

Fig. 2. Resolution of cotton wool spots 2 months later.

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N. Muruganathan ✉
 J. Vodden
 R.H. Gray
 Ophthalmology Department
 Taunton and Somerset NHS Trust
 Taunton
 Somerset TA1 5DA, UK

Sir,

Mycobacterium marinum keratitis: pigmentation a clue to diagnosis

To our knowledge there has only been one reported case of *Mycobacterium marinum* keratitis to date.¹ We present the second such case. The unique features of this case are the absence of antecedent ocular trauma, or underlying ocular pathology, and a good visual result.

Case report

A 65-year-old man was referred with a 6 week history of a foreign body sensation affecting his left eye. He had been unsuccessfully treated by his local ophthalmologist with gentamicin 0.3% drops and prednisolone acetate 1%. The patient denied a history of ocular trauma despite having been cleaning the barnacles off his boat several weeks prior to the onset of symptoms.

On examination, his unaided visual acuities were right 6/9–2 and left 6/12, improving to 6/7.5 with a pinhole. The anterior segment on the right was normal. On the left there was a corneal ulcer, measuring approximately 4 mm by 7 mm. Numerous mutton fat and pigmented keratic precipitates were seen inferiorly. There was 2+ cells in the anterior chamber. Intraocular pressure was 26 mmHg by pneumotonometry. Epithelial nodules were noted in the adjacent paralimbal area. General physical examination was unremarkable. No unusual skin lesions were noted.

Topical therapy was changed to g. tobramycin and g. atropine. A corneal scraping was taken and sent to the laboratory for microscopic examination and culture. Microscopy revealed necrotic corneal stroma, numerous Gram-positive cocci and pleomorphic Gram-positive bacilli. A decision was made not to alter the antibiotics

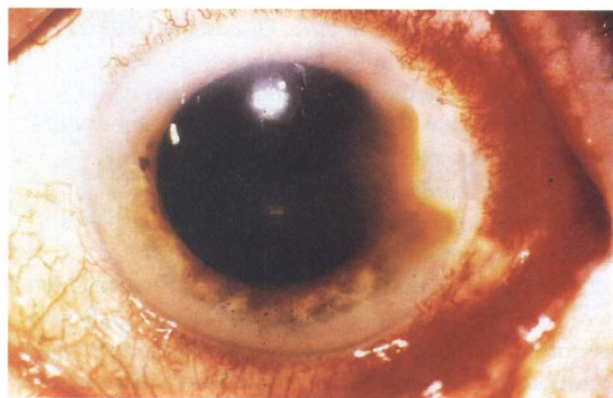


Fig. 1. Yellow pigmentation is seen adjacent to the ulcer.

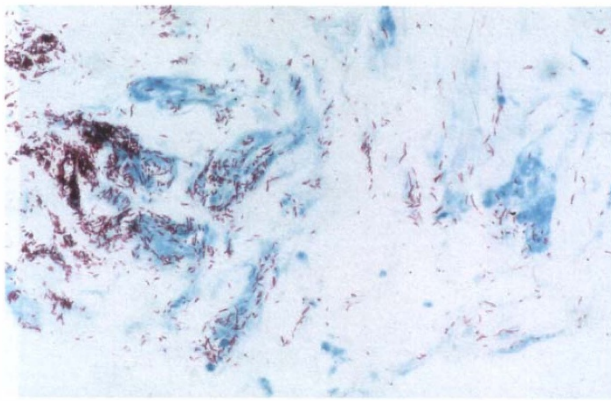


Fig. 2. Numerous acid-fast bacilli are seen in the stroma.

until culture results were available. Cultures, however, remained negative 4 days after the initial sampling; therefore topical therapy was stopped and a further biopsy was obtained.

Despite therapy the keratitis progressed and the vision dropped to counting fingers. A yellow pigmentation of the cornea adjacent to the ulcer was noted (Fig. 1). The patient was admitted to hospital and started on topical g. atropine 1%, g. timolol 0.5% (intraocular pressure had risen to 32 mmHg), and intensive g. gentamicin 1.5% and g. vancomycin 5%. Systemic antimicrobial therapy consisted of vancomycin 1 g i.v. b.i.d. and gentamicin 320 mg i.v. o.d. Vancomycin and gentamicin were, at the time, the standard parenteral antibiotics for the treatment endophthalmitis and severe keratitis. Nine days after initial biopsy an atypical mycobacterium was identified. The sample was then sent to the State Health Laboratory for further speciation. Systemic therapy was altered to amikacin and cefoxitin from vancomycin and gentamicin on empirical grounds. The combination of amikacin with cefoxitin was chosen as identification of the specific species of atypical mycobacteria can take weeks and this particular combination has been found to be effective against some of the most resistant atypicals seen in Queensland: *Mycobacterium chelonae* and *Mycobacterium abscessus*. The topical antimicrobials were changed to parallel systemic therapy. The base of the ulcer became quite thin, necessitating the use of tissue adhesive.

Mycobacterium marinum was finally identified 19 days after initial sampling. Sensitivity to amikacin and cefoxitin was confirmed *in vitro*. Discussion with the infectious diseases team led to a change in systemic therapy to i.v. trimethoprim/sulfamethoxazole and ciprofloxacin orally. This combination of antibiotics has been found to be particularly active against *Mycobacterium marinum* and achieves a high concentration in ocular tissues.² Topical therapy was continued unaltered.

No apparent resolution of the keratitis occurred after 4 weeks of intensive therapy. A corneoscleral graft was therefore undertaken. Histological examination revealed ulceration and necrosis of the cornea, acute inflammatory cells, and numerous acid-fast bacilli in the stroma (Fig. 2).

The graft was clear with no evidence of recurrence at last follow-up 3.5 years post-keratoplasty. Cataract extraction and lens implantation has also been performed. The patient's most recent visual acuity is 6/18.

Discussion

Runyon³ grouped the atypical mycobacteria into four types. Type I are photochromogenic, i.e. they produce a yellow pigment when grown in light. Type II are scotochromogenic, i.e. they produce a pigment when grown in the dark. Type III are non-chromogenic, i.e. they do not produce any pigment. Type IV are rapidly growing mycobacteria which can grow on culture, at room temperature, within 3–5 days. *Mycobacterium marinum* was first identified by Aronson in 1926 whilst studying fish.⁴ It was linked to skin infections in humans by Linell and Norden in 1951.⁵ The infections stemmed from inadequately chlorinated swimming baths, hence the name *Mycobacterium balnei*. *M. marinum* is now the preferred name.

Mycobacterium marinum grows best on culture between 30 °C and 32 °C in 8–14 days. When grown in the dark the colonies are non-pigmented; with light exposure they are bright yellow. The colour results from carotenoid production.⁶

Our case of *Mycobacterium marinum* keratitis differs from that reported by Schonherr *et al.*¹ in that mycobacteria were identified and treated with specific antimicrobials before tectonic penetrating keratoplasty was undertaken. The patient reported by Schonherr *et al.* underwent surgery before the specific agent had been identified. Three months later the patient developed infiltrates around the sutures. Biopsy of those areas demonstrated numerous acid-fast bacilli. The corneal button was then re-examined and found to have numerous acid-fast bacilli. The patient was due to undergo further surgery but the final visual result was not disclosed.

Retrospectively, the granulomatous nature of the inflammation and the bright yellow pigment may have aided in establishing an earlier diagnosis. Gram-positive bacteria were seen on the initial Gram stain and yet cultures remained negative. Subsequent Ziehl-Neelsen staining of the initial biopsies confirmed the presence of mycobacteria. When confronted with an indolent infection the clinician must consider slowly growing organisms, e.g. fungi and mycobacteria, in the differential diagnosis. This is particularly so given increasing travel to exotic places and the numbers of immunosuppressed patients. Mycobacteria can be difficult to identify. Recently some clinicians have resorted to polymerase chain reaction in order to detect mycobacterial DNA.⁷

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Don B. David, MD, FRCOphth ✉
Machen Eye Unit
Warwick Hospital
Lakin Road
Warwick CV34 5BW, UK

Lawrence W. Hirst, MB, BS, Hons (Qld), MD (Qld),
MPH (Hopkins), DO (Melb), FRACO, FRACS, FACS,
FAAO, Cert. Am Board Ophth.
University of Queensland
Division of Ophthalmology
Department of Surgery
Lions Clinical Research Building
Woolloongabba
Queensland 4102, Australia

Jason McMillen, MB, BS
Princess Alexandra Hospital
Ipswich Road
Woolloongabba
Queensland 4102, Australia

Michael Whitby, MB, BS, FRACP, FRCPA
Department of Infectious Diseases and Infection Control
Princess Alexandra Hospital
Woolloongabba
Queensland 4012, Australia

Sir,

Bilateral non-Hodgkin lymphoma of the conjunctiva

The orbit is devoid of formed lymph nodes and any lymphomas occurring in the conjunctiva, the lacrimal gland, the eyelids and the orbit represent extranodal locations. These tumours ('orbital lymphomas') are relatively rare and can often be associated with concurrent lymphomas outside the orbit.

The most widely used classification system for systemic lymphomas is the Rappaport classification,¹ which divides cases into low-grade and high-grade lesions. In 1980 Knowles and Jacobiek² proposed a classification system of orbital lymphoid neoplasms that comprises two categories: the so-called benign lesions (pseudotumour, reactive lymphoid hyperplasia and inflammatory pseudotumour) and the truly malignant lymphoma.

The orbit is the fourth most common location of extranodal presentation, occurring in 40% of patients with non-Hodgkin lymphoma (preceded by Waldeyer's ring, gastrointestinal and skin localisations).

Approximately two-thirds of patients with 'orbital lymphomas' have lesions of the orbit, 30% lesions in the conjunctiva and 10% lesions in the eyelids. Bilateral presentation was noted in 20% of patients with lacrimal gland and conjunctival lesions, and in only 5–10% of patients with non-lacrimal gland and eyelid masses.

The following case report documents the occurrence of bilateral presentation of a non-Hodgkin lymphoma in the tarsal conjunctiva with an orbital relapse.

Case report

An 81-year-old man was referred to us due to bilateral masses in the inferior fornices. Fourteen months previously he had undergone a gastrectomy followed by radiotherapy for a non-Hodgkin lymphoma originating from the stomach and involving the first lymph node stations. Eye examination revealed the presence of mobile, salmon-coloured ovoid tumours in the conjunctival fornix, bilaterally (Fig. 1).

The lesions were surgically removed and histopathological examination showed a small and intermediate lymphocytic lymphoma with a B cell



Fig. 1. Conjunctival non-Hodgkin lymphoma involving the inferior fornix of (a) the right eye and (b) the left eye.