and corneal transplants, and in preservation of the transient amplifying cells.[9] The poor long-term outcome following simultaneous limbal transplantation and penetrating keratoplasty has prompted the recommendation of a staged procedure with a 1-year gap.[8] Though a stable ocular surface and good vision have been attained following these surgeries, the inherent pathology remains. Photoprotective measures have been advocated and the patient has been cautioned about the possible development of ocular or cutaneous manifestations of XP described earlier. Indefinite immunosuppression remains necessary to ward off limbal allograft rejection.[10] The patient is on low doses of cyclosporine ensuring serum trough levels of 50 ng/ml, regular assessment of renal and hepatic parameters, and on close follow-up in conjunction with an internist.

In summary, limbal stem cell deficiency may be one of the ocular manifestations of xeroderma pigmentosum, necessitating a high degree of suspicion and early surgical intervention to prevent visual disability.

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M Fernandes[1], VS Sangwan[1] and GK Vemuganti[2]

[1]Cornea and Anterior Segment Service
LV Prasad Eye Institute, Hyderabad, India

[2]Ophthalmic Pathology Service
LV Prasad Eye Institute, Hyderabad, India

Correspondence: M Fernandes, MS
LV Prasad Eye Institute, LV Prasad Marg
Banjara Hills, Hyderabad 500 034, India
Tel: +91 040 23608262
Fax: +91 40 23548271
E-mail: fernandes@lvpei.org

Eye (2004) **18,** 741–743. doi:10.1038/sj.eye.6700717

Sir,

Intramuscular lipoma of the eyelid: a case report

Lipomas are common, benign tumours composed of mature adipose tissue. The majority are located subcutaneously around the torso, neck, and proximal limbs.[1] Intramuscular lipoma is a curious subgroup located within muscles, which can invade locally in a diffusely infiltrative pattern.

Intramuscular lipomas of the small muscles of the head and neck are reported only rarely. One previous report has identified intramuscular lipoma involving an eyelid,[2] and one recent report describes an intramuscular lipoma within a medial rectus muscle.[3] We describe a further case of an intramuscular lipoma affecting an eyelid.

Case report

A 77-year-old man presented with a left upper eyelid mass that had slowly enlarged since first being noticed 3 years previously (Figure 1). He had no pain, diplopia, or impaired vision on that side. On examination the mass was subcutaneous, soft, fatty, and nontender to palpation, and there was no proptosis or globe deviation.

The mass was biopsied under local anaesthesia without complication. During the procedure, it was noted that the lesion extended deep in to the orbital septum adjacent to the medial orbital wall.

The histological examination of the specimen (Figure 2) revealed lobulated groups of mature adipocytes surrounding groups of skeletal muscle fibres and individual muscle fibres, providing the diagnosis of an intramuscular lipoma. The presence of muscle fibres within this lesion refutes the diagnoses of orbital fat prolapse or classical lipoma.

At follow up 3 months postoperatively, the cosmetic appearance of the eyelid is very satisfactory. There is no residual mass, and the patient is asymptomatic.

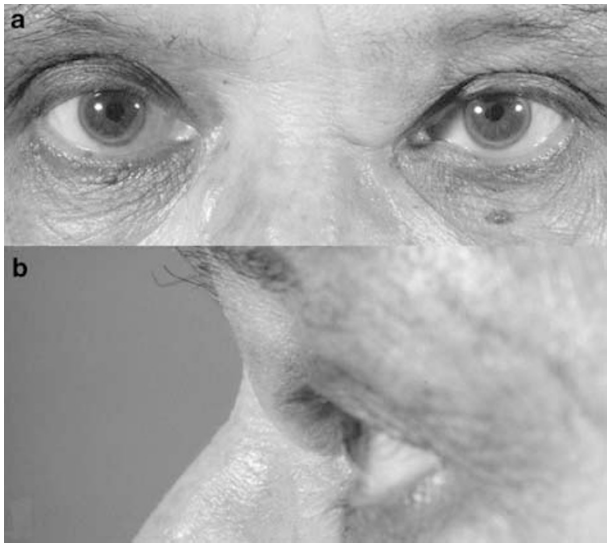

Figure 1 Frontal (a) and lateral (b) views preoperatively showing a left upper eyelid mass.

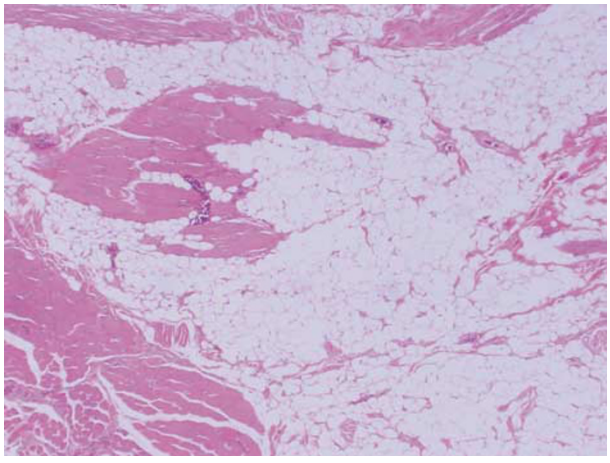

Figure 2 Histopathology image from this patient. Note the groups of mature adipocytes surrounding groups of skeletal muscle fibres and individual muscle fibres. Haematoxylin and eosin stain, original magnification $\times 60$.

Comment

Intramuscular lipomas account for 1.8% of lipomatous tumours and are the most common in middle-to-late adult life.[4] They are most commonly found within the large muscles of the trunk and limbs, and if left untreated can become greater than 20 cm in diameter. Morphologically they vary from well-circumscribed lesions to tumours with a highly infiltrative border.[4] Owing to their deep location and ill-defined borders intramuscular lipomas may be confused clinically with liposarcomas.

Reports of specific clinical problems caused by intramuscular lipomas include voice and breathing problems from infiltrating tumour of the larynx,[5] intussusception of the colon,[6] and infiltration of the paraspinal muscles.[7]

The radiological appearance of intramuscular lipomas varies considerably due to the wide spectrum of tumour morphology: from a single homogeneous mass to a large inhomogeneous lesion with infiltrative margins.[8] Muscle bundles within the tumours can occur with both well-circumscribed and infiltrating lipomas. Interestingly, intermingled muscle fibres and an infiltrative margin do not indicate malignancy.

Typically, the fat and muscle tissue within an intramuscular lipoma have an identical intensity to normal fat and muscle tissue on T1- and T2-weighted MRI images, and gadolinium-DTPA does not enhance the lesion.[3] Liposarcomas are more likely to be multinodular, with variation of signal intensity between T1- and T2-weighted images compared to normal fat and muscle tissues, and exhibit gadolinium-DTPA enhancement.[8]

Following surgical excision, reported recurrence rates of infiltrating intramuscular lipomas vary from 19 to 62.5%.[4,9,10] One study[9] with an average 7-year follow-up reported recurrence in five of the 12 lower limb tumours (42%).

Histologically, intramuscular lipomas are composed of mature fat cells, adipocytes, with varying amounts of skeletal muscle fibre bundles contained within the tumour. The histological hallmark of liposarcoma is the lipoblast, or immature fat cell.[1] Features that help to distinguish a liposarcoma from benign lipoma are the presence of lipoblasts, myxoid differentiation, cellular pleomorphism, plexiform vascularity, and mitotic activity.

The most common genetic abnormality for all lipomatous neoplasms is translocation between the long arm of chromosome 12 and other chromosomes, particularly chromosome 3.[1,11,12] Rearrangement of chromosome 12 at bands q13–15 constitutes 64% of lipomas with identified clonal abnormalities. One study identified chromosome 12 abnormalities in seven out of the eight (88%) intramuscular lipomas tested.[11]

This case reinforces intramuscular lipoma as a differential diagnosis of eyelid masses, and highlights the problems managing a benign, slowly infiltrative tumour in a location where extensive resection is difficult to achieve due to the highly specialised nature of surrounding structures.

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A Buller, A O'Donnell, RE Bonshek and B Leatherbarrow

Manchester Royal Eye Hospital
Oxford Road, Manchester, M13 9WH, UK

Correspondence: B Leatherbarrow
Tel: +44 161 276 1234
E-mail: bollin@mighty-micro.co.uk

Eye (2004) **18,** 743–745. doi:10.1038/sj.eye.6700752
Published online 16 January 2004

Sir,

Photodynamic therapy of subretinal neovascularization in radiation retinopathy

Radiation retinopathy is a delayed-onset, slowly progressive vaso-occlusive retinal disorder that develops after the head and orbital area have been exposed to radiation. In this study, we report a case of unusual subretinal neovascularization observed after external beam radiation. The subretinal neovascularization was resolved, and visual acuity was improved after photodynamic therapy (PDT) with verteporfin.

Case report

A 35-year-old female visited our clinic with decreased visual acuity in the left eye, which had started about 5 months earlier. She had an epithelial cell tumour, located on the left side of nasopharynx, and had received radiation therapy 3 years before this visit. Radiation therapy was carried out for 5 days per week for 6 weeks with a daily radiation dosage of 200 cGy. Radiation therapy was carried out 30 times with a dose of 6000 cGy in total. Adjunctive chemotherapy was performed with a methotrexate/cyclophosphamide regimen.

Her initial corrected vision was 0.5 in the right eye and 0.025 in the left eye measured with Snellen chart. A fundus examination showed multiple cotton-wool patches, retinal haemorrhages, and exudates in both eyes, which were more severe in the left than in the right eye. Fluorescein angiography (FA) of the left eye showed retinal capillary nonperfusion, telangiectactic changes, and microaneurysms in the superotemporal perifoveal area in the early arterial venous phase, and subretinal neovascularization, thought to have originated from an aberrant retinal vessel. As time passed, the subretinal neovascularization became more distinctive and terminally dilated, and formed small bullae-like shapes (Figure 1a). On approaching the late phase, leakage from the telangiectatic vessels, microaneurysms, and the subretinal neovascularization continued. Indocyanine green angiography revealed hyperfluorescent lesion that was previously documented as subretinal neovascularization on fluorescein angiography, and exudative lesions at macula showed a blockage of fluorescence. However, no abnormality of the choroidal vessels suggestive of choroidal neovascularization or choroidal vasculopathy was documented on ICGA. A dome-shaped, hyper-reflective lesion, thought to be neovascular tissue, was found over the intact retinal pigment epithelial layer by optical coherence tomogram (OCT) (Figure 1b).

We performed PDT on the left eye to treat the subretinal neovascularization. Her corrected vision improved to 0.5 after 3 months, and FA 3 months after PDT showed that the subretinal neovascularization had disappeared (Figure 2a). At 3 months after PDT, the macula oedema had decreased and the dome-shaped, highly reflective lesion had disappeared on OCT (Figure 2b). At 6 months after PDT, the patient's visual acuity remained at 0.5 in the left eye.