However, the keratitis failed to respond to antifungal therapy and surgical excision was required. Although many isolates of S. apiospermum are resistant in vitro to amphotericin B, there have been reports of successful treatment of keratitis with amphotericin B as monotherapy, or in combination with nystatin, natamycin and itraconazole.2,6 Successful treatment of S. apiospermum corneal and orbital infections has been achieved using miconazole. However, there are also reports of keratitis and endogenous endophthalmitis which have failed to respond^{5,7-11} Although Scedosporium spp. are typically resistant in vitro to fluconazole, the patient became intolerant to Itraconazole and a clinical decision was made to supplement topical antifungal therapy with oral fluconazole, but this may have been ineffective. The triazole antifungal voriconazole has been used successfully in the treatment of disseminated Scedosporium spp. infections.¹² It is clear that an optimum treatment regimen for ocular Scedosporium spp. mycoses must still be defined.

This case highlights the dilemma of severe keratomycosis and the continued role of excisional surgery. In any case of atypical keratitis, particularly if there is a history of trauma with vegetative matter, fungal infection must be considered with a high index of suspicion. If this is the case, steroid should be used with great caution due to the risk of enhancing infection. In this case, following the second graft, oral steroid was used successfully to control inflammation and prevent further graft melt. Finally, the risk of secondary glaucoma following large diameter grafts is high. This case illustrates that glaucoma can ultimately limit the visual outcome even after apparently successful surgery.

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Sir,

Primary ductal adenocarcinoma of the lacrimal gland in a patient with neurofibromatosis

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Primary ductal adenocarcinoma of the lacrimal gland is an uncommon tumour and there are only a few published reports.^{1,2} We report the third case of primary ductal adenocarcinoma of the lacrimal gland in a patient with neurofibromatosis and discuss the clinical presentation, radiological characteristics, treatment, and histopathological correlation of the tumour.

Case report

A 46-year-old Asian Indian male reported to our institution with complaints of slowly progressive proptosis of the left eye for the past 2 years. Skin examination disclosed numerous subcutaneous soft nodules suggestive of neurofibromas. He also had

bilateral axillary freckles and numerous café au lait spots on the skin. A diagnosis of neurofibromatosis was made.

Examination of the right eye was normal and left eye revealed gross proptosis and a firm irregular mass in the orbit (Figure 1), more prominent in the superior orbit. The mass was nontender with no soft or cystic areas on palpation. The mass was not mobile. The bony orbital margins appeared intact. No pulsations or thrill was felt. The patient had been earlier diagnosed as a case of pseudotumour at another ophthalmic hospital for which he had been treated with 1 mg/kg oral prednisolone for 2 weeks and had also undergone a tarsorrhaphy with a view to prevent corneal exposure secondary to gross proptosis and lagophthalmos. Since no symptomatic improvement was noted, the patient decided to seek opinion at our centre. In view of severe globe protrusion and risk of exposure keratitis, it was decided to leave the tarsorrhaphy in place.

Ultrasound examination of the left orbit revealed a large heterogeneous mass with variable reflectivity in the intraconal and extraconal spaces, especially in the medial and superior orbit. The optic nerve shadow could be seen separately from the lesion anteriorly.

Computerised tomography plain and contrast of the orbit and brain was perfomed. It showed an isodense homogeneously enhancing retrobulbar mass lesion with



Figure 1 Left eye showing proptosis with multiple neurofibromatosis on the face.

ill-defined margins in the left orbit. The mass displaced the globe inferolaterally and filled the retrobulbar space, both intraconal and extraconal. The mass was seen causing remodelling of the bony orbit; however, there was no evidence of bony destruction. No intracranial extension was noted. A clinical diagnosis of malignant orbital tumour of neural origin in a patient with neurofibromatosis was made.

After appropriate investigations, the patient was taken for early surgery. Intraoperatively, the tarsorrhaphy was divided and ocular examination revealed a normal globe with clear cornea, round pupil and attached retina. A combined superior and inferior orbitotomy was performed through a supratarsal fold incision superiorly and subciliary incision inferiorly. The firm homogeneous mass was debulked with careful dissection to separate it from levator aponeurosis and extraocular muscles. A vacuum drain was placed and the wound closed in layers.

Histopathological examination revealed a lacrimal gland tumour with large neoplastic ductal structures lined by malignant epithelial cells with large oval hyperchromatic nuclei and prominent nucleoli. The tumour showed both cribriform pattern and central comedonecrosis (Figure 2). Phosphotungstic acid haematoxylin stain (PTAH) was negative in the tumour cells.

A diagnosis of primary ductal adenocarcinoma of the lacrimal gland with orbital invasion was made. Systemic work-up for metastasis was negative. He underwent left orbital exenteration by lid-skin-sparing technique followed by postoperative radiotherapy to the socket. The patient is on a periodic evaluation by an oncologist for the past 19 months and has no recurrence.

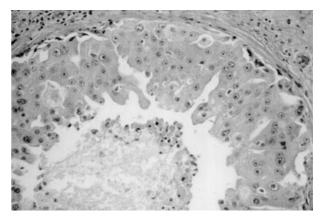


Figure 2 Photomicrograph showing lacrimal duct adenocarcinoma with well-circumscribed nodules of neoplastic ductal epithelium with comedonecrosis in the central area (haematoxylin and eosin \times 200).





Comments

Primary adenocarcinoma of the lacrimal gland is much less common than in salivary glands and represents 5–7% of epithelial malignancies in this location.³ Previous reports of lacrimal adenocarcinoma have classified these tumours together as a single entity of adenocarcinoma or adenocarcinoma not otherwise specified.⁴ However, recently ductal type of lacrimal adenocarcinomas have been reported ^{1,2} similar to histological subclassification of salivary gland carcinomas.⁵ Lacrimal ductal carcinomas have a highly aggressive nature similar to salivary ductal carcinomas.¹

In the first case, the patient underwent modified en bloc orbitectomy with postoperative radiation therapy, and the patient was alive and well without evidence of tumour recurrence 10 months after surgery.¹ In the second case, the tumour recurred in the subdural space after 2 years and it was removed.² The three cases including our case had rapid growth of the tumour, and had sought medical evaluation within 2 years of onset. Neurofibromatosis represents a major risk factor for the development of malignancy, particularly orbital meningiomas, both primary and secondary, nerve sheath tumours and optic nerve gliomas.⁶

In conclusion, malignant orbital tumours have to be considered in the differential diagnosis of proptosis in patients with underlying neurofibromatosis. Lacrimal gland carcinomas have to be subtyped, to predict the biological behaviour of the tumour and the prognosis. The invasive nature of the primary ductal adenocarcinoma of the lacrimal gland dictates aggressive therapy. Combination therapy of wide surgical excision, or even orbitectomy followed by radiation therapy is sometimes required.

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Sir,

Confocal microscopy in bee sting corneal injury *Eye* (2003) **17**, 845–847. doi:10.1038/sj.eye.6700425

A case of corneal bee sting injury with persistent corneal infiltrate was investigated with confocal microscopy, which showed multiple insect foreign bodies invisible under slit-lamp biomicroscopy. This report illustrates the additional value of confocal microscopy in detecting and perhaps identifying retained insect parts.

Case report

A 39-year-old Chinese male was stung in the right central cornea by a bee at work. He suffered immediate eye pain, tearing, blurred vision, and eye redness. He was referred to an ophthalmologist 2 h after the injury. His best corrected visual acuity was 20/30 (0.7) in the affected eye. There was a well-defined area of infiltrate, measuring $2 \times 2 \text{ mm}^2$ in diameter, just temporal to the central visual axis. It extended into the mid-stroma of the cornea with overlying epithelial defect. No foreign body, however, was visible even under the highest magnification (\times 50) of slit-lamp biomicroscopy (Figure 1). There was only mild anterior chamber reaction. Topical chloramphenicol 0.5% and steroid four times per day were given. The infiltrate slowly decreased in density over 1 month and the visual acuity improved to 1.0. However, the residual infiltrate persisted despite continued use of topical steroid four times per day. Confocal imaging was performed to aid detection of foreign bodies in the corneal stroma that might be responsible for the persistent corneal inflammation. White-light tandem scanning confocal microscope (ASL1000-ModelOS-1, New Orleans, USA) with a $\times 24/0.6$ noncontact objective was used, allowing optical sectioning of the cornea with a depth of field $10-12 \,\mu m$. Magnification was up to \times 750. The images were stored in sVHS videotapes. The ASL Image AnalyzerTM