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Case report

Severe keratomalacia after 12 months of continuous hydrogel contact lens wear in a psychiatric patient

Laura Rico-Sergado[a,*], Jose L. Pérez-Canales[a], Juan J. Pérez-Santonja[a,b], Sonia Cigüenza-Sancho[c]

[a] Department of Ophthalmology, Alicante University General Hospital, Alicante, Spain
[b] Cornea Unit, OftalVist Group, Alicante, Spain
[c] Department of Pathology, Alicante University General Hospital, Alicante, Spain

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ABSTRACT

A 53-year-old cachectic patient diagnosed with major depressive disorder was referred to our department for evaluation of a visible deformation of the right eye. She had been wearing hydrogel contact lenses on a continuous basis without removal for the last 12 months, influenced by low self-esteem and social isolation. Slit-lamp examination of the right eye showed a conical cornea, extensive neovascularization, severe stromal melting with descemetocele formation and forward bulging of the iris. Examination of the left eye revealed multiple corneal opacities, deep stromal neovascularization and anterior chamber inflammation. No sign of infection was present. Vitamin A deficiency was suspected and later confirmed. The patient required evisceration of the right eye and psychiatric treatment. Inflammatory signs of the left eye resolved within 1 week of initiating treatment. This case illustrates the synergistic effect of soft contact lens abuse and vitamin A deficiency in a psychiatric patient, and emphasizes the importance of instructing vulnerable patients on appropriate lens use and care.

1. Introduction

Vitamin A deficiency has been associated with severe malnutrition, and is still a major cause of blindness in many parts of the world, particularly in Africa and Asia. However, it is a rare event in the United States (USA) and Europe [1]. Although vitamin A deficiency has been directly related to xerosis, keratomalacia and nyctalopia, the pathogenesis of keratomalacia is usually complex, requiring the combination of multiple factors in vulnerable patients.

Since their introduction in the USA and Europe in the 1960s, hydrogel contact lenses have been frequently associated with corneal disorders such as epithelial erosions, infectious keratitis and stromal neovascularization [2]. Contact lenses interact with corneal tissue in several different ways and may interfere with normal corneal physiology. Hydrogel contact lenses act as a physical barrier to oxygen transmission, reducing the oxygen supply to the cornea. It has been suggested that chronic hypoxia is responsible for changes in corneal structure and function, inducing dysregulation of the wound-healing process, corneal neovascularization and stromal thinning [3].

This report details the case of a patient diagnosed with major depressive disorder and vitamin A deficiency who admitted a 12-month history of continuous hydrogel contact lens wear, presenting with severe keratomalacia, stromal melting and extensive corneal neovascularization.

2. Materials and methods

A 53-year-old woman was admitted to the emergency room with a history of 25 kg weight loss over 12 months, constant listlessness and depressed mood. On admission, she reported feeling depressed and worthless since her daughter's death 1 year ago. She had complete loss of appetite and erratic eating habits. She met the Diagnostic and Statistical Manual of Mental Disorders-IV (DSM-IV) criteria for a current depressive episode (depressive mood, anhedonia, weight loss, feeling of worthlessness and sleep disturbances) and was diagnosed with major depressive disorder. After psychiatric assessment, the patient was referred to our department for further evaluation of a visible deformation of the right eye.

Her past medical history showed no evidence of previous ocular disease, surgery or treatment, except myopia. She had been

* Corresponding author. Tel.: +34 965933000.
E-mail address: laurise86@gmail.com (L. Rico-Sergado).

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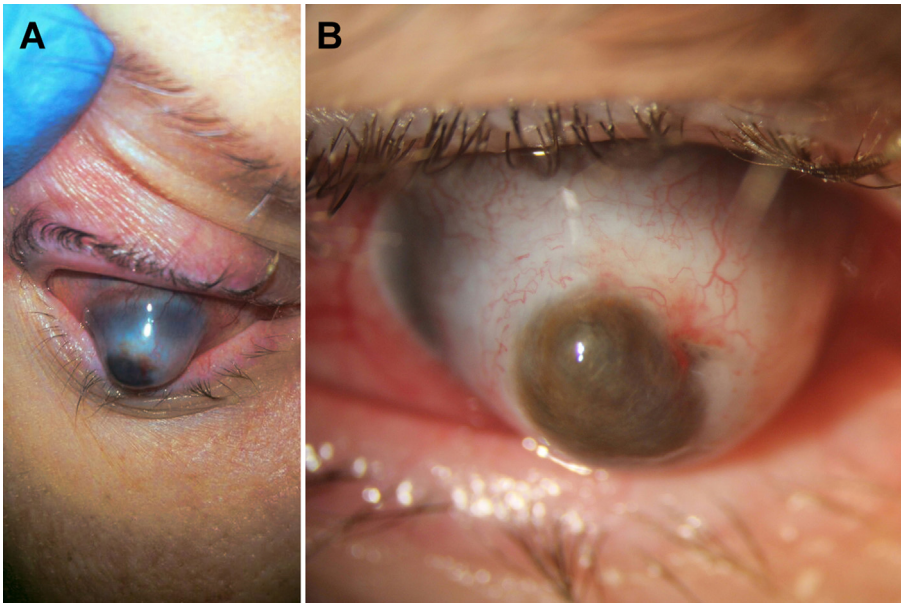

Fig. 1. (A) Macroscopic photograph of the right eye. (B) Slit-lamp image showing extensive corneal neovascularization, severe corneal melting, descemetocele and forward bulging of the iris.

wearing conventional hydrogel (omafilcon A) contact lenses (Proclear®; CooperVision Inc., Irvine, CA, USA), and had never consulted an ophthalmologist before. Asked about contact lens habits, she revealed she had been wearing them on a continuous basis (24 h a day) for the last 12 months, probably influenced by low self-esteem and social isolation.

On our initial examination, best corrected visual acuity (BCVA) was no light perception in her right eye and 20/40 in her left eye. Contact lens was still present in the left eye. Macroscopically, the right eye showed a conical cornea. Slit-lamp examination revealed conjunctival injection without mucopurulent discharge, extensive corneal neovascularization (4 quadrants) and severe keratomalacia with stromal melting and subsequent descemetocele formation (Fig. 1). Further examination of the left eye revealed conjunctival hyperemia, ciliary congestion, deep corneal neovascularization, stromal haze and non-specific endothelial precipitates associated with inflammatory cells in the anterior chamber (approximately 5–15 cells per field of 1 mm × 1 mm) (Fig. 2). No Bitot's spots were found in either eye. Corneal scraping revealed no microorganisms. Bacterial and fungal cultures were negative. Laboratory studies showed normocytic anemia and hypoproteinemia. Serum vitamin A level was low (<10 μg/dl; reference range, 30–120 μg/dl). No connective tissue disorders were identified.

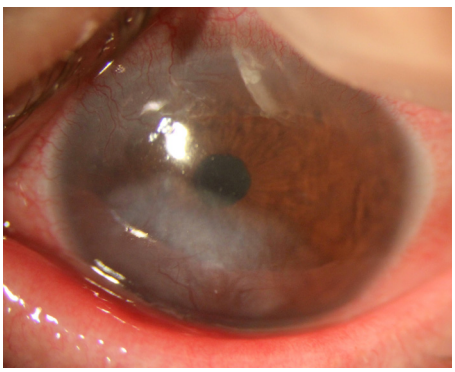

Fig. 2. Slit-lamp photograph of the left eye showing ciliary congestion, deep stromal neovascularization and stromal haze.

All available treatment options were considered and discussed with the patient. After informed consent was obtained, evisceration of the right eye was performed. The left eye was initially treated with moxifloxacin 0.5% eye drops (Vigamox®, Alcon Cusí SA, Barcelona, Spain) four times daily, prednisolone 1% eye drops (Pred Forte®, Allergan SA, Madrid, Spain) four times daily and cyclopentolate 1% eye drops (Colircusí Cicloplejico®; Alcon Cusí SA, Barcelona, Spain) twice daily.

3. Results

Histological examination of the cornea showed mild epithelial hyperplasia and microcysts, severe thinning of the central stroma, and deep stromal neovascularization and patchy lymphoplasmacytic infiltrates with absence of multinucleated giant cells at the peripheral cornea (Fig. 3).

One month after surgery, the patient reported subjective improvement of ocular discomfort. Conjunctival injection and anterior chamber reaction of the left eye subsided within 1 week of initiating treatment. Stromal haze and neovascularization remained. Prednisolone eye drops were tapered down and discontinued after four weeks of therapy. Final BCVA was 20/32 in the left eye.

4. Discussion

In subjects with severe nutritional disorders, vitamin A deficiency has been reported to induce keratomalacia and stromal melting [1]. This may explain to some extent the observed corneal changes in this patient. However, although vitamin A may induce disruption of corneal metabolism and keratocytes [4], keratomalacia is a complex phenomenon that usually arises from the converging action of several pathogenic processes.

Despite continuous advances in contact lens design, extended contact lens wear has been for long associated with a wide range of ocular surface and corneal disorders, such as dry eye, giant papillary conjunctivitis, epithelial erosions, infectious keratitis and stromal neovascularization [2]. Hypoxia is considered to be one of the major factors contributing to corneal disorders in contact lens