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Research Article

A CASE OF ACANTHAMOEBA KERATITIS: EVOLUTIONARY STAGES AND CHALLENGING DIAGNOSIS

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ABSTRACT

Resume: Acanthamoeba keratitis (A.K.) is a relativety uncommon but potentatially devastating coreal infection. It is usually missed.
The following case report describes a patient who developed radial keratitis transformed during evolution into nummular keratitis. The corneal scraping was negative. This made the diagnosis difficult.
An etiological investigation revealed that he had been hospitalized two months previously for gastroenteritis. The isolation of the entamoeba histolytica in the stool and the good evolution under antiamibian treatment are in favor of amoebic keratitis.
Clinical history, radial perineuritis and effecience of treatment are the capital items to evocate diagnosis of this clinical entity.

INTRODUCTION

Acanthamoeba is one of the most common free-living amoebae found in the environment [1]. It can cause serious human diseases such as potentially blinding keratitis and mortal encephalitis [2].

Acanthamoeba keratitis, a painful, progressive, sight-threatening corneal disease, was first reported by Naginton *et al*. [3] in the United Kingdom in 1974 and shortly thereafter by Jones *et al*. [2], in the United States in 1975. This often misdiagnosed disease is associated with trauma, exposure to contaminated water, and, more commonly, contact lens use [4].

Radial keratoneuritis has traditionally been felt to be pathognomonic for Acanthamoeba keratitis as the trophozoites often cluster around corneal nerves. It has also been described in pseudomonas keratitis [5].

The diagnosis of acanthamoeba keratitis is usually missed. It is an uncommon disease and none thought of in the differential diagnosis until weeks or months therapy for other pathogens.

The following case report describes a patient who developed radial keratoneuritis transformed during the course to nummular keratitis, making the diagnosis difficult.

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Case presentation

A 65-year-old man with no previous history of contact lens wear presented to our local ophthalmology department with a 20-day history of pain, redness, and blurred vision in his left eye. Prior to the presentation, he had started treatment with antibiotic eyesdrop and antiviral medications elsewhere (at a private eye hospital) as herpes pseudo-dendritic keratitis (fig. 1) without any improvement.

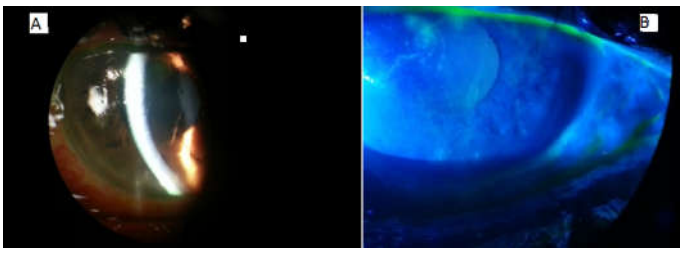

Figure1 Pictures token initially by the private eye hospital: A: multiple radial, linear, and branching anterior stromal infiltrates starting in the paracentral cornea with edematous stroma. B: After Fluo instillation: pseudo-dendritic central ulcer

At presentation, the best corrected visual acuity was 10/10 in the right eye and 1/10 in the left eye; the intraocular pressure was normal in both eyes. Slit lamp examination of the right eye was normal. The left eye had lid swelling, conjunctival injection, redness, with subepithelial infiltrates similar to nummular infiltrates (fig. 2). No corneal epithelial defects or any anterior chamber reactions were detected in the left eye. The rest of the eye examination, including the dilated fundus examination, was normal.

Figure 2 Subepithelial infiltrates (nummular infiltrates) appear after 20 days of viral treatment of radial neurokeratitis.

Corneal scarping was performed, and the culture for bacteria, fungal and Acanthamoeba infection were negative.

We noticed a Hypereosinophilic Syndrome at the CBC (complete blood count). Viral serology: herpetic, rubella, mononucleoside, acquired immunodeficiency and syphilitic serology, returned negative.

An etiological survey revealed that he was hospitalized two months before, for gastroenteritis, without definite etiology. This antecedent not mentioned by the patient at the initial interrogation, helps us a lot to suspected diagnosis. Indeed, we realized, a stool test that detected entamoeba histolytica.

Anti-*Acanthamoeba* treatment was initiated, which included metronidazole 750 mg orally 3 times a day, and tiliquinol-tilbroquinol for 10 days; in addition to a local treatment made by hexamidine, and a topical corticosteroid.

Although the exact etiology of this infectious keratitis was unclear, the patient subsequently has improved. A complete resolution of the infiltrate was achieved within 2 months with a persistence of a central corneal opacity, and no recurrence was detected in the first year of follow-up (fig. 3).

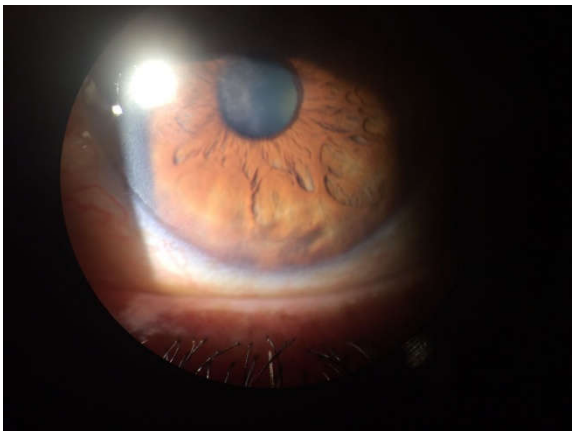

Figure 3 Complete resolution of the infiltrate was achieved within 2 months with a persistence of a paracentral corneal opacity

He achieved a final visual acuity of 6/10 at a recent follow up (2 Months after first presentation).

DISCUSSION

Acanthamoeba keratitis is an uncommon but potentially blinding corneal infection(1).

Clinical diagnosis of *Acanthamoeba* keratitis is based on the presence of keratitis that is accompanied by severe pain and photophobia, pseudodendriform epithelial lesions, stromal infiltrates, and less commonly, radial keratoneuritis. [6-7]

The clinical features of acanthammoeba keratitis are classified into three stages: initial, transient, and competed [6]. In the initial stage symptoms are redness, with severe pain. Corneal epithelium may be intact early in the course, or may have a pseudodendriform or rarely radial kertoneuritis. The next stage characterized by stromal infiltrates from partial or complete rings, or nummular keratitis. In the completed stage, appear corneal abscesses and central edema resembling to keratitis disciformis. It may evolutes to descemetocele associated with iritis and hypopion. This metamorphosis of the lesions occurring from one stage to another makes the diagnostic orientation difficult.

Many patients with Acanthamoeba keratitis are initially misdiagnosed and are treated for more common infections because the clinical characteristics of Acanthamoeba keratitis are very similar to more common forms of microbial keratitis [3]. If the response to a topical antibiotic, antiviral, or corticosteroid therapy produces an initial improvement or stabilization, the clinical picture may be modified, which in turn may complicate the diagnosis.

The patient in this case was initially misdiagnosed as having viral herpes keratitis with a pseudodendretic central ulcer and radial linear, anterior stromal infiltrates (radial keratoneuritis), after 20 days of viral treatment the clinical picture was modified to nummular keratitis

Importantly, in the initial stage, many cases were diagnosed as having HSV keratitis. Acanthamoeba keratitis can present with a dendritiform pattern of epithelial erosion that is frequently mistaken for herpes simplex viral keratitis (HSV) [7]. However, the dendritiform pattern of Acanthamoeba keratitis does not have the terminal bulbs seen in HSV keratitis[8]. Confusingly, both HSV keratitis and Acanthamoeba keratitis may have an initial positive response to topical antivirals [8]. Radial keratoneuritis is not pathognomonic for *Acanthamoeba* keratitis, and a similar appearance has also been reported in a case of *Pseudomonas* keratitis[5]. Although radial keratoneuritis is a very useful sign, it is not always present, especially late in the course of the disease.

Nummular keratitis is usually found in the second stage but can be observed in the first stage and in this case poses problem of distinguishing it from nummular keratitis to adenovirus [9].

Acanthamoeba keratitis must therefore be considered in all cases of presumed HSV or adenoviral keratitis in contact lens wearers. Features suspicious of AK include: contact lens contamination with tap water, poor response to topical antivirals, negative HSV polymerase chain reaction, radial perineuritis, and a ringshaped stromal infiltrate [in7].

The dysentery syndrome in the days preceding keratitis is another additional element to look for, allowing the suspicion of anthamoeba keratitis.

In our case the exact etiology of this infectious keratitis was unclear because of the negative culture of corneal scarping: the lesions could be due to metastatic infection of the trophozoites towards the cornea; or due to an immuno-allergic mechanism.

This latter mechanism is based on the significant association of phlyctenular keratoconjunctivitis with intestinal amebiasis [10] and the disappearance of corneal infiltrates by topical cyclosporin [9].

Acanthamoeba cysts and trophozoites are immunogenic. A study by Hussein AA et all [10] reported the role of parasitic infection in the etiology of phlyctenular eye disease. Stool examination and urine analysis of 150 cases of patients with phlyctenular keratoconjonctivitis, revealed: 115 cases (76.67%) had intestinal parasites.

In our case the oral treatment of the intestinal *amoebiasis associated to local treatment by hexomedine and* topical corticosteroid *improved the patient state,* and a complete resolution of the infiltrate was achieved.

The beneficial effect of this combination therapy, after a viral unsuccessful treatment suggests that amoebiasis is probably directly or indirectly responsible for this corneal disease.

CONCLUSION

Amoebic infection may be directly or indirectly responsible for chronic damage of the cornea. The diagnosis of Acanthamoeba keratitis can be easily missed because of the lesional metamorphosis seen during the course of this disease Clinical history, radial perineuritis and effecience of treatment are the capital items to evocate diagnosis of this clinical entity.

Conflict of interest: none.

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