References

1. Flanagan JC, Stokes DP. Lacrimal sac tumours. Ophthalmology 1978;85:1282–7.
2. Pe'er JJ, Stefanyszyn M, Hidayat AA. Non-epithelial tumours of the lacrimal sac. Am J Ophthalmol 1994; 118:650–8.
3. Glaros D, Karesh JW, Rodrigues MM, Hirsch DR, Zimmerman LE. Primary malignant melanoma of the lacrimal sac. Arch Ophthalmol 1989;107:1244–5.
4. Economides NG, Page RC. Metastatic melanoma of the lacrimal sac. Ann Plast Surg 1985;15:244–6.
5. Ryan SJ, Font RL. Primary epithelial neoplasms of the lacrimal sac. Am J Ophthalmol 1973;76:73–88.
6. Duke-Elder S, editor. System of ophthalmology, vol 13. St Louis: CV Mosby, 1974:738.
7. Lloyd WC, Leone CR. Malignant melanoma of the lacrimal sac. Arch Ophthalmol 1984;102:104–7.
8. Eide N, Refsum SB, Bakke S. Primary malignant melanoma of the lacrimal sac. Acta Ophthalmol (Copenh) 1993;71:273–6.
9. Levine MR, Dinar Y, Davies R. Malignant melanoma of the lacrimal sac. Ophthalmic Surg Lasers 1996;27: 318–20.

Sir,

Acanthamoeba as a 'Transient' in the Corneal Scrape of a Poorly Compliant Soft Contact Lens Wearer with Peripheral Keratitis

Clinical diagnosis of *Acanthamoeba* keratitis should ideally be confirmed by the isolation of the protozoan from corneal tissue.[1] Prior to culture, the amoebae are often readily detectable using 'wet' preparations of corneal cells; smears can be stained for examination using bright field microscopy,[2] or alternatively can be visualised unstained using phase contrast.[3] Such screening of the corneal tissue permits initiation of rational antiprotozoal chemotherapy at a very early stage following diagnosis, and is especially helpful if the clinical picture is not typical.

The spectrum of clinical signs recorded during early stages of the infection process are varied[4] and these can often be confused with other aetiologies. A history of contact lens wear, particularly in a younger person, may increase the index of suspicion of an *Acanthamoeba* infection.[5] In such patients, however, laboratory isolation of *Acanthamoeba* should not be regarded as the sole criterion for diagnosis, since it is likely that in certain circumstances the protozoan may be present in the tear film components but not as a contributor to any ocular disease process. We present here such a patient, who presented with a peripheral ulcer and in whom *Acanthamoeba* was isolated from the corneal scrape, but was not considered to have been the cause of the keratitis.

Case Report

A 24-year-old male medical student, using soft contact lenses to correct mild myopia, presented to an ophthalmic casualty unit in Glasgow, with a 48-hour history of discomfort and mild photophobia in the left eye. Twenty-four hours prior to presentation his contact lenses were removed and 4 drops of chloramphenicol (0.5% w/v, 1 drop, 4 times per day, for 1 day) applied to the eye. At presentation, visual acuity with spectacles was 6/6 + 4 for the right eye and 6/6 − 1 for the left eye. The left eye showed mild circumcillary injection and, at about 5 o'clock, a 1 mm diameter peripheral corneal ulcer with epithelial defect (Fig. 1). An occasional cell was observed in the anterior chamber.

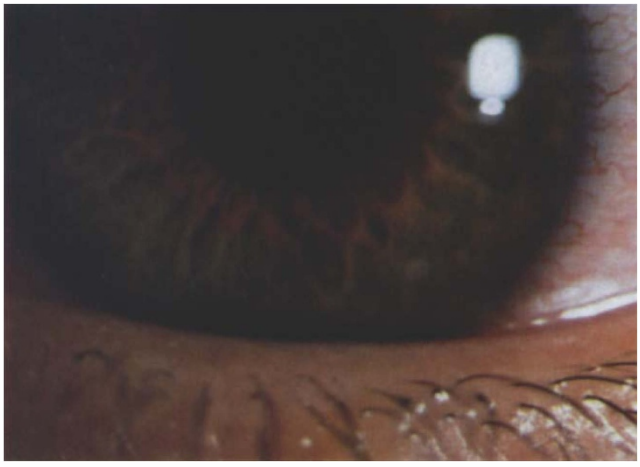

Fig. 1. *The left eye at presentation showing a peripheral ulcer 1 mm in diameter.*

The ulcer was scraped using a 23 gauge needle and smeared onto a glass slide; tissue was then plated onto blood agar. Although the clinical appearance was not reminiscent of *Acanthamoeba* keratitis, it was nevertheless considered that since the patient was a young contact lens wearer, tissue should be examined to exclude unequivocally the protozoan as a contributor to the pathogenesis of the disease. The needle with associated corneal scrape tissue was placed into a plastic centrifuge tube containing 1 ml defined axenic medium[6] and aliquots (0.05 ml) removed for microscope examination, prior to culture.

The Gram-stained smear was reported by the routine microbiology laboratory to contain a single polymorphonuclear cell; no bacteria or other organisms were recorded. Despite this, topical drop treatment was commenced with a combination of gentamicin (0.3%, hourly by day), ciprofloxacin (0.3%, hourly by day) and cyclopentolate (1%, 3 times/day). Twenty-four hours later there was no epithelial defect, a residual subepithelial haze was present but cells were not discerned in the anterior chamber. The medications were continued 2-hourly by day for 4 days, after which the clinical signs had improved considerably. The visual acuity at this time was 6/5.

Scanty growth of bacteria was subsequently recorded from the blood agar plate. After incubation

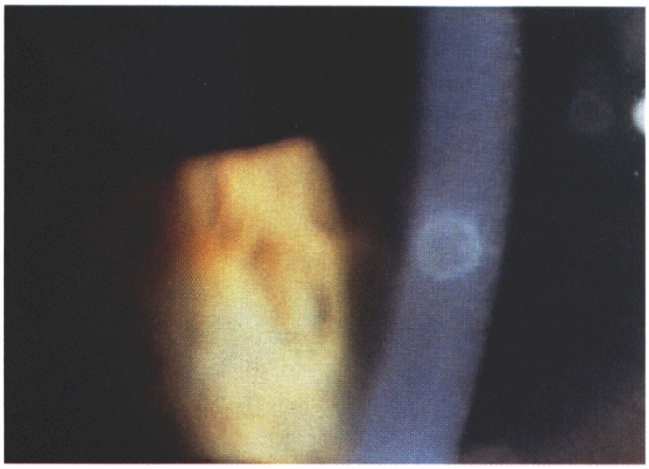

Fig. 2. *The left eye following treatment: the epithelial ulcer has resolved.*

in an enrichment medium, coagulase-positive *Staphylococcus aureus* was isolated. There was an unfortunate delay in receipt of laboratory findings for *Acanthamoeba*, which had been detected on microscope examination of the corneal scrape sample and subsequently confirmed by culture findings. On the basis of these latter observations, the treatment was altered to the combination[7] of chlorhexidine (0.02% w/v) and propamidine (0.1% w/v, as Brolene), administered alternately at 2-hourly intervals for 2 weeks, followed by 4 times by day for 4 weeks, by which time there was complete resolution of clinical signs and symptoms (Fig. 2).

Discussion

The daily-wear disposable soft contact lenses worn by the patient had been purchased some 3 years earlier. With the exception of the initial optometric consultation, there had been no further education regarding lens care and no follow-up ocular examination. The lenses were routinely worn for 12–16 hours per day, 7 days a week. The patient had used various hydrogen-peroxide-containing lens disinfecting solutions. At the time of presentation he claimed to be using a one-step peroxide system. The lenses were not subjected to mechanical cleaning and no protein remover had been used for at least 1 year. He had often used the same disinfection solution for his lenses over several nights in the mistaken belief that it retained its antimicrobial efficiency.

The clinical presentation in this patient and his symptoms were not characteristic of *Acanthamoeba* keratitis. Thus, it is likely that the protozoa detected from the corneal scrape were 'transient' in the vicinity of the peripheral infiltrate, and not actually contributing to the ulcerative process. The *Acanthamoeba* were, in all likelihood, transferred to the ocular surface by the contact lenses from the contaminated storage case; the fluid content of the storage case was replete with viable *Acanthamoeba*, and many of the protozoa were observed to be directly associated with the contact lenses. The patient denied use of tap water as part of his lens wear routines, but it is likely that this was the source of the contamination.[8,9]

Previously, we have observed 2 patients in whom keratitis-causing free-living amoebae appeared to be present on the ocular surface but not contributing directly to the disease process. One of these patients had been self-administering Brolene for a minor eye problem.[10] The other was detected during routine examination of contact lens paraphernalia and home water supply of controls for our Scottish Microbial Keratitis Study,[11] where contact lenses were removed from the eyes by the wearer to be sampled in the home.

It is our contention, based on the case described, that *Acanthamoeba* may be transient on the ocular surface, and be found in association with a peripheral infiltrate, without causing any obvious signs or symptoms of an amoebal infection. Laboratory detection of the *Acanthamoeba* may confuse the diagnosis in this situation, particularly if the clinical appearance is not typical of this infection.

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References

1. Seal DV, Hay J, Kirkness CM, Morrell A, Booth A, Tullo A, *et al.* Successful medical therapy of *Acanthamoeba* keratitis with topical chlorhexidine and propamidine. Eye 1996;10:413–21.
2. Thomas PA, Kuriakose T. Rapid detection of *Acanthamoeba* cysts in corneal scrapings by lactophenol cotton blue staining. Arch Ophthalmol 1990;108:168.
3. Hay J, Kinnear F, Kirkness CM, Seal DV. *Acanthamoeba* keratitis: laboratory diagnosis, characterisation of protozoa and treatment. Scottish Centre Infect Environ Health 1995;29(95/17):90–1.

4. Bacon AS, Dart JKG, Ficker LA, Matheson MM, Wright P. *Acanthamoeba* keratitis: the value of early diagnosis. Ophthalmology 1993;100:1238–43.
5. Radford CF, Bacon AS, Dart DKG, Minassian DC. Risk factors for *Acanthamoeba* keratitis in contact lens users: a case control study. BMJ 1995;310:1567–70.
6. Hay J, Kirkness CM, Seal DV, Wright P. Drug resistance and *Acanthamoeba* keratitis: the quest for alternative antiprotozoal chemotherapy. Eye 1994;8: 555–63.
7. Kirkness CM, Hay J, Seal DV, Aitken D. *Acanthamoeba* keratitis. Ophthalmol Clin North Am 1994;7: 605–16.
8. Seal DV, Hay J, Munro FA. *Acanthamoeba* keratitis: a waterborne disease. Br J Optom Disp 1994;2:475–9.
9. Ledee DR, Hay J, Byers TJ, Seal DV, Kirkness CM. *Acanthamoeba griffini*: molecular characterisation of a new corneal pathogen. Invest Ophthalmol Vis Sci 1996; 37:544–50.
10. Kinnear FB, Hay J, Montgomery D, Seal DV. Abuse of Brolene eye drops with putative corneal infection. J Infect 1996;32:172–4.
11. Seal DV, Hay J, Kirkness CM, West of Scotland Keratitis Study Group. Incidence study of *Acanthamoeba* keratitis in the UK. OMIG, Chicago, October 1996.

Sir,

Metastatic Adenocarcinoma Presenting as an Isolated Ciliary Body Tumour

A 69-year-old man presented to the ophthalmology outpatient department complaining of a 3-month history of burning pain in his left eye. On examination visual acuity was 6/6 in both eyes. There was a brownish mass on the temporal iris, which, on gonioscopy, infiltrated the angle from 2 to 5 o'clock (Fig. 1). Intraocular pressure was 18 mmHg, right and left. On dilating the pupil there was a large mass in the ciliary body, which on ultrasound measured 14 mm × 14 mm with a thickness of 5 mm.

A presumptive diagnosis of malignant melanoma of the ciliary body was made. The possibility of a metastatic lesion was also considered. The patient was systemically well with no other signs or symptoms.

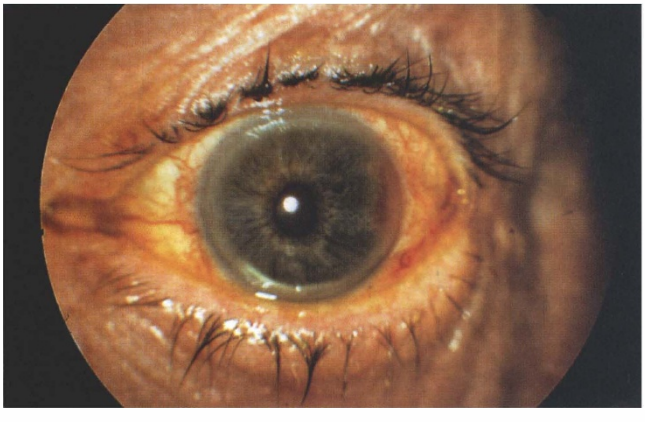

Fig. 1. *The left eye shows a brownish mass lesion on the temporal iris extending from 2 to 5 o'clock.*

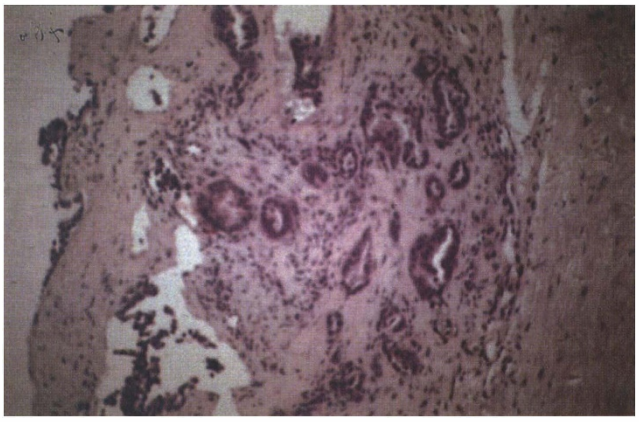

Fig. 2. *Pathology of the lesion reveals metastatic adenocarcinoma.*

A metastatic screen was negative. An excision biopsy of the lesion was performed. Local ruthenium plaque radiotherapy was then applied for 4 days, delivering a dosage of 11 400 cGy to a depth of 5.5 mm. Pathology revealed a mucin-secreting adenocarcinoma of likely lung or gastrointestinal origin (Fig. 2). A systemic investigation was negative for a primary lesion. The patient received a palliative course of chemotherapy. Clinically he deteriorated rapidly. He died 12 weeks later from pulmonary metastases, which were not present on initial examination. Permission for a post mortem was not obtained.

Discussion

This is a case report of a metastatic adenocarcinoma which presented as an isolated primary ciliary body tumour, for which no primary site was identified. Metastases to the iris are usually diffuse. Reported incidence of metastases to the eye varies between 4.9% and 7%.[1,2] The iris and ciliary body is a less common site and is involved in approximately 7.8% of cases metastasising to the eye.[3] The posterior choroid is most frequently involved – reportedly in 80% of cases.

Metastases to the eye generally present after the primary tumour has been identified. Unknown primary sites account for 11% of metastases to the eye.[4] Prognosis is poor with metastases, regardless of the origin of the primary lesion, median survival time being 13 months after diagnosis of iris metastases.[3]

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