24. Russell AD. Chlorhexidine: microbicidal action and bacterial resistance. Infection 1986;14:212–5.
25. Nisbet IT, Graham DM, Spicer PE, Tibbs GJ. Chlorhexidine is an effective agent against *Chlamydia trachomatis in vitro* and *in vivo*. Antimicrob Agents Chemother 1979;16:855–7.

Sir,

Fungal Keratitis Caused by *Curvularia lunata*, with Successful Medical Treatment

Fungal keratitis is exacerbated by topical steroid use.[1] We present a case of a non-resolving corneal ulcer, treated with topical antibiotics and betamethasone. The ulcer was found to be infected by *Curvularia lunata*. This has not previously been described in this country although it is a recognised cause of keratomycosis in tropical and subtropical areas.

Case Report

A 53-year-old man was referred in December 1995 with a month's history of a non-resolving right corneal ulcer. His symptoms had developed in Israel while swimming in the Dead Sea, when he developed a painful and red right eye. He was otherwise well. In 1993 he had undergone surgery for right congenital ptosis. An ophthalmologist in Israel diagnosed a corneal ulcer and started treatment with topical antibiotics.

On his return to England a week later he consulted an ophthalmologist again. The corneal ulcer was treated with hourly topical ciprofloxacin 0.3% and gentamicin 1.5%, and betamethasone 0.1% four times a day. Corneal scrapes were negative for bacterial culture. After 3 weeks without clinical improvement, he was referred to the corneal clinic at the Croydon Eye Unit for further management.

At that time his visual acuity was 6/12 in the right eye and 6/9 in the left. The conjunctiva was injected. There was an area of peripheral corneal ulceration at 4 o'clock measuring 2.5 mm × 1.5 mm. This was associated with overlying hard plaque formation and surrounding infiltration and corneal melting (Fig. 1). There was minimal anterior chamber activity and the left eye was normal.

A Gram stain of corneal scrapings demonstrated some fungal elements, confirming the clinical picture of fungal keratitis. The day after stopping the patient's medication, further scrapings were taken for inoculation on blood, chocolate and two Sabouraud agar plates. Treatment of the ulcer was then initiated with topical natamycin 5% (Pimaricin) hourly; 2-hourly gentamicin 1.5% was continued. The blood and chocolate plates were inoculated in CO_2 at 37 °C overnight; one Sabouraud plate was incubated at 37 °C and the other at 30 °C. Dark brown velvety colonies grew on the chocolate and on the Sabouraud plates incubated at 37 °C after 2 days. These were identified as *Curvularia* species by the distinctive curved macronidia. Bristol Public Health Laboratory Services confirmed the fungus as *C. lunata* and found it to be sensitive to amphotericin B, itraconazole and clotrimazole (Pimaricin was not available for testing).

The corneal ulcer slowly resolved and the gentamicin was discontinued. After 1 week, tetracyline 250 mg orally q.i.d. was added for 2 weeks. The area of infiltration decreased in size over 3 weeks and topical medication gradually tapered off. Four weeks after the initiation of antifungal treatment the epithelial defect had healed completely. There was a residual area of stromal thinning with underlying vascularisation (Fig. 2). The patient's final right visual acuity was 6/12.

Discussion

Fungal keratitis is rare in the United Kingdom, accounting for 3% of suppurative keratitis presenting to Moorfields Eye Hospital in 1981.[2] The incidence is much higher in tropical or subtropical regions and is related to climatic conditions and agrarian popula-

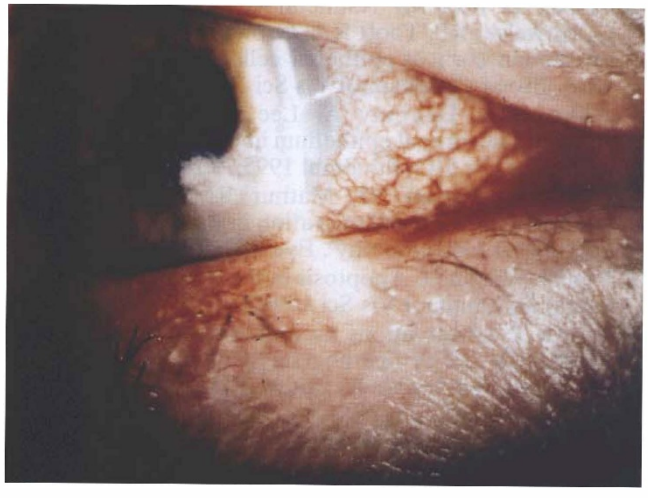

Fig. 1. *The right eye at presentation, showing corneal ulceration at 4 o'clock.*

Fig. 2. *The right eye 4 weeks after initiation of antifungal treatment, showing complete healing of the epithelial defect.*

tions. In these regions predominantly filamentous fungi rather than yeasts cause fungal keratitis. More than 70 species belonging to 40 genera have been reported as causes of keratitis.[1] *Aspergillus* species were the most common in Saudi Arabia,[3] India,[4] and Bangladesh,[5] followed by *Fusarium* species, *Curvularia* species (6–8%) and *Candida* species. In Miami *Fusarium* species are the commonest.[6] There may be a history of agricultural trauma or washing in contaminated water.

In Northern climates fungal keratitis is less common and yeasts are more frequent than filamentous fungi. Often patients present who have already been treated unsuccessfully with topical antibacterial agents and corticosteroids. Clinical features of fungal ulcers include a dry, raised, necrotic or fluffy surface. They may be associated with endothelial rings.[5] Topical medication should be stopped for 24–48 hours prior to rescraping. Staining by Gram stain is highly sensitive[7] for hyphae and spores, and calcofluor white staining has also been used.[8] Direct inoculation of a Sabouraud plate is also reported as a method of isolation.[9]

Curvularia species are filamentous fungi, present as saprophytes in soil and air as well as on plants. Keratomycosis can be caused by *C. lunata*, which has been isolated from corneal ulcers in tropical and subtropical areas, where it accounts for 6% of mycotic ulcers.[1] In addition *C. geniculata*, *C. pallescens*, and *C. senegalensis* have been isolated from eye infections and there is one report of *C. brachyspora* as a cause of mycotic keratitis.[10]

Curvularia keratitis has been successfully treated with amphotericin B and Pimaricin 5% solution.[7] In this country no previous cases of *Curvularia* keratitis have been reported, although it is well recognised in subtropical and tropical areas. In this case there was a history of foreign travel and exposure to water in the Dead Sea. In the absence of trauma, the patient may have been predisposed to infection because of previous ptosis surgery. This case emphasises the importance of prompt diagnosis in recognising and identifying fungal infection, as antifungal agents will minimise visual morbidity. We would discourage the use of corticosteroids without an established microbiological diagnosis.

It is important to establish whether there is a history of foreign travel, as this may be indicative of fungal aetiology in corneal ulcer.

S. E. Dorey
W. H. Ayliffe
C. Edrich
Department of Ophthalmology
Mayday University Hospital
Croydon
Surrey CR7 7XN
UK

D. Barrie
Department of Microbiology
Mayday University Hospital
Croydon
Surrey CR7 7XN
UK

P. Fison
Department of Ophthalmology
St Helier NHS Trust
Sutton Hospital
Surrey SM2 5NF
UK

References

1. Thomas PA. Mycotic keratitis: an underestimated mycosis. J Med Vet Mycol 1994;32:235–56.
2. Coster D, Wilhelmus K, Peacock J, Jones B. Suppurative keratitis in London. In: VI Congress of the European Society of Ophthalmologists 1981;395–8.
3. Khairallah SH, Byrne KA, Tabbara KF. Fungal keratitis in Saudi Arabia. Doc Ophthalmol 1992;79:269–76.
4. Chander J, Sharma A. Prevalence of fungal corneal ulcers in northern India. Infection 1994;22:207–9.
5. Dunlop AA, Wright ED, Howlander SA, Nazrul I, Husain R, McClellan K, Billson FA. Suppurative corneal ulceration in Bangladesh: a study of 142 cases examining the microbiological diagnosis, clinical and epidemiological features of bacterial and fungal keratitis. Aust NZ J Ophthalmol 1994;22:105–10.
6. Liesegang TJ, Forster RK. Spectrum of microbial keratitis in south Florida. Am J Ophthalmol 1980;90:38–47.
7. Forster RK, Rebell G, Wilson L. Dematiaceous fungal keratitis. Br J Ophthalmol 1975;59:372–7.
8. Arffa RC, Avni I, Ishibashi Y, Robin J, Kaufman H. Calcofluor white and ink potassium hydroxide preparations for identifying fungi. Am J Ophthalmol 1985;100:719–23.
9. Armstrong M. The laboratory investigation of infective keratitis. Br J Biomed Sci 1994;51:65–72.
10. Marcus L, Vismer HF, Van der Hoven HJ, Gove E, Meewes P. Mycotic keratitis caused by *Curvularia brachyspora.* Mycopathologia 1992;119:29–33.

Sir,

A Case of Central Retinal Artery Occlusion after Carotid Endoarterectomy

Occlusion of the central retinal artery is a well-documented entity that can be caused by trauma, thrombosis, vascular spasm or embolism.[1,2] In most cases resulting from emboli, these arise from ulcerated atheromatous plaques along the walls of the common or internal carotid arteries.[1,3]

This is the first report of central retinal artery occlusion following carotid endoarterectomy.

Case Report

A 65-year-old man was referred to the Division of Vascular Surgery of the University of Rome 'Tor