

There was minimal enhancement with contrast and no bony erosions. The extraocular muscles appeared normal.

His IgA level was repeated and found to be elevated and his thyroid function tests were normal.

On the basis of the past history and the above findings, a diagnosis of retro-orbital deposits of plasmacytoma was made. Treatment consisted of orbital radiotherapy, oral steroids and a further course of ABCM chemotherapy. There was an initial deterioration of vision within the first month that was attributed to exposure keratopathy. Two months following treatment his vision was 6/9, right and left, and the proptosis improved to 20 mm.

Comment

Multiple myeloma is a malignant proliferation of plasma cells which produces diffuse bone marrow infiltration and focal osteolytic deposits.⁷ Proliferation of malignant plasma cells may occur as a localised deposit in the bone marrow, as a medullary plasmacytoma. However, if the deposit occurs outside the bone marrow it is termed an extramedullary plasmacytoma. Extraskelatal spread of multiple myeloma most frequently occurs in the spleen, liver, lymph nodes and kidneys.⁸

Orbital manifestations of multiple myeloma are rare.^{2,4-6,9} In 1981, a review of the worldwide literature revealed 50 cases.³ Bilateral orbital involvement is the rarest subgroup, with only four cited references in the literature.^{4,10-12}

The disease can affect the orbit in a number of ways. Most commonly, it presents with unilateral proptosis without pain.^{2,5,6} Although orbital involvement is rare, when it does occur it may be the initial presentation of the systemic disease in up to 75% of cases.⁹

The prognosis and treatment regimes for solitary plasmacytoma and multiple myeloma differ.⁵ Therefore it is important to distinguish between them. Solitary plasmacytoma requires only localised treatment and it carries a better prognosis, with a mean survival rate of 8.3 years compared with 20 months for multiple myeloma.⁶

Interestingly this case of recurrence of myeloma was not limited to the orbits but at the time of diagnosis the cutaneous infiltration was also noted. This is a rare phenomenon, with only 60 cases recorded in the literature in 1978.¹³ Generally, the onset of the cutaneous plasmacytoma also proves to be a bad prognostic sign, with 50% mortality at 6 months.¹³

References

1. Henderson JW. Orbital tumors. Philadelphia: WB Saunders, 1973;77-85, 400-4.
2. Hayes JG, Petersen M, Kakulas BA. Multiple myeloma with bilateral orbital infiltration and polyneuropathy. Med J Aust 1980;6:276-7.
3. Mewis-Levin L, Garci CA, Olson J. Plasma cell myeloma of the orbit. Ann Ophthalmol 1981;13:477-81.

4. Jackson A, Kwartz J, Noble JL, Reagan MJ. Case report: multiple myeloma presenting as bilateral orbital masses. Br J Radiol 1993;66:166-268.
5. Levin SR, et al. Multiple myeloma: orbital involvement in a youth. Arch Ophthalmol 1977;95:642-4.
6. Smet MD, Rootman J. Orbital manifestation of plasmacytic lymphoproliferations. Ophthalmology 1987;94:995-1003.
7. Hope RA, Longmore JM, Hodgets TS, Ramrakha PS. Oxford handbook of clinical medicine. 3rd ed. Oxford: Oxford University Press, 1993.
8. Pasmantier MW, Azar HA. Extraskelatal spread in multiple plasma cell myeloma. Cancer 1969;23:167-74.
9. Fay AAM, Leib ML, Fountain KS. Multiple myeloma involving the orbit. Ophthalmic Plast Reconstr Surg 1998;14:67-71.
10. Dufier JL, Trecaan G, Thomas M, Saraux H. Malignant bilateral exophthalmia as a manifestation of Kahler's disease. Bull Soc Ophthalmol (Paris) 1977;77:15-7.
11. Hayes JG, Peterson M, Kakulas BA. Multiple myeloma with bilateral orbital infiltration and polyneuropathy. Med J Aust 1980;2:276-7.
12. Gonnering RS. Bilateral extramedullary orbital plasmacytomas. Ophthalmology 1987;94:267-70.
13. Alberts DS, Lynch P. Cutaneous plasmacytoma in myeloma. Arch Dermatol 1978;114:1784-7.

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Sir

Infectious crystalline keratopathy after stitch removal in a lamellar corneal graft

Infectious crystalline keratopathy (ICK) is a rare corneal infection in which the infecting organism, often bacterial commensals found in the normal conjunctival flora, propagates without ulceration between the lamellae of the corneal stroma.¹ We describe a case of ICK which was observed to spread from a suture track immediately following the removal of the corneal suture, despite the use of prophylactic chloramphenicol. This is the first case to our knowledge where ICK has occurred in a lamellar corneal graft.

Case report

A 68-year-old woman was admitted from the orthopaedic ward of an affiliated hospital suffering from a central corneal perforation in the right eye resulting in a flat anterior chamber. The left eye had a central descemetocoele which was very slightly Seidel positive.

She had severe rheumatoid arthritis and was recovering from a left knee replacement. She did not have a history of rheumatoid eye disease.

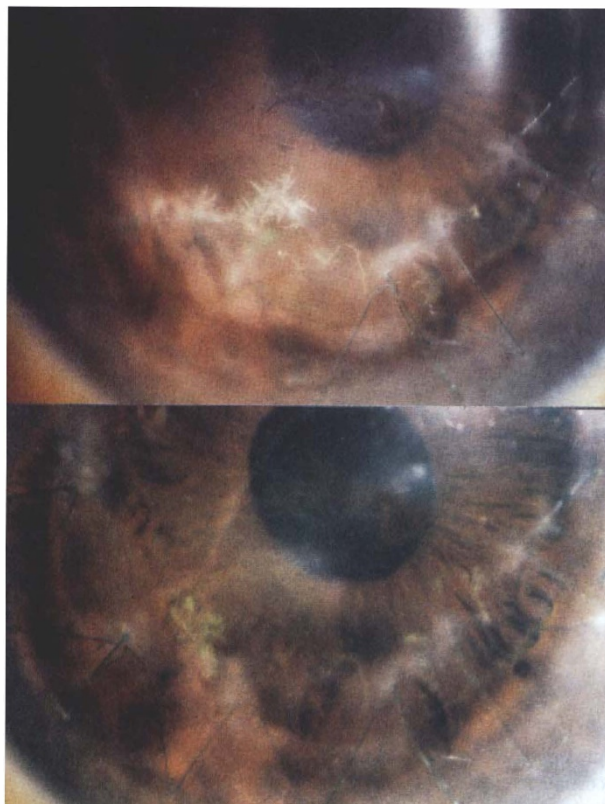


Fig. 1. Upper: Infectious crystalline keratopathy 2 weeks after stitch removal at 7 o'clock. Lower: The lesion cleared with topical penicillin, ciprofloxacin and gentamicin. The cornea remains clear 4 months after the infiltrate was first noted.

She had bilateral lamellar corneal grafts complicated post-operatively by slow epithelialisation. This was successfully managed by copious lubricants, punctal cauterisation, and botulinum ptosis induction in the left eye.

Ten months later, after an uneventful post-operative recovery period, a monofilament corneal stitch was removed from the 7 o'clock position in order to reduce corneal astigmatism of +6.00 D at 60°. At that point the patient was on g. prednisolone 0.5% o.d. R+L, g. hypromellose t.d.s. R+L, g. brimonidine b.d. LE. G. chloramphenicol t.d.s. RE was prescribed prophylactically.

The patient was examined 3 weeks later and a spreading filamentous crystal-like corneal infiltrate was noted originating from the site (Fig. 1, upper). The patient was asymptomatic and there was no epithelial defect. The anterior chamber was deep and quiet and there was no sign of graft rejection.

The diagnosis of ICK was made on the characteristic appearance and the patient was commenced on g. ciprofloxacin every 2 h, oc. gentamicin q.d.s., and g. penicillin every 2 h. Superficial keratectomy to identify the organism was considered but, because the patient was very frail and the area of infiltrate small, conservative management with antibiotics was attempted first in the hope that it would cause resolution.

One week later, the stromal infiltrate had disappeared. In its place there was an overlying epithelial defect and a diffuse ciprofloxacin deposit. The g. ciprofloxacin and oc. gentamicin were stopped and g. chloramphenicol every 2 h started. The g. penicillin was continued.

Two weeks later, the cornea had cleared and there was no evidence of infection. Three months later, the cornea remains clear (Fig. 1, lower).

Comment

In our differential diagnosis, we considered fungal keratitis. However, the needle-like spreading filamentous appearance of the lesion in this case, with the history, made ICK much more likely. Fungal keratitis should always be considered, especially if there is a history of a wooden foreign body or if the patient lives in an agricultural setting. These did not apply in our case.

ICK is a rare condition first described by Gorovoy in 1983.² It is characterised by a spreading, filamentous, branching, needle-like stromal opacity. It is most commonly observed in penetrating corneal grafts. It has also been reported in cases of disciform keratitis due to herpes simplex. Long-term use of topical steroids appears to be a common factor. A break in Bowman's membrane appears to be a prerequisite for the organism to be admitted. In our case, infection appears to have occurred down a suture track.^{1,2}

Histologically, pathognomonic features are microbial proliferation between and limited by the corneal lamellae and a remarkable lack of a host immune response. The overlying epithelium is usually intact and the infection is indolent and progressive. Occasionally the histological features of ICK are present without a visible stromal infiltrate.³

In the largest series,¹ in 18 patients with unilateral ICK, the most common cause found on culture was Gram-positive cocci (10 cases, 55%). Of these, *viridans*-type streptococci was the most common. Five cases grew Gram-negative rods. Three cases were caused primarily by *Candida* yeasts. Four cases grew two different isolates.

ICK is often resistant to antibiotic treatment. 'Blind' treatment should be as broad-spectrum as possible, bearing in mind the statistical likelihood of *Streptococcus viridans*, e.g. penicillin + ciprofloxacin + gentamicin. Another potent combination is gentamicin and vancomycin.

Where 'blind' antibiotics are unsuccessful, a lamellar keratectomy is advised in order to obtain a sample for microbiological culture and histology. With appropriate intensive antibiotics or antifungals, slow resolution of the ICK usually occurs.

It has been pointed out that the term 'infectious crystalline keratopathy', first coined by Meisler in 1984,⁴ is a misnomer, for the condition is non-infectious and there are no crystals present. The term 'arborescent bacterial keratopathy' has been suggested as an alternative,⁵ but this name has not become popular.

References

1. Reviewed in: Khater TT, Jones DB, Wilhelmus KR. Infectious crystalline keratopathy caused by gram-negative bacteria. *Am J Ophthalmol* 1997;124:19–23.
2. Gorovoy MS, Stern GA, Hood CI, Allen C. Intrastromal noninflammatory bacterial colonization of a corneal graft. *Arch Ophthalmol* 1983;101:1749–52.
3. Morrison DA, Fahy GT, Brown LJR. Unsuspected infectious crystalline keratopathy masquerading as corneal graft rejection. *Br J Ophthalmol* 1997;81:608.
4. Meisler DM, Langston RH, Naab TJ, Aaby AA, McMahon JT, Tubbs RR. Infectious crystalline keratopathy. *Am J Ophthalmol* 1984;97:337–43.
5. Watson AP, Tullo AB, Kerr-Muir MG, Ridgway AE, Lucas DR. Arborescent bacterial keratopathy (infectious crystalline keratopathy). *Eye* 1988;2:517–22.

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Sir,

Solitary choroidal metastasis managed by transpupillary thermotherapy

The choroid is the most common site of for uveal metastatic deposits and breast carcinoma accounts 47% of all uveal metastases.¹ External beam radiotherapy² or episcleral plaque brachytherapy³ are currently the favoured modalities in the management of such tumours. We describe a patient with a clinically solitary choroidal metastatic tumour that was treated with transpupillary thermotherapy (TTT).

A 46-year-old woman complained of rapidly decreased vision in her right eye within a month. Two years previously she had undergone radical mastectomy followed by systemic chemotherapy and radiotherapy

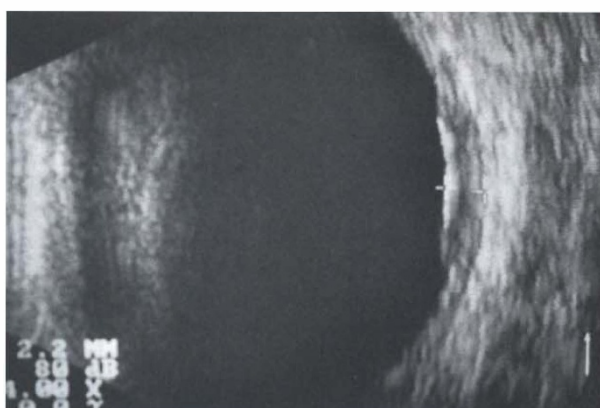
for ductal breast carcinoma. Her visual acuity was 20/40 in the right eye and 20/20 in the left. Examination of the right fundus showed a solitary, yellow-light brown choroidal mass 1.5 mm temporal to the fovea and within the vascular arcades (Fig. 1a). There was a shallow serous retinal detachment, which also partly involved the fovea. A- and B-scan ultrasonography showed a 2.2 mm thick, acoustically solid tumour with medium internal reflectivity (Fig. 1b). A single session of infrared diode laser TTT, using a power setting of 500 mW, 18 overlapping 3 mm spots each for 1 min, was performed. At the end of the treatment the lesion became greyish-opaque. Three months later the patient's right visual acuity was 20/20 and the lesion became flat (Fig. 2). The subretinal fluid resolved completely. Six months later, she maintained the same level of visual acuity with no evidence of recurrence.

Comment

Choroidal metastatic tumours that are inactive or regressed are most often observed. Treatment is usually recommended if the tumour appears active and threatens the vision or continues to grow despite concomitant systemic chemotherapy.¹ However, as patients with choroidal metastases have a median expected life span of about 9 months, too aggressive an approach is generally avoided.^{2,4} External beam radiotherapy (EBR) has a proven efficacy in the management of intraocular metastases. A recent study has shown that useful functional vision could be restored or maintained in 58% of cases and the globe could be saved in 98%.² Age less than 55 years, tumour size smaller than 15 mm at the base and a visual acuity better than 20/50 were more significantly correlated with positive visual outcome.² In the short term, however, 12% of patients develop several complications and dry eye is a significant problem.⁵ It should also be noted EBR takes 3–4 weeks to complete, which is relatively long considering the life expectancy of these patients.



(a)



(b)

Fig. 1. (a) Fundus view of the right eye showing the relatively ill defined choroidal metastatic lesion within the temporal vascular arcades, 1.5 mm temporal to the fovea. (b) B-scan ultrasonography displays the acoustically solid, 2.2 mm thick choroidal metastatic tumour.