

# A case of non-Acanthamoeba keratitis with radial keratoneuritis

Xiu Wang<sup>1</sup>, Patrick Glencer<sup>2</sup>, Rui-Hua Wei<sup>1</sup>

<sup>1</sup>Tianjin Medical University Eye Hospital, Tianjin 300384, China

<sup>2</sup>Nova Southeastern University College of Optometry, 2640 S University Dr #312 Davie, FL, USA

**Correspondence to:** Rui-Hua Wei. Tianjin Medical University Eye Hospital, No.251 Fu Kang Road Nankai District, Tianjin 300384, China. braveheart0717@sina.com

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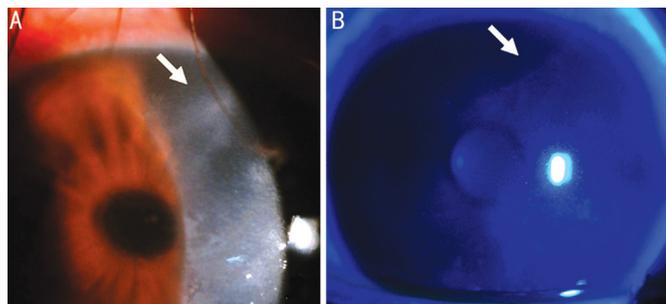
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## Dear Editor,

I am Dr. Xiu Wang, from Tianjin Medical University Eye Hospital, Tianjin, China. I write to present one case report of non-Acanthamoeba keratitis with radial keratoneuritis (RK). Keratitis that is caused by free living amoeba (Acanthamoeba) can cause devastating ocular damage. It is associated with trauma and contamination with water, soil, sewage, *etc*<sup>[1]</sup>. RK is pathognomonic for Acanthamoeba keratitis (AK) and is apparent in the early stages<sup>[2]</sup>. Because of this, RK is useful in identifying and diagnosing AK. This case report shows suspected AK because of the presence of RK. However, the laboratory examination, clinical progression, patient's symptoms, slit-lamp biomicroscopy and the effectiveness of the therapeutic drugs finally lead to the diagnosis of non-Acanthamoeba keratitis with RK.

A 22-year-old Asian male patient presented with eye redness, tearing, mild pain and difficulty opening in his left eye. Written informed consent was obtained from the patient for publication of this case report and any accompanying images. All symptoms in his left eye had occurred for two months. Symptoms worsened dramatically four days prior to being referred to Tianjin Medical University Eye Hospital. He stated his symptoms first occurred with an accompanying headache after a large consumption of alcohol. At a local county hospital, he was diagnosed with left ocular keratitis and was given topical levofloxacin and ganciclovir. After three days with no improvement, the treatment regimen was

changed to intravenous (I.V.) antibiotics. Three days after I.V. treatment, the patient's tearing symptom improved, but the discontinuation of therapy resulted in a relapse. I.V. antibiotics were resumed for an additional six days. Once again his tearing subsided, but after discontinuing the drugs the symptoms returned. He was then referred to our hospital for further evaluation. The patient reported no history of ocular trauma, contact lens wear, or exposure to contaminated water. Upon eye examination, visual acuity was 20/80 (OD) and 20/50 (OS). The intraocular pressures were within the normal range. The corneal sensation was decreased. Due to eye irritation, photophobia and other severe symptoms, the best corrected visual acuity (BCVA) was not checked. The right eye had no significant signs or symptoms. The left eye had no secretion, but had ciliary congestion temporally and the eyelid was swollen. A branch-like defect due to corneal inflammation was seen around the nerve. The area affected was seen traveling along the corneal nerves within the superior temporal corneal stroma at about 1 o'clock position. Flakes 3×4 mm<sup>2</sup> were seen in corneal stroma superficially. The invasion of cells could be seen partially at the temporal side of the central zone. The corneal stroma was mildly edematous and cloudy (Figure 1A). The corneal epithelium showed no sodium fluorescein staining (Figure 1B). The patient did not have any corneal deposits and the anterior chamber was clear of cells and flare. Iris texture was normal, the pupil was round (drug-induced mydriasis) and the lens was transparent. Corneal scrapings detected edematous epithelial cells and a small amount of inflammatory cells. No pathogenic microorganisms, amoebic cysts or trophozoites were found. Culture experiments were performed with various types of media and the results were all negative. Liver and kidney function tests, a complete blood count (CBC), prothrombin time (PT), and routine autoimmune tests came back negative without any conspicuous abnormalities. Due to the idiopathic nature of the corneal inflammation, the use of topical levofloxacin was continued. Topical pranopfen and artificial tears (*q.i.d*) were added at the beginning of treatment. The patient had a regimen of 100 mg oral acyclovir after breakfast five times a day. After three days, the patient had no significant changes in symptoms. Corneal staining showed that the corneal epithelium had been repaired, but the subepithelial infiltration was still apparent. The patient was placed on 0.1% fluorometholone (*b.i.d*). The following day, the patient's tearing



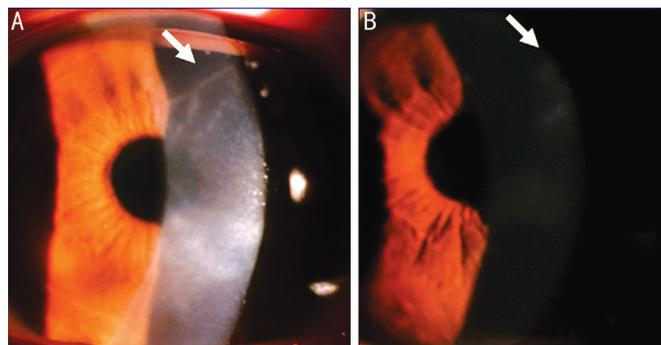
**Figure 1** There were radial keratoneuritis at presentation within the corneal stroma superior temporally about 1 o'clock and  $3 \times 4 \text{ mm}^2$  flake were seen in corneal stroma superficially (A); Left eye's corneal epithelium showed no sodium fluorescein staining (B).

symptoms improved. The application of fluorometholone was increased to *q.i.d* and the use of tobradex ointment was applied once a day at night. During the first five days, eye pain and tearing had diminished and both eyes were able to open freely. Visual acuity gradually increased to 20/32, with a BCVA to 20/20 (-1.75 DS). Partial temporal branching of infiltrates became thinner and the irregular flakes in the partial temporal were faded. Corneal edema was mitigating (Figure 2A). After using 0.1% fluorometholone *q.i.d* and tobradex ointment every night, the patient was treated with 0.1% fluorometholone once a day for another two months. After 28d of treatment, there were no signs of corneal edema and the branching of infiltrates was not obvious (Figure 2B). We have followed the patient for half a year and there was no recurrence.

Although RK is a very helpful sign, it is not always present, especially late in the process of AK. A study by Sun *et al*<sup>[3]</sup> reported an incidence of RK in 2 (10%) of the 20 patients with AK. In another study, Bacon *et al*<sup>[4]</sup> reported an incidence of RK at presentation of 57% among 36 eyes diagnosed within 1mo of the onset, declining to 29% among 24 eyes diagnosed after 2mo. Bernauer *et al*<sup>[5]</sup> summarized 70 cases of AK. Five cases showed signs of RK (7%). Bacon *et al*<sup>[4]</sup> examined 72 cases (77 eyes) with AK, nine of which were reported to have had RK (13%). At this present time there have a few other cases of non-Acanthamoeba keratitis with RK. Feist *et al*<sup>[6]</sup>, Roels *et al*<sup>[7]</sup> and Robbie *et al*<sup>[8]</sup> had reported that they diagnosed a RK in Pseudomonas keratitis.

Shinoda *et al*<sup>[9]</sup> have been reported a RK case with no evidence for any bacteria, fungi, or Acanthamoeba of these examinations, in that case, antifungal drugs were only used for a short period of time and the keratitis was cured. Mutoh *et al*<sup>[10]</sup> reported one case which was diagnosed first as AK based on the corneal smear, then was treated as Herpes simplex keratitis (HSK) due to lack of response to anti-Acanthamoeba treatment, and finally was treated with topical steroids. Kapoor *et al*<sup>[11]</sup> reported a case of fungal keratitis presenting as RK.

The patient, in this case, already had symptoms for two months prior to treatment. The paracentral of cornea showed turbidity



**Figure 2** After 4d of topical steroids treatment, the partial temporal branching of infiltrates became thinner and the irregular flakes in the superficial stroma were faded (A); After 28d treatment, corneal edema was missing and the branching of infiltrates was not as obvious (B).

consistent with the infiltrates which had invaded the stroma. In the late stage of AK a corneal opacity can appear and can coexist with a hypopyon.

Before the onset of symptoms, this patient had a history of heavy alcohol consumption, but no history of trauma or foreign bodies. His signs and symptoms included photophobia, lacrimation without mucopurulent secretion, and mild eye pain associated with an ipsilateral migraine. Confocal microscopy was left to be desired as our hospital is unequipped. Because of the negative corneal scrapings, the diagnoses of a bacterial, fungal and Acanthamoeba infection could be ruled out. The three main risk factors (use of contact lenses, contaminated water and corneal trauma) were not associated with this patient per the history given. No significant epithelial defects or ulcers were found clinically. Steroid treatment proved to be effective but the RK still remained. The patient had no autoimmune disorders, so any immunological condition that could cause keratitis may also be excluded. In this case, a virus should be considered given the fact that a virus can remain latent in human tissues for a long period of time. Given the human bodies' ability to promote homeostasis, a virus may not cause symptoms and remain in its dormant state. Once the immune system has been compromised, the virus may multiply and cause a reaction. The predisposing factors of this patient were a relatively large consumption of alcohol and fatigue. The fact that the other diagnoses were ruled out, the herpes viral infections that can cause corneal disruption should be considered.

Only one case report in the literature has been reported investigating viral keratitis associated with RK<sup>[10]</sup>. This case report may provide new ideas of RK analysis and differential diagnoses.

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**Conflicts of Interest:** Wang X, None; Glencer P, None; Wei RH, None.

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