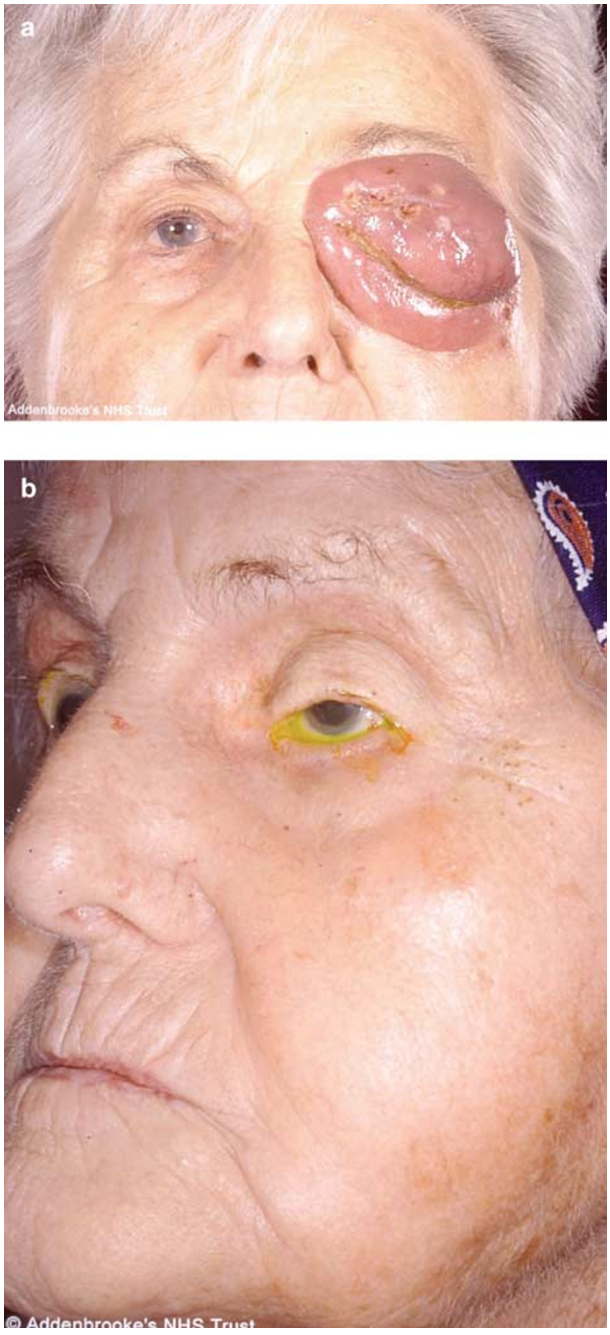

Figure 2 (a) Rapidly enlarging left orbital mass. (b) Clinical resolution of the mass postchemotherapy.

can prove invaluable and should be performed even when the diagnosis appears to be clear.

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Eye (2005) **19,** 108–110. doi:10.1038/sj.eye.6701407
Published online 16 April 2004

Sir,

Orbital amyloidosis presenting as ptosis

Amyloidosis is a condition characterised by the deposition of amorphous proteinaceous material that may involve many organs (systemic form) or be localised to a single organ (localised form). It can be deposited in any part of the orbit, globe, or adnexa. Orbital involvement is uncommon with varied presentations. We report two patients who presented with ptosis that was initially thought to be involutional in nature.

Case report

Patient-1

A 60-year-old woman presented to the eye clinic in 1986 with right-sided ptosis of 1-year duration and was diagnosed as having involutional ptosis. She subsequently suffered recurrent episodes of subconjunctival haemorrhage and developed an orbital mass in 1993. Examination revealed a right-sided 3-mm ptosis. A diffuse mass was palpable through the right upper lid (Figure-1a and b) that was seen to prolapse through the upper fornix. The rest of the ocular examination including visual acuity was normal in both eyes. An orbital CT scan showed a diffuse orbital mass over the right globe, which enhanced with contrast (Figure 1e).

Biopsy via the superior fornix in February 1993 (Figure 1c and d) showed the presence of extensive deposits of amyloid maximally around blood vessels with typical features of amyloidosis. Further debulking was performed in February 1994. CT scan was repeated in 2002 and showed stable findings (Figure 1f).

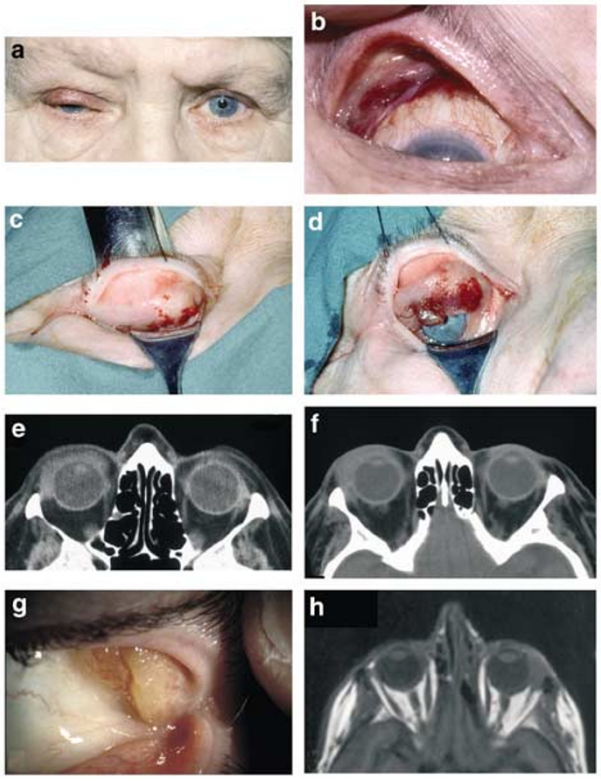

Figure 1 Patient-1. **(a)** Right-sided ptosis. **(b)** Orbital mass prolapsing through the right upper fornix. **(c) and (d)** Intraoperative photographs at the time of biopsy. **(e)** First CT orbits showing the right-sided orbital mass. **(f)** Repeat CT in 2002 showing the mass to be unchanged. **Patient-2. (g)**. Translucent, yellow, and nodular orbital mass prolapsing through the left upper fornix. **(h)** MRI of orbits showing the mass in the left orbit.

Patient-2

A 61-year old woman was seen in July 1995 with a left-sided ptosis of a few months duration. On examination, she was found to have 1-mm ptosis on the left side that was thought to be due to dermatochalasis. Her visual acuity in the left eye was normal and was 6/36 in the right eye due to macular changes following previous retinal detachment.

She was re-referred in 1999 with a lump on the lateral aspect of the left upper lid. On examination she had a 1-mm ptosis and a translucent, yellow, and nodular mass visible in the upper fornix in the lacrimal gland area of the left eye (Figure 1(g). Magnetic resonance imaging (MRI) showed a well-defined mass in the lacrimal gland region with no adjacent bony changes (Figure 1h). A transconjunctival biopsy of the lesion performed in March 2000 showed deposition of amyloid in the tissue. A repeat MRI in May 2002 showed the mass to be unchanged.

Comment

Orbital amyloidosis can occur in the lacrimal glands, extraocular muscles, and orbital fat, and is usually localised.[1,2] The CT findings of lacrimal involvement may mimic other inflammatory or lymphoproliferative disorders involving the lacrimal gland.[3] Ptosis may be a manifestation of localised orbital amyloidosis.[4]

In our patients, ptosis preceded the detection of orbital mass by many years. In the first patient, it took 7 years and in the second patient it was 3 years before the mass was seen. The pathogenesis of ptosis is not clear. It does not appear to be neurogenic in nature as there was no abnormality of ocular motility. It is probable that the amyloid deposition in the levator palpabrae superioris was responsible for the ptosis.[5]

Savino *et al*[6] reported on a patient with ptosis, who was presumed to have myasthenia gravis and was treated. The patient was subsequently found to have localised amyloidosis of the orbit. This case is similar to the two patients whom we have described. It is important to keep this rare condition in mind when faced with a situation where there is unexplained unilateral ptosis with no convincing underlying pathology.

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Eye (2005) **19,** 110–112. doi:10.1038/sj.eye.6701411
Published online 16 April 2004

Sir,

Pseudohypopyon due to malignant infiltration of the anterior chamber in multiple myeloma

Ocular involvement is rare in multiple myeloma and is mainly due to the presence of paraproteins.[1–3] Direct infiltration of plasma cells into the eye is exceedingly rare. Here, we report a case of abnormal plasma cell infiltration in the eye of a patient with myeloma, which presented as a pseudohypopyon.

Case report

A 75-year-old female, who had been diagnosed with myeloma 4 months ago, was referred for an ophthalmic evaluation. She had complained of a 2-week history of visual deterioration with mild discomfort in the right eye. Ocular examination revealed conjunctival hyperemia, posterior synechiae, fine keratic precipitates, a hypopyon (Figure 1), and a mild vitritis in the right eye with a visual acuity of 6/18. The left eye was normal. Clinically there was no evidence of systemic infection. Vitreous and aqueous taps obtained did not reveal any microbial growth, but demonstrated the presence of small and mature multinucleated plasma cells on microscopy (Figure 2a Giemsa staining). Flow cytometric immunophenotyping of cells both from the blood and from the aqueous sample (Figure 2b–d) demonstrated the presence of the myeloma phenotype characterised by $CD19^-/CD20^-/CD45^-/CD38+/CD138^+/CD56^+$.[4–6]

The patient, already on thalidomide at this point, was commenced on a course of mild topical steroids (0.5% prednisolone) four times daily as well as 0.5% cyclopentolate bd. Although there was symptomatic improvement, a small pseudohypopyon remained after 8 weeks of therapy with no change in visual acuity. Sadly, this patient succumbed to the disease 3 months later.

Comment

Direct ocular involvement in myeloma has been reported,[7,8] but only one report has demonstrated definitively, by the use of immunohistochemistry, the identity of the malignant cells within the anterior chamber. [8] Myeloma plasma cells are phenotypically abnormal, and have been shown to be distinguishable from normal plasma cells with flow cytometric analysis on the basis of their low expression of CD19 and CD45 and higher levels of expression of CD138 and CD38 with or without increased expression CD56.[4–6] Our study has demonstrated that flow cytometric analysis for the detection of malignant phenotypes is possible in small

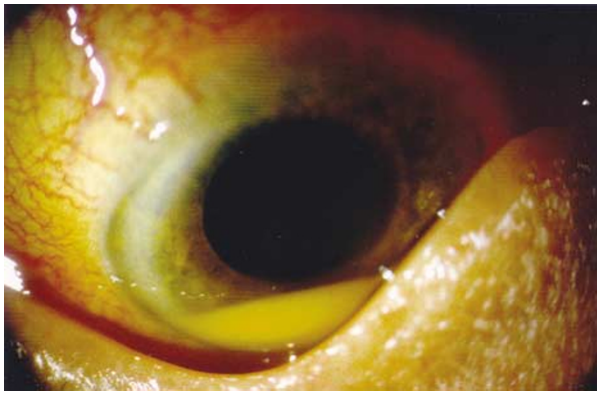

Figure 1 Hypopyon in the anterior chamber.