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A Case Series of Acanthamoeba Keratitis in Singapore

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ABSTRACT

Acanthamoeba keratitis has become an important cause of severe ocular inflammation and visual loss in the past two decades. Its prevalence has been linked to the increasing use of contact lens. Early diagnosis, effective treatment regimes and education on proper contact lens wear are important in the management and prevention of visual loss from this debilitating disease.

We described a series of two cases of culture positive acanthamoeba keratitis and their subsequent management.

Keywords: Acanthamoeba Keratitis, contact lens, ring infiltrate

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INTRODUCTION

Acanthamoeba keratitis is a form of severe corneal inflammation caused by the protozoa Acanthamoeba, which belong to the order amoebida, suborder Acanthopodina, and family Acanthamoebidae. It is a free-living ubiquitous organism, which can exist in two forms. In favorable enviromental conditions, the Acanthamoeba exists in mainly the active trophozoite form, which moves with pseudopodium and phagocytoses small organisms that it encounters. In adverse enviromental conditions, the Acanthamoeba has the ability to encyst and the double walled cyst is resistant to desiccation, temperature extremes, damage by chemicals and limited food supply. Reproduction is through binary fission.

Acanthamoeba has been isolated from soil and dust, freshwater, seawater and air. Human contact with the organism is therefore inevitable and frequent through inhalation, swimming in lakes, swimming pools and hot tubs, domestic tap water and contact lens casing and solutions.

The rise in the prevalence of Acanthamoeba keratitis in the past two decades is probably related to the increase use of contact lens during this period as well as the increased awareness and recognition of this condition[1]. In Singapore, although there is no large-scale study into the epidemiology of this condition, it appears that the commonest cause is probably due to contamination of contact lens wear. This paper describes two cases of Acanthamoeba keratitis secondary to contact lens wear.

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Case 1

A 24 year-old Chinese man presented to us in Nov 1997 with symptoms of redness and pain of the left eye of 3 weeks duration associated with blurring of vision. He has been wearing daily wear non-disposable soft contact lens for less than ten hours per day for the past 2 years. He claimed to be compliant to the contact lens care regime with daily cleaning and regular deproteinising. He did not use home-made saline for contact lens cleaning. He denied contact with swimming pool water or lakes. On examination, he had a best-corrected Snellen visual acuity of 6/6 in the right eye and 6/12 in the left eye. The slit lamp examination of the left eye showed mild conjunctiva hyperemia, puntate epithelial erosions and stromal haze over the central cornea. There was no evidence of corneal ring infiltrate, perineural invasion or anterior chamber inflammation. He was diagnosed to have a resolving corneal abrasion and treated with ocular lubricants. His symptoms persisted and he developed anterior corneal stromal infiltrate with worsening stromal haze 1 month later during which he was started on gutt spersedexoline 1 drops q.i.d. His best corrected Snellen visual acuity was reduced to 6/18 (Fig. 1). Corneal specimen for culture was obtained by scrapping the clinically involved epithelium and stroma with a Kimura spatula. The corneal specimen was then plated onto a non-nutrient agar medium with E.coli overlay. The culture was positive for Acanthamoeba sp.

He was treated with gutt propamidine isethionate 0.1% 1 drop q.i.d and gutt Tobramycin 1 drop q.i.d. His best corrected Snellen visual acuity improved to 6/7.5 after 1 month of treatment. During the last review 3 months later, his best corrected Snellen visual acuity was 6/6 and examination revealed residual stromal scar with no inflammation.

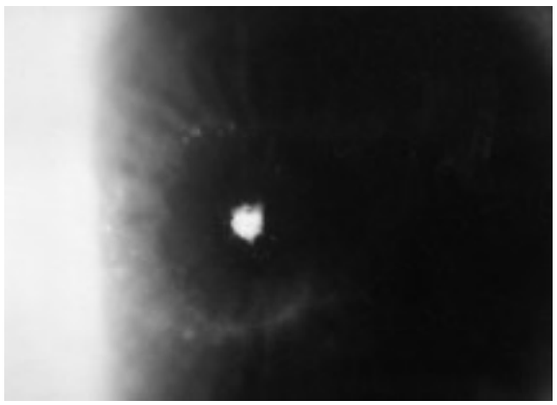

Fig. 1 Initial slit-lamp examination of Case 1 showing corneal epithelial and anterior stromal haze.

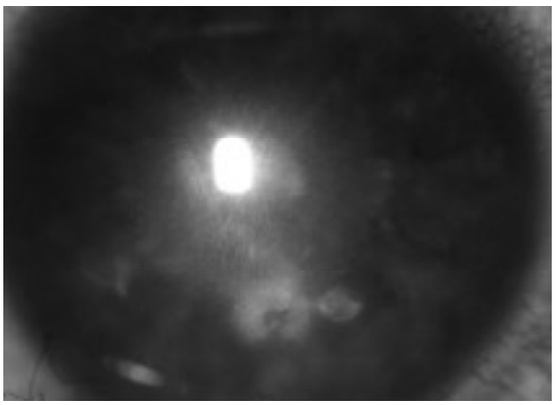

Fig. 2 Initial slit-lamp examination of Case 2 showing corneal epithelial and stromal infiltrate in a linear pattern.

Case 2

A 26-year-old Malay man presented to us with pain and redness of the left eye of 1 month duration. He was treated by a general practitioner with tetracycline ointment and neosporin eyedrops 1 week before the review. He was wearing daily-wear non-disposable soft contact lens before the onset of symptoms for 8 to 10 hours daily for 6 to 7 months. He claimed to be compliant to the recommended lens care regime. On ocular examination, his best-corrected Snellen visual acuity was 6/6 in the right eye and 6/9 in the left eye. Slit lamp examination showed epithelial and stromal infiltrate with punctate epithelial erosions (Fig. 2). A provisional diagnosis of Acanthamoeba keratitis was made. Corneal specimen for culture was similarly obtained by scrapping the clinically involved epithelium and stroma with a Kimura spatula. The corneal specimen was then plated onto a non-nutrient agar medium with E.coli overlay (Fig. 3, 4). Culture results were positive for Acanthamoeba sp. and the patient was started on gutt propamidine isethionate 0.1% 1 drop q.i.d. His pain and discomfort resolved after 1 week of therapy and the lesion healed with minimal stromal scarring after 2 weeks of treatment. His best-corrected visual acuity was 6/7.5 at the end of 1 month of treatment.

DISCUSSION

Acanthamoeba keratitis has been associated with a wide spectrum of clinical presentation. Most patients presented with symptoms of photophobia, pain, and tearing usually affecting one eye. The pain in acanthamoeba keratitis may be very severe and its intensity is disproportionate to the signs. Dendritic and pseudodendritic keratitis are also commonly described and may be mistaken for herpetic infections. Contact lens wear has been, increasingly implicated as the precipitating event. Home-made saline and inappropriate disinfecting regimes are responsible for the introduction of Acanthamoeba to the system[2]. In this series, both the patients had been using daily-wear non-disposable contact lens before the onset of symptoms.

The early clinical signs may be non-specific and comprises of punctate erosions, opacities, microcystic edema and anterior stromal infiltrates[3,4,5]. Moore[2] described the following helpful clinical signs and history which may suggest an early acanthamoeba keratitis: (1) previous treatment for herpes simplex keratitis with no improvement; (2) contact lens wearer with exposure to home-made solution, tap water rinse, well, intravenous saline; (3) pain out of proportion to the clinical findings; (4) epithelial irregularities or vesicular appearance in a pseudodendritic or linear pattern, epithelial haze, spherules, cystic lesions, refractile bodies, or recurrent epithelial erosions; (5) 0.5mm white, discrete anterior stromal opacities or patchy anterior stromal greyish white infiltrates forming a crescent or semicircular pattern. Perineural infiltration, which is the infiltration along corneal nerves although rare, is virtually pathognomic of Acanthamoeba keratitis[6]. As only one or two nerves are affected each time, a detailed slit lamp examination is necessary to identify these infiltrates. The accompanying anterior chamber reaction can range from trace cell and flare to hypopyon formation. A ring infiltrate usually with an accompanying epithelial defect may be present as well. Theodore, et al[7] described the annular keratitis as usually a 360 degrees infiltrate or abscess but sometimes only a partial ring may be present instead.

Extra-corneal involvement has also been reported. Sectoral or nodular scleritis with subsequent scleral ectasia can occur with severe disease. Burke JP, et al[8] described a case of acanthamoebic panophthalmitis with nodular anterior scleritis, iritis with cataract, marked retrolental inflammation with fibrosis and tractional retinal detachment and optic neuropathy.

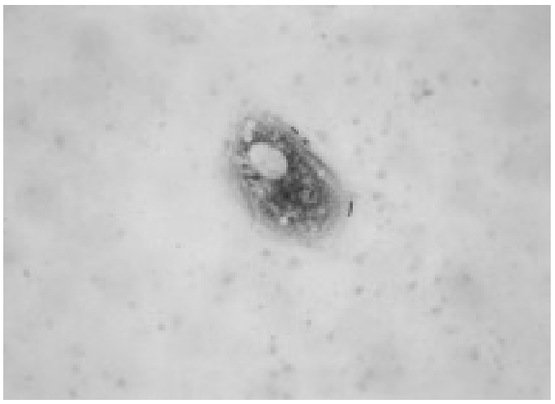

Fig. 3 Giemsa Stain of acanthamoeba cyst.

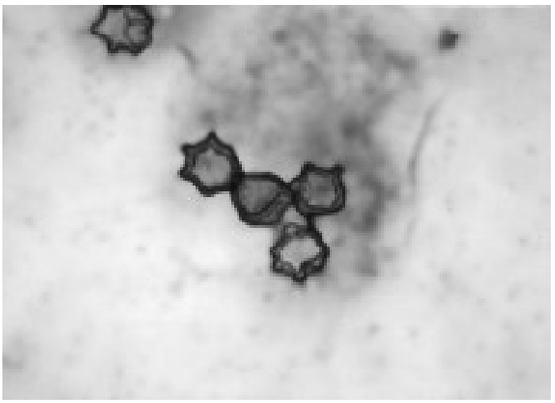

Fig. 4 Giemsa Stain of acanthamoeba trophozoite.

DIAGNOSIS

Laboratory confirmation of clinical suspicion should be instituted prior to instituting therapy. In this centre, corneal specimen for culture is obtained by scrapping the clinically involved epithelium and stroma with a Kimura spatula. The specimen is then plated onto a non-nutrient agar with Escherichia cold (E. Coli) overlay. The plate is then incubated at 32°C. If the amoebae were present they would phagocytose the E. Coli forming migratory tracks. These tracks are usually visible within 48 hours. Direct visualisation of the trophozoite or the double walled cysts are also diagnostic of the disease. Both the patients in this report were diagnosed using the above culture method (Fig. 3, 4). If the patient is a contact lens wearer, the lenses, contact lens case and the solutions should all be cultured as well.

Other culture methods have also been reported. The clinical specimen can be inoculated into a small sterile flat-bottomed tissue culture flask containing a shallow suspension of E. coli. This has the advantage of having the entire sample exposed to the bacteria. Any Acanthamoeba sp. present will rapidly settle and adhere to the base of the flask where they can be identified by a careful search using the inverted microscope[6].

Smears are examined for cysts and trophozoites with the chemofluorescent dye Calcofluor white. Viewed with a fluorescent microscope, cysts appear bright green and trophozoites appear bright orange. Viewed with an ultraviolet light, the chemofluorescent cysts appear white.

Other stains include Giemsa-Wright, Wheatley's trichrome, fluorescein-conjugated lectins, Gomori-methenatophenol cotton blue, PAS and electron microscopy. Thomas PA, et al[9] described the use of Lactophenol blue, a stain commonly used for preparing mounts for fungal cultures. He recommended the use of Lactophenol aniline blue stained mounts as a rapid, inexpensive, and simple demonstration of Acanthamoeba cysts in corneal ulcer scrappmgs.

TREATMENT

Medical

The mainstay of treatment of Acanthamoeba Keratitis is medical with the use of antiamoebicidal agents. The common antiamoebicidal agents used are as follows:

a. Propamidine isethionate 0.1%
b. Polyhexamethylene biguanide (PHMB)
c. Chlorhexidine 0.02%
d. Aminoglycide
e. Imidazole

Propamidine isethionate 0.1% was used as first line therapy for the two cases in this series as it has been found to be efficacious in the treatment of acanthamoeba keratitis[10]. It is an aromatic diamidine and is both trophocidal and cysticidal. In this series, propamidine is found to be highly effective and there was clinically resolved infection within one month of treatment. Combination regimes comprising of propamidine and miconazole 1% [4], propamidine with clotrimazole 1-2% and neomycin[11], has been tried with varying success.

Polyhexamethylene biguanide (PHMB), a cationic disinfectant has been found to be highly effective with consistently high cysticidal activity[12]. It also does not appear to have toxicity in contrast to propamidine[13], which is characterised by the presence of corneal epitheliopathy with punctate epithelial erosions. It may be used together with propamidine.

Brassseur, et al[14] reported that hexamidine has greater cysticidal activity as compared to propamidine. Chlorhexidine 0.02% has recently been used as an antiamoebicidal agent. Seal et al[15] reported good results with a combination of topical chlorhexidine and propamidine. The use of aminoglycoside and imidazole are usually as a combination therapy with propamidine.

Antibacterial agent has often been used together with antiamoebicidal therapy. Its main indications are to eliminate any bacteria that may infect the cornea either at the time of original amoebal infection or in a secondary infection. This may be especially important

in cases of persistent epithelial defect whereby there may be an increased risk of secondary bacterial infection.

Topical corticosteroids use in active Acanthamoeba keratitis is controversial. It may suppress host immune response, leading to prolonging of the course of disease and development of infectious crystalline keratopathy. It has been reported to inhibit both excystment and encystment in in-vitro studies.

Surgical Treatment

Penetrating keratoplasty is generally not indicated in acute inflammation except when there is corneal melt with consequent risk of perforation or extension of disease beyond the limbus. This is usually associated with high incidence of graft failure. The more common indication for penetrating keratoplasty is to improve vision in a previously infected eye with severe corneal scarring.

CONCLUSION

Acanthamoeba keratitis is a potentially sight threatening condition that is increasing more prevalent with the increased popularity in contact lens wear in Singapore as well as around the world. Successful management of this potentially devastating condition involves better patient education, effective, easy-to-use contact lens disinfecting systems, high index of suspicion and early accurate diagnosis and treatment of the condition.

REFERENCES

1. Moore MB, McCulley JP, Newton C, Cobo LM, Foulks GN, O'Day DM, et al. Acanthamoeba keratitis; a growing problem in hard and soft contact lenses wearers. Ophthalmology 1987; 94:1654-61.
2. Moore MB, McCulley. Acanthamoeba keratitis associated with contact lenses: six consecutive cases of successful management. Br J Ophthalmol 1989: 73:271-5.
3. Berger ST, Mondino BJ, Hoft RH, Donzis PB, Holland GN, Farley MK, et al. Successful medical management of acanthamoeba keratitis. Am J Ophthalmol 1990; 110:395-403.
4. Cohen EJ, Parlato CJ, Arentsen JJ, Genvert GI, Eagle J, Wieland MR, et al. Medical and surgical treatment of acanthamoeba keratitis. AM J Ophthalmol 1987; 103:615-25.
5. Key SN, Green WR, Willaert E, et al. Keratitis due to Acanthamoeba castellanii. A clinicopathologic report. Arch Ophthalmol 1980; 98:475-9.
6. Illingworth CD, Cook SD. Acanthamoeba Keratitis. Survey Of Ophthalmol 1998; 42:493-508.
7. Theodore FH, Jakobiec FA, Juechter KB, et al. Diagnostic value of a ring infiltrate in Acanthamoebic keratitis. Ophthalmology 1985; 92:1471-9.
8. Burke JP, Webber SK, Kerr-muir MG, Talbot JF, Parsons MA. Acanthamoeba polyphaga panophthalmitis. Cornea 1992; 11:274-5.
9. Thomas PA, Kuriakose T. Rapid detection of Acanthamoeba cysts in corneal scrapings by lactophenol cotton blue staining. Arch Ophthalmol 1990; 108-68.
10. Moore MB, McCulley JP, Luckenbach M, Gelender H, Newton C, McDonald MB, et al. Acanthamoeba keratitis associated with soft contact lens. Am J Ophthalmol 1985; 100:396.
11. D'Aversa G, Stern GA, Driebe WT. Diagnosis and successfu1 medical treatment of acanthamoeba keratitis. Arch Ophthalmol 1995; 113:1120-3.
12. Elder MJ, Kilvington S, Dart JK. A clinicopathologic study of in vitro sensitivity testing and acanthamoeba keratitis. Invest Ophthalmol Vis Sci 1994; 35:1059-64.
13. Johns KJ, Head WS, O'Day DM. Corneal toxicity of propamidine. Arch Ophthalmol 1988; 106:68-9.
14. Brasseur G, Favennec L, Perrine D, Chenu JP, Brasseur P, et al. Successful treatment of acanthamoeba keratitis by hexamidine. Cornea 1994; 13:469-2.
15. Seal D, Hay J, Morrell A, Booth A, Tullo A, Ridgway A, Armstrong M. Successful Medical therapy of Acanthamoeba keratitis with topical chlorhexidine and propamidine. Eye 1996; 10:413-21.