AIDS related Kaposi's sarcoma follows a much more fulminant course and is less responsive to therapy than the more rare indolent HIV negative Kaposi's sarcoma, which affects elderly males of Mediterranean or East European Jewish origin or the type which appears in young adult black African males.[1] The therapeutic options for adnexal ocular Kaposi's sarcoma are: (a) observation of asymptomatic lesions; (b) surgical excision; (c) cryotherapy; (d) irradiation.[5 6]

Dugel *et al*[5] classified conjunctival Kaposi lesions into three clinical stages: stage I and II are patchy and flat (less than 3 mm in height); stage III lesions are more nodular and more than 3 mm in height.[5] They found a better rate of success for surgical excision and/or cryotherapy for stage I and II tumours.[5]

Because our patient's conjunctiva was affected by several lesions, necessitating excision of a large area of the conjunctiva, and because of the benign course of this type of Kaposi's sarcoma, we chose the cryotherapy option of treatment. It is also obvious that the risk/benefit ratio of this method is much lower than that of radiotherapy of the adnexa oculi.

This patient is unique as his disease course was benign, his immunological profile was normal, he had never received immunosuppressive therapy, and his blood tests were negative for HIV antigens.

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Acinetobacter exposure keratitis

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Acinetobacter is a pleomorphic Gram negative bacterium. Different names have been used to describe the organism. Recently its taxonomy has changed considerably. *Acinetobacter* strains are now referred to as the *Acinetobacter calcoaceticus–A baumannii* complex.[1] *Acinetobacter* keratitis is rare. *Acinetobacter* keratitis has been reported in association with soft contact lens wear,[2 3] penetrating keratoplasty,[4] and in a patient with chronic lymphatic leukaemia.[5] Corneal perforation caused by *Acinetobacter* has been described.[6] We report a case of corneal ulcer caused by *Acinetobacter calcoaceticus–A baumannii* complex in a gravely ill patient who also suffered from exposure keratitis.

Case report

A 74-year-old man with chronic lung disease, diabetes mellitus, and right hemiparesis secondary to an old cerebrovascular accident was admitted to Kaplan Hospital with pneumonia, urinary tract infection, cachexia, and deep vein thrombosis. He improved after 12 days of intravenous cefuroxime and was discharged in good health. He was readmitted 1 week later with fever, cough, and general deterioration. Another course of treatment with intravenous cefuroxime improved the patient's status but 4 days later the right eye became inflamed. On examination the patient exhibited minimal blinking movements which led to exposure keratitis. A corneal infiltrate in the lower mid-periphery as well as an abundant conjunctival discharge were present. There was no anterior chamber reaction. The corneal ulcer was scraped and cultured. No organisms were detected in the Gram or Giemsa stained smears. The patient was treated with gentamicin (15 mg/ml) and cephazolin (50 mg/ml) eyedrops hourly and tarsorrhaphy was performed. There was marked improvement during the following days. A glucose oxidising member of the *Acinetobacter calcoaceticus–A baumannii* complex grew out of the culture which was sensitive to imipenem, gentamicin, tobramycin, polymyxin B, doxycycline, minocycline, tetracycline, cotrimoxazole, and sulfa. The isolate was resistant to ampicillin, mezlocillin, piperacillin, aztreonam, bacitracin, the cephalosporins, amikacin, neomycin, chloramphenicol, clindamycin, ciprofloxacin, and ofloxacin. After 5 days of treatment the corneal ulcer cleared. *Acinetobacter*, demonstrating the same sensitivity profile as in the corneal ulcer, was also isolated in the urine. Two days later the patient became febrile; a chest *x* ray revealed bilateral lung infiltrates. Despite treatment with intravenous chloramphenicol, the patient's status continued to deteriorate and he died. Permission for autopsy was not granted.

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Comment

Acinetobacter is ubiquitous in nature; it is found on the normal skin of 17–25% of adults. In recent years *Acinetobacter* has developed as an important cause of nosocomial infections.[7,8] The human skin is a reservoir for *Acinetobacter* in hospitals and is the likely source of most outbreaks of hospital infections. In ventilated patients *Acinetobacter* may cause pneumonia after contaminating ventilation devices.[8]

Fifty four to 62% of hospital strains of *Acinetobacter* are resistant to the aminoglycosides. Almost all isolates are resistant to ampicillin, chloramphenicol, first generation cephalosporins, and tetracycline. However, more than 95% of isolates are susceptible to mezlocillin.[8,9] The majority are susceptible to minocycline, doxycycline, and colistin.[10] In vitro studies showed that 4-fluoroquinolones, piperacillin, ceftazidime, and imipenem are effective against gentamicin resistant strains.[9]

Acinetobacter is a normal human commensal of relatively low virulence which colonises rather than infects. Infections occur in patients with (a) decreased resistance, (b) after instrumentation – that is, endotracheal tube and urethral catheter, or (c) with prior broad spectrum antimicrobial therapy. They can be community acquired but most of the infections are nosocomial. Serious infections caused by *Acinetobacter* include: (a) meningitis, (b) subacute and acute bacterial endocarditis, (c) pneumonia, (d) urinary tract infections, and (e) bacteraemia.[7]

Our patient suffered from debilitating diseases and was in a compromised condition. He was infected during hospitalisation. He suffered from exposure keratitis and improved markedly with antibiotic treatment and tarsorrhaphy. *Acinetobacter* with identical sensitivity profile was also isolated from the urine, a sign of generalised infection with the organism. The isolate was resistant to mezlocillin, piperacillin, and the 4-fluoroquinolones that are usually effective drugs against *Acinetobacter*.

We treated another case of corneal ulcer caused by a mixed infection of a glucose oxidising member of the *Acinetobacter calcoaceticus–A baumannii* complex and *Staphylococcus* coagulase positive. The patient showed similar characteristics as the first case. He suffered from diffuse lymphoma and prostatic carcinoma. He had exposure keratitis and his corneal ulcer developed during hospitalisation. He was treated by tarsorrhaphy and topical antibiotics. Although the corneal ulcer resolved completely, his general condition deteriorated and he died.

The clinical distinction of a corneal ulcer caused by *Acinetobacter* may be critical. Smears of *Acinetobacter* show Gram negative intracellular diplococci resembling *Neisseria*. Since most *Acinetobacter* isolates are resistant to penicillin and chloramphenicol, while members of the genus *Neisseria* are sensitive to these drugs, the ophthalmologist must be aware of the similarity between these two genera in smears.

The two patients presented in this study focus the attention of the ophthalmologist on yet another cause of nosocomial eye infection. Exposure keratitis in debilitated patients can be an important factor for subsequent contamination with *Acinetobacter*.

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