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Ultraviolet-A Light and Riboflavin Therapy for *Acanthamoeba* Keratitis: A Case Report

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Key Words

Acanthamoeba keratitis · Ultraviolet light · Riboflavin

Abstract

Purpose: To report ultraviolet-A (UV-A) light treatment in a patient with *Acanthamoeba* keratitis (AK).

Methods: Interventional case report. A standard protocol for ultraviolet corneal therapy, with a power emission of 3 mW/cm² and a wavelength of 370 nm, was used. The protocol included an 8-nm bandwidth at a 54-mm distance measured with a collimation system of diodes as well as a protective shield of riboflavin in a case of documented AK.

Results: A 54-year-old female patient with AK, showing no therapeutic response to a wide variety of topical antimicrobial agents and with a visual acuity of 20/400, was treated with UV-A therapy. The patient displayed a favourable response in the first 24 h after treatment, with improvement of symptoms, visual acuity (to 20/200) and biomicroscopy cornea with haze degree I. By the third week post-treatment, the patient was symptom-free. Her visual acuity was 20/30, and the affected cornea was clear. Five months after treatment, there had been no recurrence, and her vision was 20/20.

Conclusions: Treatment with UV-A light was an effective therapy in this case of AK.

Introduction

Since 1973, it has been recognised that *Acanthamoeba* may cause severe blinding keratitis. Early diagnosis and aggressive medical treatment using various combinations of specific antiprotozoal drugs has improved the possibility of successfully managing this corneal disease. However, penetrating keratoplasty may be found in cases that progress

despite maximal medical therapy and these cases can show evidence of severe stromal melting with imminent perforation [1].

Numerous studies demonstrate the efficacy of ultraviolet (UV) light for the inactivation of many types of microorganism, including protozoa [2–5]. With this in mind, a case of *Acanthamoeba* keratitis (AK) treated with UV-A therapy is reported.

Case Report

A 54-year-old female with a history of soft contact lens use and nail scratch trauma in her right eye was referred to our hospital after approximately 8 weeks of failed intensive oral and topical empirical therapy for presumed bacterial keratitis.

The patient reported severe pain, blepharospasm, photophobia, eye tearing and foreign body sensation. The visual acuity in her right eye was 20/400. The patient also suffered from hyperaemic conjunctiva and a large corneal paracentral epithelial defect (approximately 8 × 7 mm) with underlying dense anterior stromal infiltrate not involving the visual axis (fig. 1). Right-eye intraocular pressure was 12 mm Hg, with a normal posterior segment. In the left eye, the vision was 20/20, and the anterior and posterior segments were normal. Previous topical antimicrobial therapy was stopped for 24 h, and right corneal scraps were sent for new bacterial and fungal cultures. Negative Gram stain and cultures at 24, 48, and 72 h of incubation were reported. Anti-HSV-1 and 2 antibodies were also not detected. Corneal ulcer scrapings mounted on 10% potassium hydroxide revealed *Acanthamoeba* cysts, which coincided with a positive *Acanthamoeba* culture in *Escherichia coli*-enriched media. As an alternative to topical commercial treatment, which is not available in Mexico, the patient was offered corneal UV-A therapy and consented to this treatment.

Our patient was treated using the standard protocol for UV corneal therapy. After topical anaesthesia of the cornea (with eye drops of 0.1% tetracaine every 5 min for 15 min), the epithelium was removed within a 9-mm diameter zone that included all corneal infiltrates. After inserting a lid speculum, 0.1% riboflavin (Ricrolin; Sooft, Italy) drops were administered every 2.5 min for 30 min. Afterwards, the cornea was irradiated for 30 min with 3 mW/cm² UV-A, at a wavelength of 370 nm, from a VEGA-CSO UV light device (Firenze, Italy); the light was placed at a distance of 54 mm from the eye and was measured with a collimation system of diodes (LED) (fig. 2).

After the first 24 h, the outcome was favourable, with decreased symptoms, improved visual acuity (20/200) and a slit-lamp examination revealing partial resolution of the epithelial defect and stromal infiltrate with a mild corneal haze. At the third week after treatment, the patient's best corrected visual acuity was 20/30, and anterior segment exploration of her right eye showed no hyperaemic conjunctiva, a minimally hazy epithelium, and a clear chamber (fig. 3).

Discussion

Acanthamoeba is a ubiquitous, free-living protozoan that can cause severe keratitis. The outcome of AK has improved significantly over recent years because of earlier diagnosis and the introduction of effective amoebicidal drugs. The main treatment includes a combination of three classes of drugs: diamidine derivatives, imidazole derivatives, and aminoglycosides (topical and oral). However, toxic keratopathy may develop at any time, and these drugs are not readily available in every country. Although acanthamoebic organisms can be eradicated from the cornea by medical therapy in a great number of cases, severe inflammation and necrosis may spread to the paracentral corneal stroma or into the peripheral cornea, and this is an indication of penetrating keratoplasty. However, the prognosis after keratoplasty is sometimes uncertain, and medical therapy should be continued for at least several months to ensure elimination of

any residual *Acanthamoeba* cysts in the recipient tissue [1]. From this perspective, it is helpful to have another therapeutic option for treating infectious and melting keratitis that does not respond to broad-spectrum antibiotics.

We hypothesised that corneal UV therapy was a viable option in this case, mainly because there are no available diamidine and imidazole derivatives in commercial eye drops in Mexico. Soon after UV-A cross-linking treatment in our patient, the symptoms improved; the epithelial defects healed; and the infiltrate was smaller. UV-A therapy was effective for inactivation of AK by increasing resistance against enzymatic digestion and because of its inherent antimicrobial power.

First, UV light (wavelength 320–400 nm) permanently cross-links helical regions of DNA and RNA, inactivating a broad range of viruses, bacteria and protozoa. An inactivation level of 93.7% of some protozoan cysts, from species such as *Giardia lamblia* and *Cryptosporidium hominis*, has been demonstrated with a UV dose range of 3 to 40 mJ/cm² [2, 3]. Riboflavin easily diffuses into the stroma once the epithelial barrier has been broken, and *Acanthamoeba* cysts are killed as deep as 250–300 µm inside the stroma. Microbes located in the interface, which is usually located <200 µm deep, can be killed by the same process via the free radicals produced during cross-linking interfering with the microbial cell wall [4, 5].

Second, Spoerl et al. [6] found that cross-linking the cornea by using riboflavin and UV-A increases collagen resistance to digesting enzymes. Additionally, there are other reports that, like ours, describe a favourable outcome in AK treated with UV light and riboflavine [7].

For these reasons, we believe that UV-A therapy should be studied as an alternative treatment for selected cases of AK.

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Fig. 1. Ulcerative *Acanthamoeba* keratitis defect prior to UV-riboflavin therapy.

Fig. 2. Device used for the UV therapy.

Fig. 3. Right-eye corneal appearance 3 weeks after treatment, showing resolution of keratitis.

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