# *MYCOBACTERIUM CHELONEI* KERATITIS: A CASE REPORT AND REVIEW OF PREVIOUSLY REPORTED CASES

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## SUMMARY

**A 56-year-old woman who wore hard contact lenses developed a keratitis due to *Mycobacterium chelonei*. The organism was only sensitive to imipenem and partially to ciprofloxacin and erythromycin. After an initial response to topical therapy with these antibiotics the infection relapsed and a penetrating keratoplasty was performed, with resulting cure. *M. chelonei* has not previously been reported as a cause of keratitis associated with *hard* contact lens wear; neither has its treatment with imipenem and/or ciprofloxacin. A detailed photographic record showing the natural history of the keratitis is presented. Previously reported cases of *M. chelonei* keratitis are reviewed.**

*Mycobacterium chelonei* keratitis is rare. Only 2 cases have previously been reported in the United Kingdom,[1,2] 13 in the United States and Canada,[3–11,14] 2 in China[13] and 1 in Australia.[12] In these cases the organism was multiresistant to antimicrobials but where specified was sensitive *in vitro*, or showed a clinical response to amikacin. A further 11 cases have recently been reported in the United States, but clinical details are not available.[15]

Delay in diagnosis and multiresistance mean that medical therapy alone is frequently unsuccessful in the management of *M. chelonei* keratitis.

## CASE REPORT

A 56-year-old woman with high myopia who had worn hard contact lenses for 18 years presented with a red painful left eye of 2 days' duration.

At the age of 17 years she had mild dysthyroid eye disease and her corrected visual acuities were 6/9, N5 in each eye. Clinical signs included lid retraction, lid lag, mild exophthalmos and superior limbic keratoconjunctivitis, all of which subsequently improved.

With the onset of the painful left eye, she ceased wearing her contact lenses and was treated for 'conjunctivitis' with G. neomycin 0.5%/hydrocortisone 1.5%. After initial improvement, the condition relapsed and 1 month later the treatment was changed to G. chloramphenicol 0.5% q.d.s.

The corrected (−10.00 DS) left visual acuity was 6/18, the eye was red with a small inferior corneal ulcer plugged with mucoid material. The lesion was debrided, treated with Oc. betamethasone 0.1%/neomycin 0.5% and padded. Initial improvement was followed by relapse. A chalky anterior stromal infiltrate with associated epithelial defect developed. Surrounding epithelial oedema, localised stromal oedema and a few small keratic precipitates (KPs) were evident. The patient was admitted for corneal scraping and treated with hourly topical G. gentamicin 0.3% and G. methicillin 2%. The following day the condition was thought to resemble staphylococcal corneal disease and treatment was changed to G. methicillin 2% 2-hourly and G. prednisolone 0.3% q.d.s. Three days later bacteriological culture revealed growth of an atypical mycobacterium (later identified as *M. chelonei*) sensitive to gentamicin. However, since the condition was improving the patient was discharged with no change in treatment.

Four days later she returned; the infiltrate was thicker, the KPs larger and there was mild iritis. Treatment was changed to G. gentamicin 0.3% 2-hourly and G. prednisolone 0.3% q.d.s. Four days later the condition had deteriorated further and the visual acuity was 6/24–1. Corneal sensation was normal but the lesion was now thought to look herpetic. Oc. acyclovir 3% 5×/day was added. Again there was an initial clinical improvement but this was not sustained. After a further 6 weeks of treatment the patient was referred to St Thomas' Hospital.

The patient's best corrected (−12.00 DS) left visual acuity was 6/18. The eye was injected and the corneal

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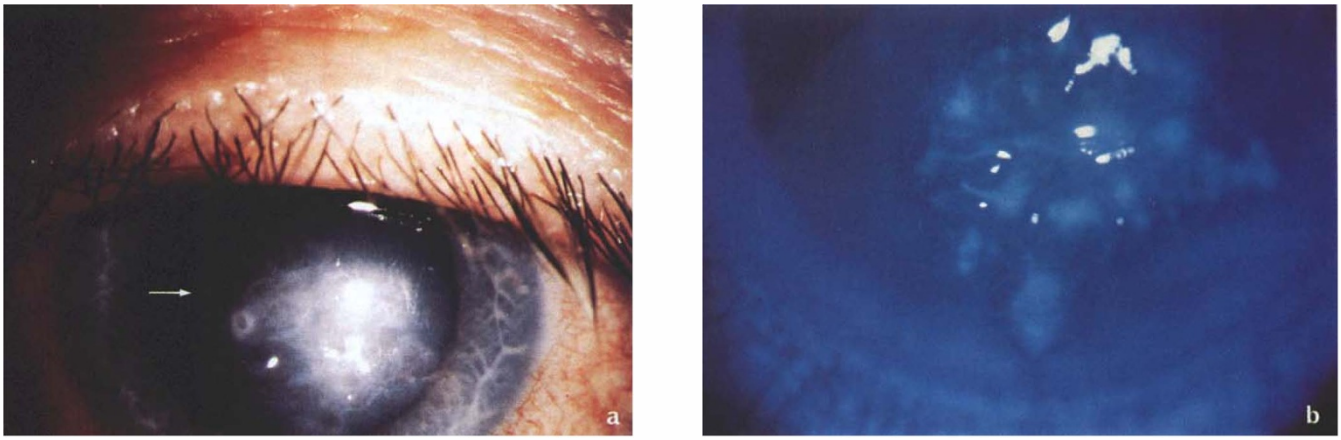



**Fig. 1.** *The left eye at presentation to St Thomas' Hospital. (a) The corneal lesion. (b) The lesion stained with fluorescein. There is a craggy, opaque, central and anterior corneal plaque measuring 5 × 4 mm (a) with associated epithelial staining defects (b). Beneath this were several discrete but faint stromal lesions with surrounding satellite lesions and adjacent deeper stromal infiltrate. The lesions were mainly globular, although some had a spicule or crystal-like appearance. A separate, small deep-stromal satellite lesion 0.75 mm from the main lesion can be seen superonasally (arrow).*

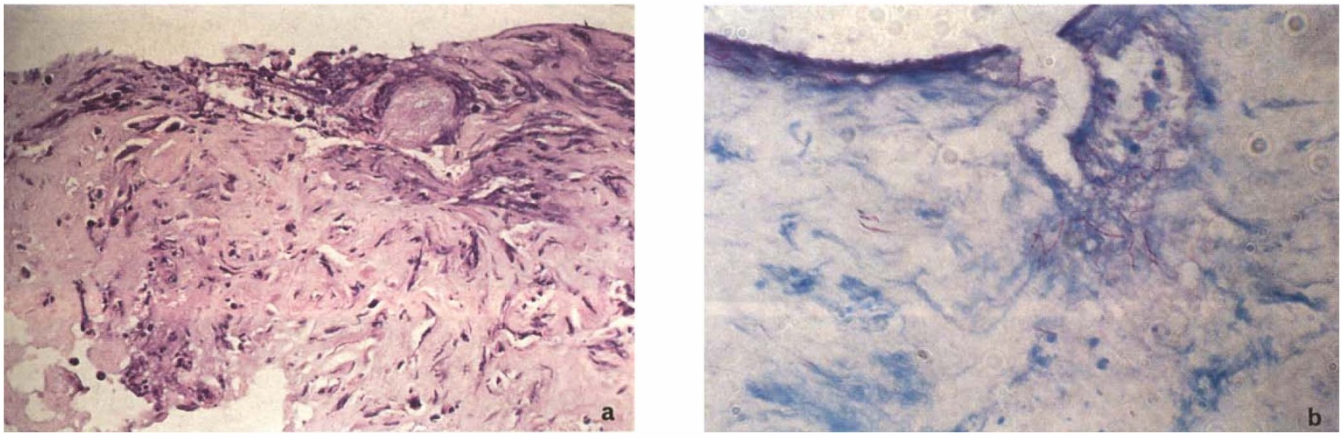



**Fig. 2.** *The corneal biopsy. (a) There is ulceration of the surface epithelium and stromal infiltration with a moderate number of polymorphs. (b) In the slough of the ulcer base there are numerous acid–alcohol fast bacilli (AAFB) (Ziehl–Neelsen stain).*

lesion consisted of a large diffuse sub-epithelial infiltrate below which a number of more focal opacities were present. A satellite lesion was identified supranasally (Fig. 1). There was mild iritis. Treatment was stopped for 2 days and a corneal biopsy taken. Gram stain showed pus cells only, and no acid–alcohol fast bacilli (AAFB) were detected with auramine stain. A fungal keratitis was suspected and treated with G. miconazole 1% 2-hourly and G. atropine 1% q.d.s. Deterioration occurred with extension of the keratitis and development of a fibrinous iritis and small hypopyon.

Meanwhile histopathological examination of the corneal specimen showed numerous AAFB in the slough of the ulcer base (Fig. 2) and after 5 days' incubation a light growth of AAFB was obtained on blood agar and Lowenstein–Jensen medium. The organism was later identified by the Regional Tuberculosis Centre (Dulwich Public Health Laboratory) as *M. chelonei.* Pending the results of *in vitro* sensitivity tests treatment was changed to G. amikacin 2.5% ½-hourly/day and 1-hourly/night, G. dexamethasone 0.1% 1-hourly/day and 2-hourly/night with G. atropine 1% q.d.s. Intensive steroid therapy was considered necessary in view of the anterior chamber (AC) activity and to prevent steroid withdrawal rebound iritis.

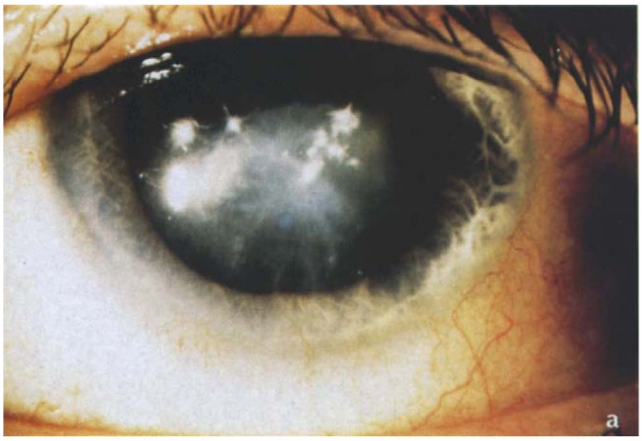
Over the subsequent week a subjective improvement occurred, the eye became less injected and reduction in corneal stromal oedema revealed pigmented KPs. There was only mild AC activity, with no hypopyon. The corneal lesion and visual acuity, however, remained unchanged. The topical antibiotics were subsequently administered at longer intervals.

Ten days later the corneal stromal lesion was more discrete, but the individual opacities, at various depths throughout the stroma, began to change in character (Fig. 3).

The *M. chelonei* was reported sensitive to imipenem and partially sensitive to erythromycin and ciprofloxacin. It was resistant to amikacin.

The patient was then admitted for intensive topical therapy with G. imipenem 0.5% 1-hourly/day and 2-hourly/night, G. ciprofloxacin 0.2% 1-hourly/day and 2-hourly/night, Oc. erythromycin 5×/day, G. atropine 1% b.d. and G. dexamethasone 0.1% t.d.s.

After 2 days there was subjective improvement but little



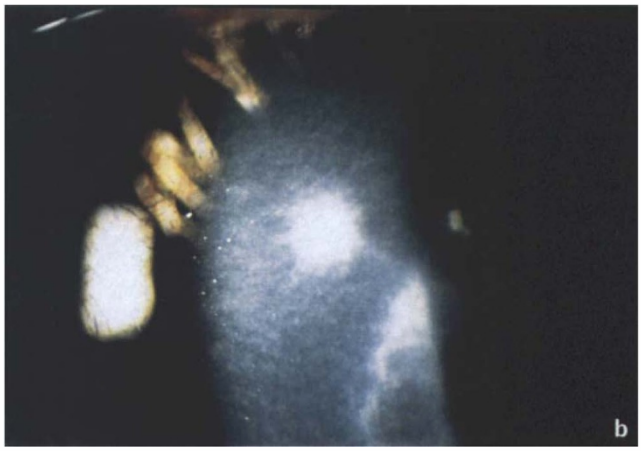





**Fig. 3.** *The corneal lesion 4 weeks after presentation. (a) Localised opacities within the main lesion. (b) The upper-nasal satellite lesions. (c) The upper-temporal quadrant of the main corneal lesion showing some of the localised stromal opacities. Discrete, dense, deep stromal lesions have appeared within the main lesion. Two satellite lesions can be identified superonasal to the main lesion, the original one having enlarged, the second, situated at the edge of the main lesion, being small and new. A group of similar lesions can be seen at the upper-temporal edge of the main lesion. The lesions were initially globular in nature with no adjacent cellular activity. The corneal epithelium was intact at this stage. Four days later some of the globular opacities were noted to have migrated to the mid-stroma, become stellate shaped and to show polychromatic arborisation.*

change on examination. The main lesion developed an irregular surface and became thinned centrally. The discrete globular and stellate stromal lesions (including the satellites) persisted (Fig. 4). Over the next 3 weeks the corneal lesion changed. The central components of the main lesion were extruded; the satellites remained (Fig. 5). Topical antimicrobial therapy was reduced to 3-hourly and the steroid increased to 4-hourly.

For 5 weeks there was a reduction in inflammation and an increase in visual acuity to 6/12 + 1. Over the subsequent 10 days a new satellite lesion appeared temporally (Fig. 6), the upper-nasal lesions disappeared and AC activity increased. Steroid administration was increased to 2-hourly. Ten days later a new focal globular opacity was evident within the central part of the main lesion. Over 2 weeks this opacity enlarged and simultaneously the upper-temporal lesion became less feathery, more anteriorly situated and associated with a small epithelial defect and localised stromal oedema which responded to hourly G. dexamethasone 0.1%. Both lesions were eventually extruded in the same manner.

Over the following month the condition remained stable with no further development of focal stromal opacities, although the central opacification had increased to 5 × 8 mm in area (Fig. 7). After 11 weeks of specific treatment the condition worsened. Visual acuity fell to 6/36, there was central epithelial loss and new peripheral satellite lesions appeared.

A further corneal biopsy was performed and histopathological examination revealed numerous AAFB. Cultures were unfortunately contaminated. Thus, a left penetrating keratoplasty (PK) was performed about 10 months after onset of the initial symptoms. Histopathological examination of the corneal button (Fig. 8) demonstrated clear surgical margins.

Two years later the graft remains clear and corrected visual acuity is 6/12.

## DISCUSSION

The atypical mycobacterium *M. chelonei* was first identified by Freidmann in 1903 when he cultured AAFB from two Berlin Zoo sea turtles (*Chelona corticata*) which had developed pulmonary disease. Distribution of the organism within the environment remains unknown,[16] but it was first recognised as a human pathogen in 1953 by Moore and Frerichs[17] who isolated it from an infected knee joint. Since then it has been increasingly reported as a pathogen in a variety of human diseases.[6,16,18–25] *M. chelonei* keratitis is rare. We have identified 18 cases,[1–14] summarised in Table I, and a further 11 of which there are no clinical details.[15]

*M chelonei* is an opportunistic pathogen and most patients with this infection have had predisposing injury (including surgery). Others have been immunocompromised or worn extended-wear contact lenses. Preceding local trauma had occurred in 16 of the 17 reported cases of *M. chelonei* keratitis where relevant details were provided

**Fig. 4.** *The corneal lesion 7 weeks after presentation. A mixture of globular and stellate stromal lesions can be seen.*

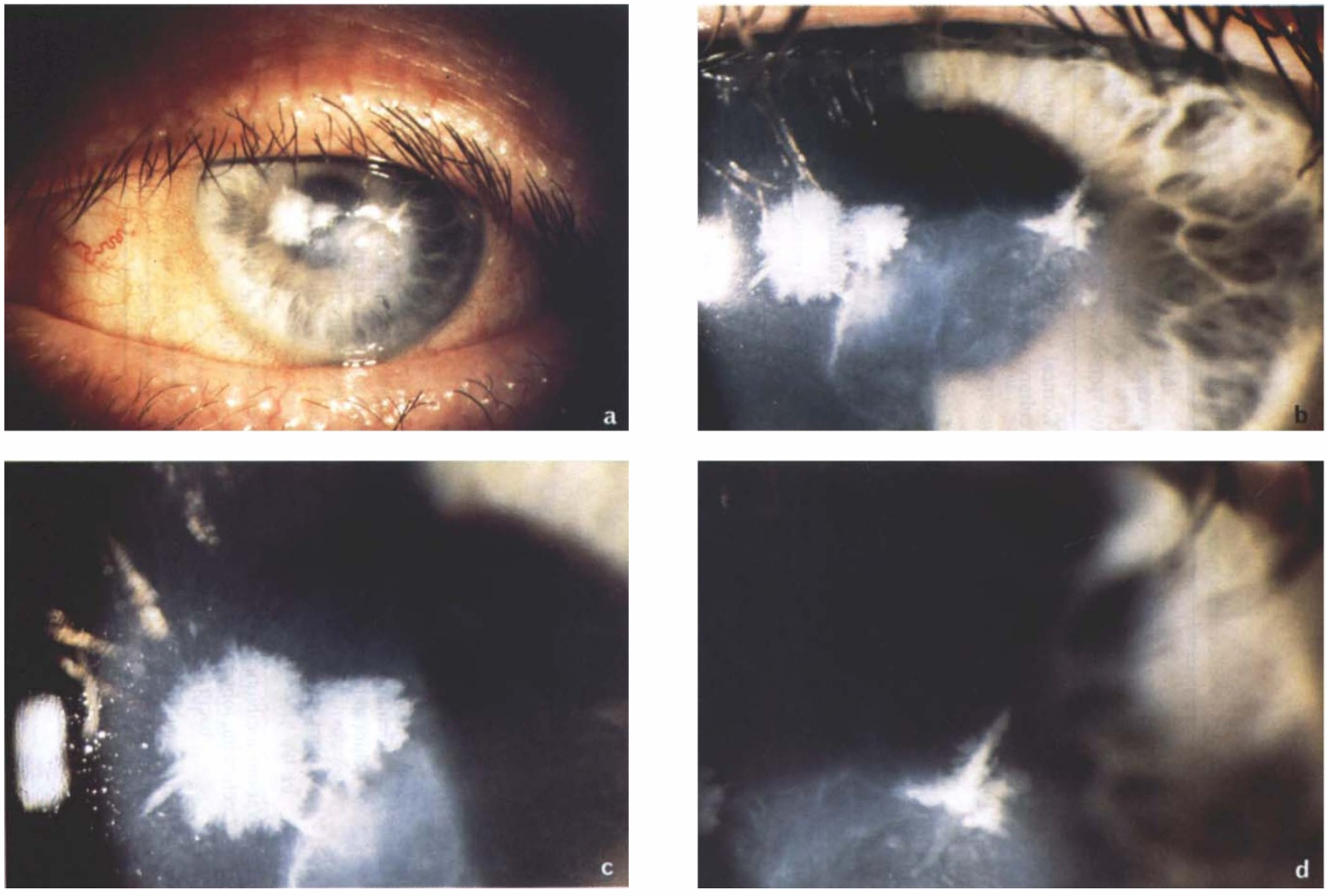



**Fig. 5.** *The corneal lesion 10 weeks after presentation. (a) The left eye. (b) The corneal lesion. (c) The upper-nasal satellite lesions. (d) The opacity at the upper-temporal margin of the main lesion. Each of the discrete opacities within the main lesion migrated forwards within the cornea, passing through a transient stellate appearance in the mid-stroma. Finally, each was extruded to disappear and leave behind an epithelial erosion. The two satellite lesions (c) and one on the upper-temporal margin of the main lesion (d) remained, becoming larger and more fluffy or feathery.*

(see Table I). In this case the patient had worn hard contact lenses for 18 years and had mild dysthyroid eye disease which may have predisposed to the infection.

In most of the reported cases there was a latent period of 2–8 weeks after corneal insult before the keratitis presented. The condition is characterised by a long delay between presentation and correct diagnosis for a number of reasons: the keratitis is rare and readily mistaken (as in this case) for herpetic, fungal or other bacterial disease. Repeated failure of a variety of therapeutic regimens should alert the clinician to seek expert laboratory assistance. Most microbiology laboratories do not perform special staining for AAFB on corneal specimens unless specifically requested, and most specimens are incubated for only 48 hours – an inadequate time for *M. chelonei* to grow. The first indication of this unusual infection may well come from a histology report.

Descriptions of the *M. chelonei* lesions from previous reports are similar to this case, the characteristic lesion being an irregular infiltrate with radiating projections, the

**Table I.** Cases of *Mycobacterium chelonei* keratitis

| No.[ref] | Date | Age and sex | Insult to cornea | Interval between injury and keratitis | Initial antimicrobial treatment | Steroid | Method of diagnosis and *in vitro* sensitivities | Definitive antimicrobial treatment | Surgery | Duration of disease | Result |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 1[3] | 1978 | 39 F | Dendritic ulcer | Not specified | Idoxuridine | — | Corneal scrape and culture; gentamicin | Kanamycin, erythromycin, gentamicin | PK (for failure of medical treatment) | 2 months | Clear graft 16 months later |
| 2[4] | 1982 | 35 M | Metal FB (welding) | 6–8 weeks | Prednisolone/ sulphacetamide, neomycin/ polymyxin B, amphotericin B, bacitracin | + | Corneal scrape and culture; amikacin | Amikacin (condition improved but could not be eradicated) | PK | 4–5 months | Not specified |
| 3[5] | 1983 | 84 F | FB (raking leaves); dendritic ulcer? | ≤5 weeks | Idoxuridine, trifluoridine, prednisolone/ sulphacetamide, phenylephrine, erythromycin, polymyxin B/neomycin/ gramicidine | + | Corneal scrape and culture; amikacin | Amikacin and erythromycin (condition improved but could not be eradicated) | PK | 5 months | Clear graft 6 months later |
| 4[6] | 1983 | 60 M | Suture abscess (post triple procedure) | 3 months | Not specified | + | Corneal scrape and culture; no sensitivities specified | Amikacin | — | Not specified | Resolution |
| 5[7] | 1984 | 68 M | Suture removal (post PK) | 3 weeks | Various antibiotics including natamycin | + | Corneal scrape and culture; amikacin, erythromycin | Erythromycin (10 days), Amikacin (5 months) | — | 5 months | Resolution (with residual scarring) |
| 6[7] | 1984 | 78 F | Needle PC | 2 weeks | Various antimicrobials (unspecified) | + | Corneal button culture; amikacin, erythromycin | Various antimicrobials (unspecified) | PK (for failure of medical treatment) | 3 months | Clear graft |
| 7[7] | 1984 | 82 F | Needle PC | 2.5 weeks | Gentamicin, chloramphenicol | + | Corneal button culture; amikacin, tobramycin, erythromycin | Tobramycin | PK and vitrectomy (for failure of medical treatment) | Not specified | Clear graft |
| 8[8] | 1986 | 37 F | RK | 2 weeks | Various antibiotics including cefazolin, tobramycin and bacitracin | + | Corneal biopsy and culture; amikacin | Amikacin (condition improved but could not be eradicated) | PK | 6 months | Clear graft |
| 9[8] | 1986 | 33 F | RK | 2 weeks | Various antibiotics and anti-fungal drugs (unspecified) | + | Corneal biopsy and culture; not specified | Amikacin | — | >6 weeks | Resolution |

(*Continues*)

**Table I.** (*Continued*)

| No.[ref] | Date | Age and sex | Insult to comea | Interval between injury and keratitis | Initial antimicrobial treatment | Steroid | Method of diagnosis and *in vitro* sensitivities | Definitive antimicrobial treatment | Surgery | Duration of disease | Result |
|---|---|---|---|---|---|---|---|---|---|---|---|
| 10[1] | 1987 | 65 F | PK (×2) and extended-wear contact lens use | 6 weeks (after second PK) | Methicillin, gentamicin, penicillin, chloramphenicol, polymyxin B/bacitracin, cephaloridine, tetracycline | + | Corneal scape and culture; ethionamide, gentamicin (resistant later) | Amikacin, erythromycin | — | 6 months | Resolution (with residual scarring) |
| 11[9] | 1987 | 60 M | PK | 2 months | Various topical medications (unspecified) | ? | Corneal scrape and culture; amikacin | Amikacin (condition improved but could not be eradicated) | PK | 1–2 months | Clear graft 14 months later |
| 12[10] | 1987 | 41 F | PK | 3 months | Isoniazid, rifampicin, ethambutol, pyridoxine, streptomycin | – | Corneal scrape and culture; amikacin, erythromycin, streptomycin, tetracycline | Erythromycin, gentamicin, tetracyline, rifampicin, streptomycin | — | 2 months | Resolution |
| 13[11] | 1988 | 29 F | Extended-wear contact lens use | Unknown | Prednisolone/ sulphacetamide, idoxuridine, vidarabine | + | Corneal biopsy and culture; not specified | Amikacin | — | 9 months | Resolution (with residual scarring) |
| 14[12] | 1989 | 77 F | ECCE + IOL | 6 months | Not specified | + | Corneal scrape and culture; amikacin, cefoxitin, erythromycin | Amikacin, oxytetracycline | PK (for failure of medical treatment) | >6 weeks | Clear graft 8 months later |
| 15[13] | 1989 | ? | Corneal trauma | Not specified | Not specified | – | Corneal scrape and culture; not specified | Various antimicrobials | — | 2–3 months | Not specified |
| 16[13] | 1989 | ? | Corneal trauma | Not specified | Not specified | – | Corneal scrape and culture; not specified | Various antimicrobials | — | 2–3 months | Not specified |
| 17[2] | 1990 | – F | Not specified | Not specified | Not specified | ? | Not specified; not specified | Not specified | — | Not specified | Not specified |
| 18[14] | 1990 | 64 M | FB (mowing lawn) | ≤1 month | Tobramycin, natamycin and ketoconazole with various antimicrobials (unspecified) | | Corneal biopsy and culture; not specified | Amikacin | — | 11 months | Resolution (with residual scarring) |
| This case | 1991 | 56 F | Hard contact lens wear | Unknown | Various antibiotics (see text), acyclovir, miconazole | + | Corneal biopsy and culture; imipenem, ciprofloxacin (partial), erythromycin (partial) | Imipenem, ciprofloxacin, erythromycin | PK (for failure of medical treatment) | 10 months | Clear graft 24 months later |

Abbreviations: PK, penetrating keratoplasty; RK, radial keratotomy; PC, posterior capsulotomy; ECCE, extracapsular cataract extraction; IOL, intraocular lens; FB, foreign body.

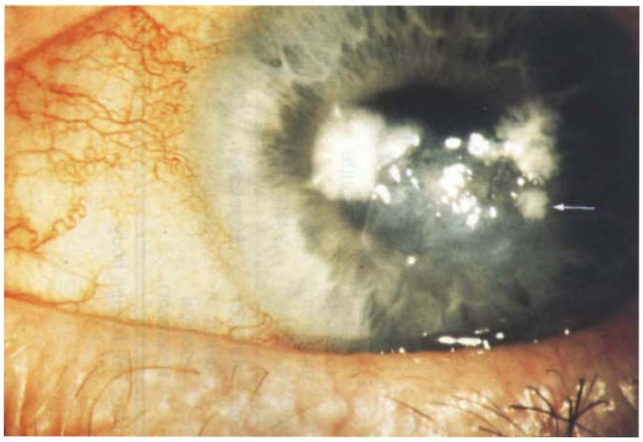


**Fig. 6.** *The corneal lesion 17 weeks after presentation showing the new temporal satellite lesion (arrow). Initially globular and located in the deep stroma, the new satellite lesion gradually increased in size, its appearance becoming more feathery as it migrated into the mid-stroma. In contrast, over the same period of time the two older satellite lesions became confluent and less feathery before being extruded, this being associated with epithelial loss and increased discomfort. The upper-temporal opacity continued to increase in size, maintaining a feathery appearance.*

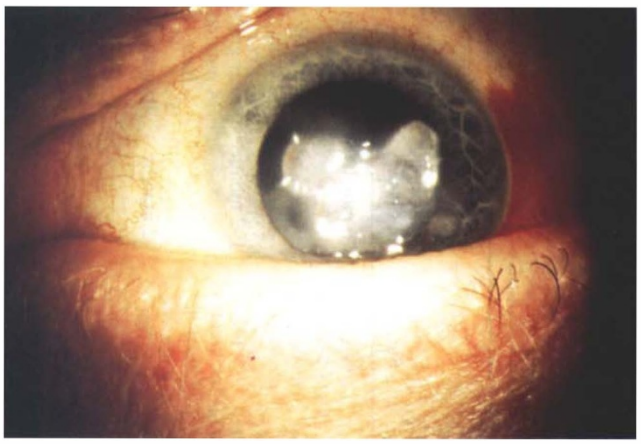


**Fig. 7.** *The corneal lesion 22 weeks after presentation. No focal opacities are present in the corneal lesion, which has increased in size by incorporation of the regions previously occupied by the satellite lesions.*

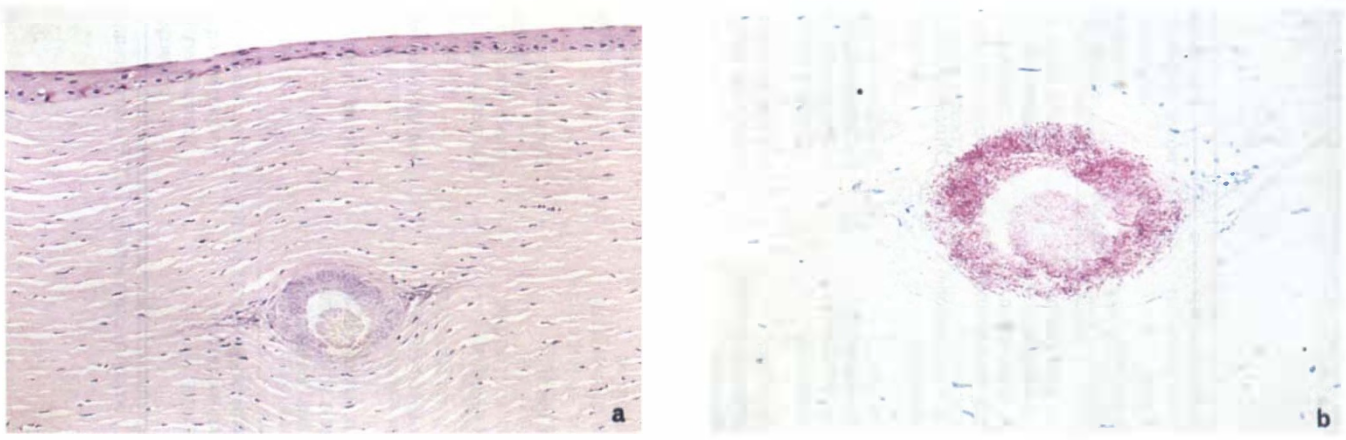



**Fig. 8.** *The penetrating keratoplasty corneal button. (a) There is a central mid-stromal cavity containing a colony of microorganisms associated with some granular material. There is a noticeable absence of inflammation in the specimen. (b) The colony of microorganisms consists of numerous AAFB (Ziehl–Neelsen stain).*

lesion margins having an indistinct, fluffy or feathery appearance. The lesions can involve all levels of the stroma with a predilection for the mid to deep stroma. They have been described as 'snow-flakes'[8] or to have a 'cracked windshield' appearance[5] – this latter appearance has also been described in *M. fortuitum* keratitis.[26] Satellite lesions, a prominent feature in this case, are also reported.[1,9,11,12] An associated epithelial defect appears to be universal.

In none of the previous reports has the natural history of the corneal lesions been discussed. In this case focal lesions appeared initially in the deeper stroma and were globular in shape. These then moved forward in the cornea, the margins becoming indistinct and feathery, giving small lesions a stellate appearance. This appearance diminished as the lesions reached the anterior stroma and eventually the lesion or part of it would become extruded with associated epithelial loss.

In this case definitive diagnosis was by corneal biopsy and culture. The method of diagnosis in 15 previously reported cases was corneal biopsy,[7,8,11,14] scrape [3–5,7,9–13] or re-scrape[1,6] and culture. In 2 of the cases reported by Newman *et al.*[7] the diagnosis was made after penetrating keratoplasty by culture of corneal buttons.

*M. chelonei* is resistant to conventional anti-mycobacterial agents but often sensitive *in vitro* to amikacin[27] which has thus emerged as the drug of choice. Sensitivity testing of atypical mycobacteria is technically difficult and *in vitro* results are poor predictors of clinical response. In 9 of the reported cases of *M. chelonei* keratitis the organism was reported sensitive to amikacin.[4,5,7–10,12] In 2 of these cases[7,8] treatment with amikacin was successful.

In another 5 cases[4,5,8,9,12] there was an initial improvement with amikacin but the infection could not be eradicated, and all were eventually managed by penetrating keratoplasty. Amikacin sensitivity was not specified in 7 cases[2,3,6,8,13,14] but in 3 of these there was a favourable clinical response to amikacin.[6,8,14] In 2 cases,[1,11] although *in vitro* resistance to amikacin was reported, the keratitis responded to amikacin used alone[11] or in combination with erythromycin.[1] These cases indicate that *in vitro* testing to *M. chelonei* can be unreliable.

In this case there was no evidence of *in vitro* sensitivity or clinical response to amikacin, but there was *in vitro* sensitivity to imipenem and partial sensitivity to ciprofloxacin and erythromycin. Treatment with these drugs produced an initial response, but the improvement was not maintained.

Erythromycin has been used successfully to treat *M. chelonei* pulmonary infection[25] and a *M. chelonei* orbital granuloma which occurred after tear duct probing.[24] *In vitro* sensitivity has been demonstrated in some strains of *M. chelonei* causing keratitis,[7,10,12] and some non-ocular infections,[20,21] but most strains are resistant.[27,28]

Imipenem is a carbapenem antibiotic with a broad spectrum of activity. Although experience is limited, its intravenous use has been suggested for the treatment of bacterial endophthalmitis[29] and on the basis of animal studies its topical use has been suggested for the treatment of aminoglycoside-resistant *Pseudomonas* keratitis.[30] Clinical use of a topical preparation has not previously been reported.

Ciprofloxacin is a quinolone antibiotic, also with a broad spectrum of activity. Although it is reported to penetrate the aqueous humour after oral administration[31] there are no reports of its successful use in serious eye infections and there are only a few reports on its topical use in the treatment of corneal infections.[32,33]

Topical treatment with combination therapy was initially successful in this case, but the infection persisted, as in the 5 cases where initial responsiveness to amikacin was reported.[4,5,8,9,12] This failure may be a consequence of reduced bioavailability of drugs as a deep stromal abscess forms or may reflect the development of antibiotic resistance. In addition prolonged treatment with various topical agents including steroids may have compromised the immune status of the patient's cornea. Use of topical steroids, at some stage during their management, was specified in 12 of the 18 previously reported cases.[1,4–8,11,12,14] Only 3 of the 18 responded to medical therapy alone.[6,8,10] In another 4 cases[1,7,11,14] medical therapy was successful in eradicating the infection but there was residual scarring, requiring surgery in 3 cases.[1,7,14] In 8 cases[3–5,7–9,12] surgery was performed after failure of medical therapy and this case falls into this latter category.

The treatment policy suggested by Meisler *et al.*[4] should be re-emphasised, namely that deep corneal infections caused by *M. chelonei* should be surgically excised after an initial trial of antibiotics has failed. This avoids unnecessary topical therapy and prevents progressive ocular morbidity. In retrospect, we delayed too long.

*M. chelonei* keratitis is a rare infection and diagnostic delay is virtually inevitable. Persistent failure with assorted therapeutic regimens should alert the clinician to the presence of an unusual microbe and expert microbiological and histopathological help should be sought. Ophthalmologists should be particularly aware of the possibility of mycobacterial corneal infections after surgery or ocular trauma. Once suspected there should be an aggressive approach directed to obtaining adequate corneal tissue for staining and culture. Corneal biopsy rather than scraping is ideal. In addition, pathology departments should be provided with adequate clinical information since routine cultures may be discarded before mycobacteria have had time to grow.

Key words: Ciprofloxacin, Imipenem, Keratitis, *Mycobacterium chelonei*.

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