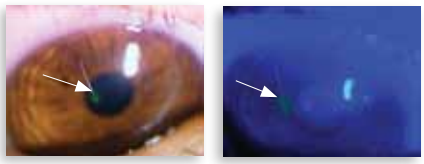
Answer to Ophthaproblem *continued from page 667*

2. Filamentary keratitis

Filamentary keratitis (FK) is a relatively uncommon disorder[1-3] characterized by the presence of corneal filaments (**Figure 1**),[3] which are fine strands of degenerated epithelial cells and mucous attached to the cornea.[1,2,4] It can be chronic and debilitating, with recurrent exacerbations.[1,5]

Figure 1. Corneal filament viewed with normal and cobalt blue light after fluorescein staining



Patients often present with discomfort, photophobia,[3] foreign body sensation, blurred vision,[1] grittiness, blepharospasm, and increased blinking.[2,5] Signs include decreased corneal sensation, corneal filaments, mucous debris, redness,[1] superficial punctate keratopathy, and punctate epithelial erosions, as well as abnormal tear break-up time and Schirmer test results.[5] Impression cytology of the conjunctiva might reveal squamous metaplasia and reduced goblet cell density with inflammatory cell infiltrates.[1] The size and shape of corneal filaments might vary and, with time, become fatter, longer, and more twisted owing to mechanical forces of the eyelids.[1,2,5] They stain best with rose bengal dye and less well with fluorescein.[2,5]

Filamentary keratitis can be a functionally debilitating and sight-threatening complication of a number of systemic and ocular conditions,[1] most commonly the aqueous-deficient dry eye conditions, also known as keratoconjunctivitis sicca (KCS).[1] Both the autoimmune (Sjögren syndrome) and nonautoimmune forms of aqueous-deficient dry eye can feature corneal filaments.[1] Kowalik and Rakes reported that 95% of corneal filaments were secondary to KCS and that evidence of rheumatologic disease was present in 75% of patients with KCS.[2] Superior limbic keratoconjunctivitis is the second most common disease associated with corneal filaments; as many as 50% of patients develop FK during the course of their disease.[2] Other conditions associated with FK include exposure keratitis; corneal edema; postcataract, corneal, or refractive surgery; and contact lens use.[1-5] Less often, FK is associated with acne rosacea and adenoviral, herpetic, and bacterial keratitis.[2] Systemic medications such as diuretics and antihistamines are capable of altering aqueous tear and mucous production, and have been associated with FK.[5] Filaments might also result from extended eyelid closure secondary to medications, extended patching, or psychiatric conditions.

The exact pathogenesis of FK is unclear.[1,4] Zaidman et al[4] hypothesized that an underlying process damages the basal corneal epithelium, basement membrane, or Bowman layer, leading to focal areas of basement membrane detachment. With time and mechanical shearing of the eyelids, these areas become elevated and act as receptor sites for mucous and degenerated epithelial cells. Once established, corneal filaments are firmly attached to the underlying epithelium and the friction between them and the upper lid results in epithelial tears, pain, and inflammation, leading to further filament formation.[2,4,5]

Management

For some, FK can be managed with preservative-free artificial tears.[2,5] In other cases, several treatments might be required. Debridement of filaments might cause swift relief of symptoms, but it is important to treat the underlying disease when possible.[1,2,5] Other options include mucolytic agents, hypertonic saline, topical nonsteroidal antiinflammatory drugs, topical steroids, punctual plugs, and bandage contact lenses.[1,2,5] Judicious use of topical medications is important, as the cornea is more susceptible to the intrinsic and preservative toxicities of the medications.[1] Topical steroids should be reserved for acute exacerbations, as overuse can lead to cataract formation and increased intraocular pressure.[1,5] Short-term treatment with soft contact lenses is beneficial to allow the basal epithelial cells to re-attach to the basement membrane.[2,4,5] It is important to treat concomitant ocular surface problems such as meibomitis[1] with warm compresses, eyelid margin hygiene, and in severe cases oral tetracyclines.

Patient education and counseling are crucial for long-term management.[1,5] Many of the underlying etiologies have no cures, and patients should be prepared to incorporate a maintenance regimen into their daily routines.[5] Acute exacerbations are common, and regular follow-up visits might be necessary to ensure vision is preserved.[5]

Our patient was treated with acetylcysteine eye drops 4 times daily for 2 weeks, combined with 5 minutes of warm compresses twice daily to promote proper eyelid margin meibomian gland function. Symptoms and corneal filaments resolved without debridement. She uses an ocular lubricant and warm compresses to prevent recurrence.

Conclusion

Filamentary keratitis is relatively uncommon but has potentially sight-threatening consequences.[1] Thorough systemic examination and hematologic workup are important in chronic cases to investigate for conditions such as Sjögren syndrome, rheumatoid arthritis, and thyroid disease.[5]

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Competing interests

None declared

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