ΚΧ.

Fig. 27. Corneal neoplasia. Dog, Collie-cross, 12 years old, suffering from primary corneal hemangiosarcoma. Intense superficial neovascularisation is noticed.

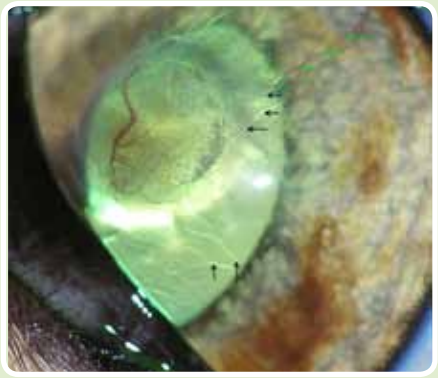


Εικ. 28. Ανάπτυξη κοκκιώδους ιστού στον ΚΧ. Σκύλος, φυλής Boxer, ηλικίας 5 ετών που πάσχει από χρόνιο υποτροπιάζον επιθηλιακό έλκος του ΚΧ. Τα όρια του ενεργού έλκους σημειώνονται με πράσινα βέλη. Εξαιτίας της αδυναμίας της φυσιολογικής επιθηλιοποίησης του έλκους, υπάρχει δευτερογενής ανάπτυξη κοκκιώδους ιστού στην επιφάνεια του ΚΧ (μαύρα βέλη). Παρατηρείται επίσης οίδημα και νεοαγγείωση του ΚΧ.

Fig. 28. Corneal granuloma tissue formation. Dog, Boxer, 5 years old, suffering from chronic recurrent superficial corneal ulcer. Margins of active ulcer are highlighted with green arrows. Secondary granule tissue formation occurs on the surface of the cornea due to ulcer's inability to heal (black arrows). Corneal edema and neovascularisation are also noticed.

Εικ. 29. Εωσινοφιλική διήθηση του ΚΧ. Γάτα, φυλής Siamese, ηλικίας 5 ετών που πάσχει από εωσινοφιλική κερατίτιδα.

Fig. 29. Corneal, eosinophilic infiltration. Cat, Siamese, 5 years old, suffering from eosinophilic keratitis.



Εικ. 30. Ανάπτυξη ουλώδους ιστού στον ΚΧ. Η ίδια περίπτωση της εικόνας 21, 45 ημέρες μετά τη χειρουργική αποκατάσταση με τη χρήση βιολογικού αλλομοσχεύματος. Στην περιοχή της νέκρωσης έχει αναπτυχθεί αραιός ουλώδης ιστός με τη μορφή νεφελώματος. Παρατηρείται υποστροφή των νεοαγγείων του ΚΧ (πράσινα βέλη) τα ίχνη των οποίων παραμένουν σαν αγγεία-φαντάσματα (μαύρα βέλη).

Fig. 30. Fibrous tissue formation on cornea. Same case as in Figure 21, 45 days later after surgical restoration using a biological allograft. In the area of sequestrum sparse connective tissue appears as a nebula lesion. Collapse of the newvessels is noticed (green arrows) and their traces appear like ghost vessels (black arrows).

Εικ. 31. Συμφυτικό λεύκωμα. Γάτα, φυλής Κοινής Ευρωπαϊκής, ηλικίας 2 ετών που πάσχει από παλαιό διατιτραίνον τραύμα του ΚΧ. α. Πλάγια εικόνα της αλλοίωσης όπου απεικονίζονται οι πρόσθιες συμφύσεις της ίριδας. β. Κατά μέτωπο εικόνα στην οποία παρατηρείται πορσελανώδης, λευκού χρώματος ουλή στη περιοχή των συμφύσεων.

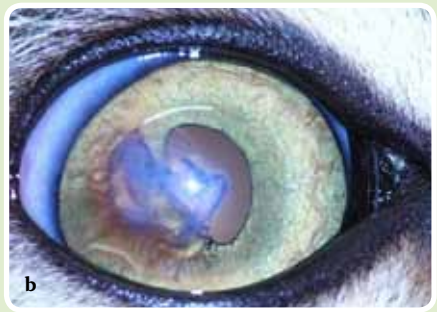


Fig. 31. Adhesive leukoma. Cat, DSH, 2 years old, with old, perforating, corneal trauma. a. Lateral view of anterior iris synechiae b. Front view. A porselanoid, white scar on synechiae site is noticed.

Εικ. 33. Πολλαπλές αλλοιώσεις του ΚΧ. Σκύλος, φυλής Γερμανικός Ποιμενικός, ηλικίας 5 ετών που πάσχει από βαθύ έλκος του στρώματος, επί εδάφους χρόνιας επιφανειακής κερατίτιδας. Διάμεσο οίδημα του στρώματος του ΚΧ, επιφανειακή νεοαγγείωση, εναπόθεση χρωστικής (μελανίνη), φλεγμονώδης διήθηση (βέλη), έλκος του στρώματος και επιθηλιακής προέλευσης οίδημα του στρώματος του ΚΧ. Το τελευταίο εντοπίζεται γύρω από το έλκος και είναι πυκνότερο από το διάμεσο οίδημα που εντοπίζεται στον υπόλοιπο ΚΧ.

Fig. 33. Multiple corneal lesions. German Shepherd suffering from deep stromal corneal ulcer and chronic superficial keratitis. Interstitial stromal edema, superficial neovascularisation, pigmentation (melanin), inflammatory infiltration (arrows), stromal ulceration and stromal edema of epithelial origin. The last is presented around the ulcer with higher density compared to interstitial edema which is noticed in the rest of the cornea.

Fig. 32. Symblepharon. Cat, DSH, 5 months of age, with history of neonate keratoconjunctivitis.

Εικ. 32. Συμβλέφαρο. Γάτα, φυλής Κοινής Ευρωπαϊκής, ηλικίας 5 μηνών με ιστορικό συνδρόμου κερατοεπιπεφυκίτιδας της νεογνικής ηλικίας.

Clinical signs of corneal lesions in dog and cat

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Key words

- cornea
- dog
- cat

> Abstract

Corneal damage in dog and cat can alter its clarity and transparency in light, which are essential for its function. Corneal lesions include edema, neovascularisation, pigmentation, microcrystal depositions, ulceration, inflammation or regenerative tissue formation, scar formation and finally those involving its size and curvature. This lesion may occur solely or in conjunction and may be caused by corneal, other ophthalmic or systemic diseases. In this study the above mentioned corneal lesions of the dog and cat are extensively reported and described.

> Introduction

Cornea is the frontier, clear and transparent in light part of the fibrous tunic of the eye. Cornea is the first and most important diopter of the eye's refractive structures, since it ensures the 2/3 convergence of light rays on the retina. Any corneal damage, which leads to change of its size, shape or transparency, may alter more or less the vision. Clinicians should to be able to understand the pathophysiology of corneal lesions, as well as to identify and describe them, in order to be able to correlate them with certain diseases. This study describes the most commonly presented corneal lesions of the dog and cat in everyday practice.

> Anatomy and physiology[1, 2, 3]

Mean corneal thickness in the dog is 0,562mm[4] and in the cat[5] 0,546mm and it consists from outwards to inwards of:

- Anterior epithelium
- Stroma
- Descemet's membrane
- Endothelium

In these four anatomical layers of cornea one more has to be added (Fig. 1), the precorneal tear film (PTF).[6]

Corneal integrity is essential for vision quality, while its role is mechanical and optical.

Mechanically, cornea contributes in the formation of eye globe, supporting and protecting its inner anatomical structures. It is worth to be mentioned, in regards to its fine structure, the high resistance of cornea to elevated intraocular pressure and environmental injuries.

Optically, cornea functions as a convergent lens. Moreover, it is the most important convergent lens of the eye's refractive system, as its convergence capability is 42 D. Corneal function is correlated to its own clarity and transparency in light. These are based on:

1. Normal corneal curvature and smooth optical surface. Normal curvature depends on the anatomical integrity of cornea. Its smooth and shiny surface is provided by the PTF.
2. Nonkeratinized surface epithelium. Corneal epithelium is stratified squamous epithelium with high regenerative capability. It consists of 7-9 cell layers. The anterior layers are not keratinized in order to remain transparent.
3. Lack of blood vessels. Healthy cornea is avascular. Nutrition is provided in a limited level by the limbus vessels and mainly from PTF through epithelium and from aqueous humor through endothelium. In contrast to vascularisation, sensitivity of cornea is very extended and is considered as one of body's most sensible tissues. Nerve endings are provided from ciliary nerves, which are the ending branches of the ophthalmic branch of trigeminal nerve. Nerve endings are distributed into epithelium and superficial stromal layer which may explain that superficial corneal lesions are more painful than deeper ones.
4. Special stromal architecture, which forms 90% of total cornea's thickness. Stroma is mainly consisted of lamellae of fibrous tissue, few fibrocytes (keratocytes) and interstitial matrix (glycosaminoglycans). The architectural arrangement of stromal collagen fibrils and lamellae differs from those of other connective tissue, providing stromal transparency.
5. Regulation of stromal humidity level. Stromal transparency depends on the maintenance of stromal humidity level. Increase in corneal humidity leads to corneal edema and loss of transparency. The integrity of both epithe-

lium and endothelium is essential for maintaining the hydrostatic state of stroma. Epithelium is water resistant and consisting a barrier to water diffusion from PTF to stroma. Endothelium, even though consisting of a single cell layer, is responsible for energy-dependent ion and water transfer from stroma to aqueous humor and anterior chamber.

> CORNEAL LESIONS

1. Size and curvature abnormalities

Microcornea: The decrease of corneal size may be congenital, as a part of microphthalmia[7] or acquired following a phthisis bulbi (Fig. 2).

Magalocornea: Magalocornea is a rare condition, usually congenital.[7] When acquired it follows the total increase in bulbi size, due to increased intraocular pressure (end stage chronic glaucoma- buphthalmus) (Fig. 3).

Ceratoconus. Unlike human, in dogs and cats ceratoconus is not a primary disease. It is usually secondary to chronic keratitis or central corneal ulcer, which results in central corneal thinning and increased compliance centrally (Fig. 4).

2. Corneal edema[7]

Edema is the most common corneal defect. Usually it coexists with other corneal defects. Corneal edema is characterized by the overhydration of corneal tissue, which leads to corneal opacity and decreased transparency. There are several causes of corneal edema and sometimes more than one can occur concurrently. Corneal edema can be focal or diffuse and concerns epithelium, stroma or both.

Epithelial edema (Fig. 5) usually is mild. In slit lamp biomicroscopy epithelial edema appears like a vapour on a pane of glass. Following installation of glycerine dilute, epithelial edema is temporally disappeared. Epithelial edema is caused by erosion or loss of the epithelium, which results in imbibition of PTF fluid by the inner epithelium layers or superficial stroma. For this reason the edema is confined to the area of epithelial defect.

Stromal edema appears as a mild or severe corneal opacity which in some cases may appear milky. In the beginning, slit lamp biomicroscopy reveals multiple white corneal spots with haze edges which tend to join each other. Upon slit lamp examination low or high degree of corneal thickness (proportional to the degree of edema) is typically observed. A special form of stromal edema is bullous keratopathy, in which there is not a corneal fluid imbibition by stromal interstitial tissue but micro-drops accumulation in the stroma[7] (Fig. 6). Causes of corneal edema can be epithelial, endothelial or both in origin. A unique form of edema is the interstitial stromal edema in which epithelium, as well as endothelium is intact.

Stromal edema of epithelial origin is caused by the disruption of the epithelial barrier between PTF and stroma, due to epithelial injury (Fig. 7). This results in absorbing water by glycoaminoglycanes of stromal interstitial substance. Corneal ulceration is the most common cause of epithelial origin edema. In these cases stromal edema is located in and around the lesion and its density is proportional to its extension, depth and chronicity.

Stromal edema of endothelial origin (Fig. 8) is caused by endothelial functional incapability to move water from the stroma to aqueous chamber.[3-7] In these cases the flow is reversed and aqueous humor enters stroma. Decrease in endothelial functional capacity is commonly caused by inflammation (endothelitis), which may be primary or secondary to anterior uveitis.[8] Typical stromal edema of endothelial origin is noticed in canine adenovirus-1 infection (CAV-1).[9] Less common causes of endothelial damage and subsequent stromal edema formation is endothelial dystrophy and degeneration,[10-11] iris anterior synechiae, as well as trauma during intraocular procedures.[12] The extension and density of edema is also related to the severity of endothelial injury.

Corneal edema of mixed, epithelial and endothelial, origin is caused by injury of both epithelium and endothelium (Fig. 9). In these cases endothelial injury causes a diffuse homogenous corneal edema, while epithelial regional defect increases locally its density.

In interstitial stromal edema both epithelium and endothelium are intact (Fig. 10). Initially, it is peripheral and is extended gradually centrally to the cornea. Interstitial keratitis is the most common cause of interstitial edema which is commonly accompanied by inflammatory cell infiltration. Another form of stromal interstitial edema is the one accompanying canine leishmaniosis, even though in these cases edema is caused mainly by anterior uveitis and endothelitis.

3. Corneal Neovascularisation[13]

Normal cornea is avascular. Vascularisation beyond limbus, as part of corneal tissue defense function is always pathologic and leads to decrease corneal transparency. The form, density and depth of vascularisation is of high diagnostic importance. Morphologically, corneal new-vessels may form bunches, branches or may expand circularly to limbus.

Density of neovasularisation may vary and is correlated to the severity of the underlying disease. The density and the length of corneal new-vessels are proportional to chronicity of the lesion, as their development does not exceed 1-2mm/24h.

Corneal neovascularisation may be either superficial or deep (Fig. 11). In superficial vascularisation (Fig. 9, 10, 11), new-vessels are derived from limbus vessels and are continuous with the conjunctival vessels. They are located in the corneal epithelial layer or underneath; they form brightly red colored branches. Superficial neovascularisation is related to acute

or chronic superficial corneal lesions (e.g. superficial ulcers, superficial keratitis). Frequently, only vascular walls (ghost vessels) are observed in cornea, which indicate previous corneal vascularisation (Fig. 13). In deep corneal neovascularisation (Fig. 14, 15) new-vessels begin from ciliary vessels; and thus they appear to start from limbus and they are not continuous with the conjunctival vessels. They are located in stroma, in parallel order and are minimally branched. Frequently they have a brush appearance. They are dark red. Deep corneal neovascularisation is related to stromal diseases (e.g. deep ulcers, interstitial keratitis), as well as to uveitis and glaucoma.

4. Corneal pigmentation

Corneal pigmentation is most commonly presented in the dog than in the cat. Melanin is the most common pigment, while infrequently pigmentation with hemoglobin or metal dyes due to foreign metal body fixation in the cornea, may occur.

4α. Melanin deposition[13]

Melanin is the most common pigment deposited on the cornea. It suggests chronic lesion. Cornea melanin originates from limbal, ciliary body and iris melonocytes. Transportation takes place through vessels, thus it is highly correlated with neovascularisation. Melanin, depending on its cause, is accumulated superficially and more rarely in stroma or endothelium.

Epithelial deposition of melanin suggests complication of chronic corneal edema or chronic corneal irritation (e.g. distichiasis, trichiasis, entropion, corneal overexposure due to exophthalmos etc.) (Fig. 16). Moreover, superficial accumulation of melanin accompanies frequently or follows superficial inflammations, such as chronic superficial keratitis and keratoconjunctivitis sicca (Fig. 17).

Stromal deposition of melanin is observed mainly in corneal sequestrum of the cat[14] (Fig. 18).

Deep deposition of melanin is less common and usually it is noticed in chronic anterior uveitis (Fig. 19). In such cases, melanin granules, originated from the inflamed anterior uvea, swing in the aqueous humor and accumulate on the posterior corneal surface.

4β.Hemoglobin deposition

Corneal hemoglobin deposition is rare. It results from anterior chamber hemorrhage and stromal infiltration of hemoglobin through endothelium and Descemet's membrane. Hemoglobin deposition in the beginning is brownish in color and with time it becomes green and yellow-green.

5. Microcrystal deposition[7]

It concerns lipid, cholosterole or calcium microcrystal deposits of metallic lustre in the cornea. They are usually bilateral and symmetrical. Usually they are correlated with corneal dystrophy or degeneration (Fig. 20). Rarely are they associated with systemic diseases, which cause alterations in lipids or calcium metabolism (hypothyroidism, diabetes mellitus, pancreatitis) (Fig. 21).

6. Corneal ulceration[7, 13, 15]

Corneal ulceration refers to any corneal tissue loss. Epithelial ulcers concern only epithelial loss. A particular epithelial ulcer form is the chronic recurrent corneal ulcer (Fig. 22). When it concerns stromal defect, it is characterized as stromal ulcer (Fig. 23). In case of full thickness stromal defect, it is characterized as pre-descemetic ulcer (Fig. 7). In these cases Descemet's membrane is protruding within the ulcer in various degrees due to its elasticity. It must be mentioned that in most cases, stromal ulcer is accompanied by other lesions (edema, neovasularisation). Fluorescein staining is positive in ulcers although in case of pre-descemetic ulcer, only its vertical walls are stained, if they are not epithelialized and not its floor (Fig. 24). Finally, in Descemet's membrane rupture the ulcer is characterized as perforated, while from its floor it may protrude a part of iris (staphyloma), which is usually covered by fibrin (Fig. 25).

7. Neoplastic tissue development/ inflammatory reaction

Dermoid is congenital and it results from the development of skin tissue on cornea (Fig. 26). This tissue may include all anatomical features of the skin, as well as hair follicles.[16]

Several types of corneal neoplasms have been described such as squamous cell carcinoma, papilloma, lymphosarcoma, hemangiomas, hemangiosarcoma and adenocarcinoma.[17] They are presented like raised areas, rich in vascularisation (Fig. 27).

In some chronic keratitis or during ulcer healing process, cornea is infiltrated by inflammatory cells/ granular tissue. This inflammatory infiltrations form smaller or larger red-white raised corneal spots. Usually, they are rich in vascularisation with an irregular surface (Fig. 28). When these infiltrations are located away from limbus they are irrigated from one or two vessels. Occasionally, in the inflamed area erosions are apparent and fluorescein staining is positive but this does not correspond to corneal ulcer. A special form of inflammatory corneal infiltration is the eosinophilic keratitis of the cat in which eosinophils predominate[18, 19] (Fig. 29).

8. Scar formation[13]

Superficial lesions of the cornea concerning epithelium as well as superficial stromal layers heal without scar formation, even though a minimal alteration in corneal curvature may be noticed leading to astigmatism. In deeper stromal lesions scar tissue formation is usual. This is caused by the inability of proper reparation of corneal architecture, which is responsible for its clarity. The scar tissue may have the form of nebula (Fig. 30) which is transparent or of a dense

white to percenaloid area which is opaque. A special category of scar formation is the adherent leukoma in which iris attaches cornea posteriorly and suggests previous corneal penetration (Fig. 31). Finally, severe keratoconjunctivities may result in adhesions between cornea and conjunctiva or/and eyelids (symblepharon). These lesions vary in extension and appear frequently in cats suffering from keratoconjunctivitis due to the feline upper respiratory syndrome (Fig. 32).

The aforementioned lesions may appear solitary or in combination (Fig. 33). When more than one lesion coexists, clinician should observe and describe them using a bright light source and appropriate magnifying equipment. Then, history and other findings of the ophthalmic examination are taken under consideration paying attention in the sequence in which these signs first appeared. Differentiation of corneal lesions as primary or secondary is of great importance, for both the diagnostic approach and the treatment of corneal diseases.

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