primary cutaneous lesion and several known visceral metastatic sites that precede diagnosis of the ocular metastasis. In contrast, visceral metastases are rare at the time of diagnosis of primary choroidal melanoma. In this case the ocular tumour was the first indication of any metastatic spread and prompted the discovery of other visceral secondaries. This report therefore emphasises the fact that the eye can be the initial site of a clinically identifiable recurrence of cutaneous malignant melanoma[6,8] and that ocular symptoms can develop up to 10 years after the excision of the primary tumour.[8]

Treatment of cutaneous malignant melanoma metastatic to the viscera and eye is usually palliative, and consists of a combination of external beam radiotherapy and chemotherapy. Enucleation was required in this case as the tumour had become extensive, resulting in a blind painful eye. External beam radiation therapy may relieve ocular pain and can be considered in painful eyes with visual potential. The response rate of metastatic melanoma to conventional forms of external beam irradiation is approximately 30–50%.[9] Radioactive plaque treatment can also be considered in patients with an isolated choroidal metastasis.[6]

The survival rate of patients with cutaneous malignant melanoma is partly related to the depth of dermal invasion,[10] which was deep in this reported case. The prognosis with ocular metastases is poor, due to the presence of disseminated disease and the fact that current chemotherapeutic regimes are of limited potential against this malignancy. Studies have shown a median survival of 72 days[6] and fewer than 10% of patients survive more than 8 months.[2]

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Sir,

Bilateral Orbital Metastases from Breast Carcinoma Masquerading as Thyroid Eye Disease

Carcinoma of the breast is the most frequent cause of death due to malignant disease in women[1] and is the most common cause of ocular and orbital metastases.[2,3] Despite this, misdiagnosis of orbital metastases commonly occurs.[4] There are several reasons cited for this in the literature: lack of suspicion, diverse clinical manifestations[4] and difficulty in obtaining an accurate history with regard to previous cancer. Metastatic tumours to the orbit usually result in a focal mass. With breast metastases, however, the growth pattern may be diffuse and can simulate an orbital inflammatory process clinically.[5] We present a case of orbital breast metastases which proved diagnostically challenging because of bilateral, almost completely symmetrical signs and symptoms, which were entirely consistent with a clinical diagnosis of thyroid eye disease.

Case Report

A 64-year-old Caucasian woman was referred from the local radiotherapy department for an urgent ophthalmic opinion. She had been referred for radiotherapy by her local ophthalmologist for treatment of compressive optic neuropathy secondary to thyroid eye disease. The diagnosis of thyroid eye disease had been made 4 months previously on the basis of a history of primary hypothyroidism managed with thyroxine, and the presence of

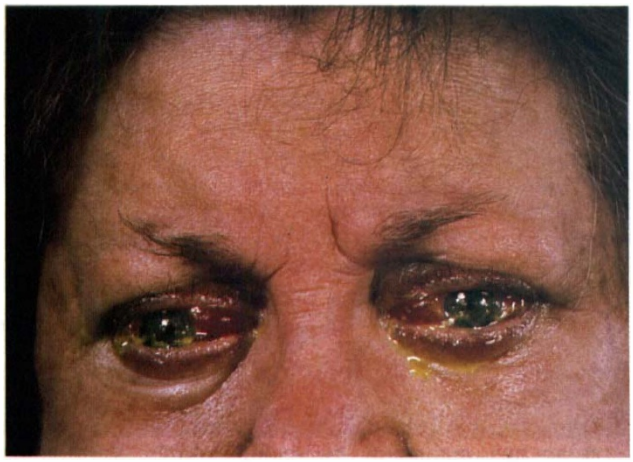

Fig. 1. *This photograph shows the patient after referral from the radiotherapist for an urgent ophthalmic opinion. There are marked bilateral soft tissue inflammatory signs with chemosis, periorbital oedema, proptosis and severe exposure keratopathy.*

bilateral proptosis and upper eyelid retraction. The patient had presented 8 years previously with lassitude, weight gain and cold intolerance, and the clinical diagnosis of hypothyroidism was confirmed by a reduced T4 of 2.9 IU/l (normal 0.3–6.0) and an elevated TSH of 91.3 pmol/l (normal 10–26). She was known to have had a left mastectomy 5 years previously followed by radiotherapy, for an adenocarcinoma of breast with nodal metastases. Systemic prednisolone and cyclophosphamide had been commenced 1 year previously to treat leucoerythroblastic anaemia. This type of anaemia was suggestive of bone marrow infiltration; however, an isotope bone scan revealed no diagnostic features of metastatic disease. An orbital CT scan, with axial views only, was performed 2 months following her clinical diagnosis of thyroid eye disease, and was reported as normal. Over the next 2 months the proptosis increased, bilateral soft tissue inflammatory signs developed and, when visual acuity deteriorated, a diagnosis of compressive optic neuropathy was made. The patient was clinically and biochemically euthyroid at this time and the chest radiograph showed no evidence of lung or bone metastases. As she had previously developed steroid-induced diabetes mellitus, it was decided that radiotherapy for her presumed compressive optic neuropathy was the preferred treatment option. Despite two low-dose orbital radiotherapy sessions (500 cGy fractions to each eye per session), the ophthalmic signs and visual acuity worsened dramatically during the following week and the patient was referred for an urgent ophthalmic opinion.

This assessment revealed an ill patient, in distress with marked ocular discomfort and a visual acuity in each eye of only counting fingers at 0.5 m. The eyes were proptosed with Hertel exophthalmometry readings of 27 mm. There were severe bilateral symmetrical orbital soft tissue inflammatory signs with marked chemosis and severe exposure keratopathy (Fig. 1). There was an almost complete external ophthalmoplegia with upper lid retraction and 10 mm of lagophthalmos. There was marked resistance to retropulsion but no palpable orbital masses. The exposure keratopathy prevented a clear assessment of the optic discs. The eye signs were almost completely symmetrical and as the overall clinical picture was felt to be consistent with malignant thyroid eye disease, the patient was admitted for a CT scan of orbits to be followed by urgent orbital decompression. The CT scan, however, showed no evidence of thyroid eye disease, but instead revealed extensive soft tissue masses encasing the globes (Fig. 2). A revised diagnosis of orbital breast carcinoma metastases was confirmed by histopathological examination of orbital biopsy material obtained via a trans-septal approach (Fig. 3). The patient's general medical condition had now also deteriorated and she was referred to her oncologist. She underwent palliative orbital radiotherapy but succumbed to her metastatic disease 2 weeks later.

Discussion

This case highlights a number of potential pitfalls in the diagnosis of orbital metastases. The history of mastectomy should alert the clinician to the possibility of orbital metastases. The original CT scan, with only axial views, was inadequate. It did not show extraocular muscle enlargement which would have supported the clinical diagnosis of thyroid eye

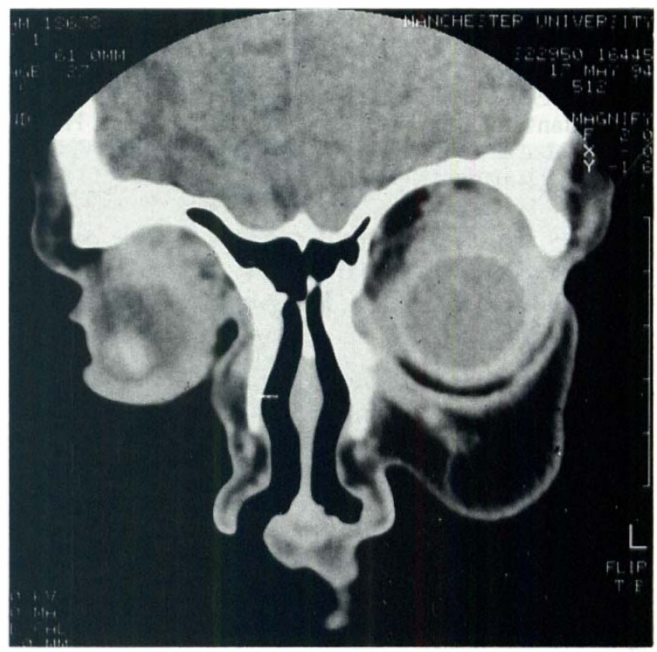

Fig. 2. *A coronal CT scan showing extensive infiltration of both orbits by diffuse soft tissue masses encasing both globes.*

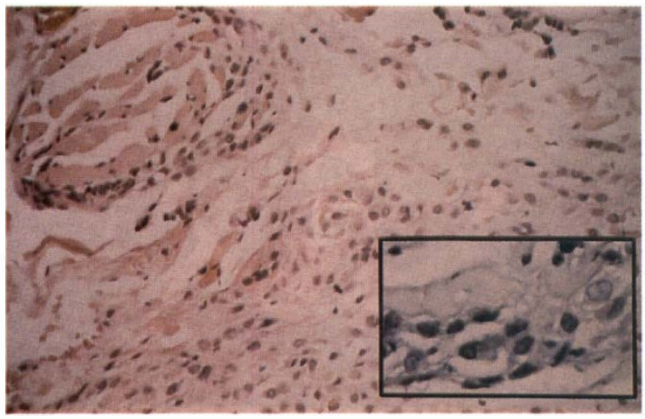

Fig. 3. *Photomicrograph of the histological specimen obtained from the orbital biopsy (H&E stain, ×125). This shows orbital fat, skeletal muscle and nerve widely infiltrated by metastatic adenocarcinoma. The tumour cells infiltrate singly and in cords and many contain intracytoplasmic lumina consistent with an infiltrating lobular carcinoma of breast of primary origin. The inset (combined alcian blue and periodic acid–Schiff stain, ×600) shows mucin-containing cells.*

disease, whereas coronal views at that time may have revealed the presence of orbital masses. Though metastases to the orbit from breast carcinoma are known to occur bilaterally, it is very unusual for bilateral clinical signs to occur so floridly and symmetrically. The diffuse growth pattern simulating an inflammatory process favoured the clinical diagnosis of thyroid eye disease. The lid retraction in the presence of proptosis was also consistent with a clinical diagnosis of thyroid eye disease, but was in fact due to infiltration of the levator palpebrae superioris and upper eyelids by the tumour mass. Though no papable mass was detected, the texture of the lid skin may have offered a helpful clue to the diagnosis. In thyroid eye disease the lid skin has a doughy feel, whereas in our patient the upper eyelid skin texture was board-like and, at biopsy, the upper eyelids were tethered and immobile. Orbital metastases usually respond to a low dose of radiotherapy; however, in this case there was a rapid deterioration in the patient's overall condition after the first radiotherapy session for her suspected thyroid eye disease, and death occurred before a palliative effect was achieved.

This case demonstrates that patients presenting with exophthalmos require a full ophthalmic and general medical examination, followed by confirmation of the clinical diagnosis with CT scan, especially if there is a history of malignancy.

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Sir,

Visual Loss in Metastatic Sclerochoroidal Calcification

Metastatic calcification involving the eye is usually limited to the anterior segment, and posterior segment involvement is both rare and asymptomatic. We report a patient with hypercalcaemia in whom involvement of the posterior segment led to profound visual loss. This has not been reported previously in the literature.

Case Report

An 84-year-old woman was referred to St Thomas' Hospital with gradual deterioration of vision in the right eye, the left eye having had poor acuity for many years. She was symptomatically well and had no past medical history of note.

On examination, visual acuity was 2/60, N36 in the right eye and 6/24, N8 in the left eye. No abnormalities were detected in the anterior segments or vitreous cavities and funduscopy revealed multiple large raised white choroidal masses with overlying retinal atrophy in both eyes. The lesions varied in size from 2 disc diameters to several disc diameters and there was a lesion at the right macula (Fig. 1). Both optic discs appeared normal and general examination of the patient revealed no abnormalities; in particular there were no features suggestive of malignancy.

Ultrasound examination of the eyes and a CT scan of the orbits confirmed the calcific nature of the lesions (Fig. 2). The corrected serum calcium was elevated at 3.04 mmol/l with a normal serum phosphate (1.0 mmol/l). The serum parathyroid hormone (PTH) level was 0.29 U/l (normal range 0–0.79 U/l); chest, abdominal and skeletal radiographs were normal. There was no evidence of myeloma or sarcoidosis and the patient was not on any medication. In the presence of hypercalcaemia and normally functioning parathyroid glands, PTH levels would be expected to be much lower than seen