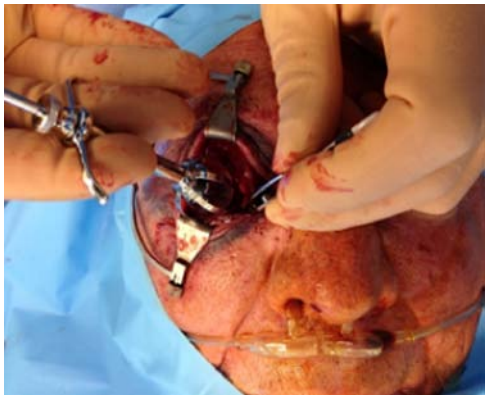

Fig. 5—Orbital prosthesis implant.

effect with the fulcrum at the anterior portion of the nasal bone, and thus causing severance near the insertion of optic nerve into the globe. Two alternate mechanisms described include entrance of an angular-shaped object along the medial aspect of the orbit to create a wedge effect in the orbit, or severing of the optic nerve and sheath by direct laceration or compression of these structures against the posterior orbital bones by the sharp corner of the incoming object. These 2 latter described mechanisms would lead to a longer disrupted optic nerve. A recent German article in *Ophthalmologe*[12] reported 2 cases of traumatic enucleation where the proposed accident did not match the injury. Both cases were cleared by psychiatry and raised the question whether interdisciplinary evaluation should be initiated in cases with suspicious traumatic injury as in cases of known auto-enucleation. Although our patient did not admit either psychiatric disorder or an assault, his history and mechanism are quite suspicious. We believe he was the victim of an assault because of the clean surgical cuts, lack of tethering structures, and long, intact optic nerve.

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Unilateral primary cutaneous amyloidosis of the eyelid masquerading as a chalazion

Amyloidosis is a constellation of disease entities that are characterized by the abnormal extracellular deposition and accumulation of protein and protein derivatives. Amyloidosis can be systemic (involving multiple organs) or it may present as a localized, organ-specific condition. In this correspondence, we report an unusual case of a primary localized amyloidosis of the eyelid, not involving the conjunctiva and without systemic amyloidosis or myeloma, but previously diagnosed and treated as a lower lid chalazion.

A 32-year-old Asian-Indian male presented with a painless, nodular mass on the right lower eyelid measuring 34 × 6 × 3 mm extending from the lateral to medial canthus (Fig. 1A). No ulceration, bleeding, or loss of lashes was present. There was no family history of multiple myeloma or any lymphoproliferative disorder. The increase in size of the mass was insidious and painless. He had been treated earlier with warm compresses and local antibiotic ointment application as treatment for a multiple chalazia of the lower lid. Slit-lamp examination showed normal anterior segments in both eyes; in particular, no conjunctival lesion was seen. An incisional biopsy was performed on the nodular lid mass. Hematoxylin and eosin–stained slides revealed subcutaneous deposits of amorphous eosinophilic material. Examination of the Congo red–stained slides showed red–orange staining of the deposits (Fig. 1B), which under polarized light exhibited apple-green birefringence, confirming the presence of amyloid deposits (Fig. 1C). Clinical examination of all systems was normal. Systemic

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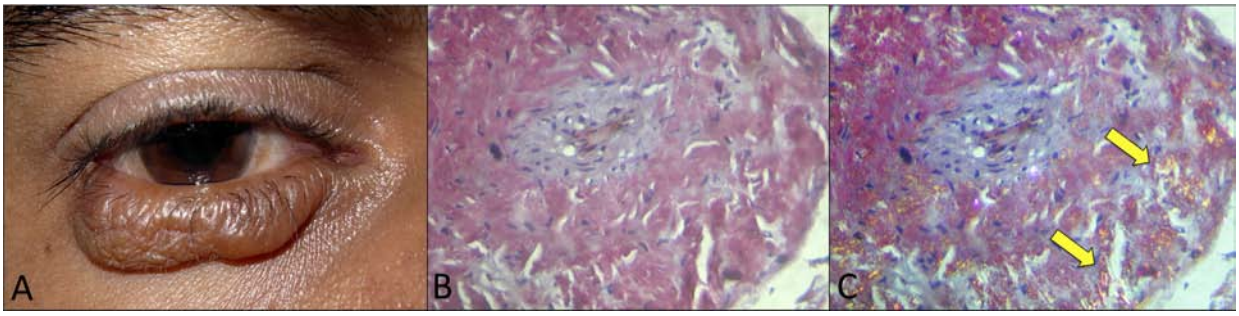

Fig. 1—A, External colour photograph showing a nodular mass on the right lower eyelid measuring 34 × 6 × 3 mm extending from the lateral to medial canthus. B, Congo red–stained slide shows red–orange staining of the deposits. Original magnification ×40. C, Congo red–stained slide under polarized light exhibiting characteristic apple-green birefringence of amyloid deposits (*yellow arrowheads*). Original magnification ×40.

investigations were as follows: hemoglobin was 13.6 g/dL (normal range, 13.5–17.5 g/dL), normal kidney and liver function tests, and serum albumin level was 3.7 g/dL (normal range, 3.5–5 g/dL). Urine examination was negative for Bence-Jones proteins. Serum protein and urine electrophoresis revealed no abnormal spikes. Examination of the bone marrow aspirate was normal, therefore ruling out multiple myeloma. Ultrasonography of the abdomen and echocardiogram were performed, which were unremarkable. Abdominal wall fat pad biopsy and rectal biopsy were negative for amyloidosis. The patient was therefore diagnosed to have primary, localized amyloidosis of the eyelid. The patient was advised to undergo local excision of the mass; however, he opted for conservative management when explained the chances of recurrence even after complete excision of the residual lesion. The mass was noted to be of the same size at last follow-up, which was 8 months after incisional biopsy.

Cutaneous amyloid deposits may occur in systemic forms or may be localized to the skin in the absence of other organ involvement. In addition to primary systemic amyloidosis, cutaneous amyloid lesions may be seen secondarily in myeloma-associated systemic amyloidosis, familial amyloid polyneuropathy, and Mediterranean fever.[1] Eyelid amyloidosis has been found to be associated with both primary and systemic amyloidosis. However, conjunctival amyloidosis usually is seen to be the manifestation of a local immunologic disorder, and affected patients rarely have systemic amyloidosis.[2] Hence our patient has been advised 6-month follow-up with an ophthalmologist, as well as an annual systemic evaluation as per the advice of a rheumatologist.

While discussing ocular adnexal amyloidosis, it is of interest to note that there have been reports of localized primary systemic amyloidosis of only the eyelid skin and those involving both – the conjunctiva and eyelids including eyelid skin.[1,3] However, unilateral primary localized amyloidosis of the eyelid alone without associated conjunctival lesion is an uncommon entity.[4] In fact, cutaneous involvement of the eyelid skin is considered to be virtually pathognomic of primary amyloidosis.[1]

Given the rarity of this condition, few reports have discussed the surgical approaches for localized amyloid deposition of the eyelid. The suggested modalities of treatment include en bloc removal of the involved eyelid tissues, superficial radiation, and, in advanced cases, no treatment.[5] Patrinely and Koch[6] used a spooned curette to debulk the mass through a subciliary incision to preserve the anatomical planes of the eyelid while treating advanced ocular adnexal amyloidosis. In cases such as ours, superficial curettage using a transcutaneous upper- and lower-eyelid blepharoplasty approach has been reported to be effective.[4]

In such cases, the chance of local recurrence must always be borne in mind. Furthermore, between 15% and 50% of patients with nodular cutaneous amyloidosis have or will have systemic amyloidosis.[1] Therefore, given the associated possible systemic associations, it is not only important to systemically evaluate in any diagnosed case of amyloidosis, but also continually follow up these cases. Amyloidosis, although rare, may be considered as a possible diagnosis while dealing with lid margin tumours; furthermore, it can mimic other common clinical conditions such as chalazia.

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Case of rapid bilateral cataract development in teenager using selective serotonin reuptake inhibitors

Antidepressants have long been investigated in correlation to cataract development. Quetiapine (Seroquel; AstraZeneca, Wilmington, Del.), an inhibitor of cholesterol biosynthesis, has been linked to lens opacities in beagle dogs.[1] However, later large-scale studies showed that quetiapine had no correlation to cataract formation, and regular eye examinations were deemed unnecessary.[2] Selective serotonin reuptake inhibitors (SSRIs) are the most commonly prescribed antidepressants in the world and have a relatively safe profile. A recent study demonstrated that SSRIs including fluvoxamine (Luvox; Abbott Laboratories, Chicago, Ill.) and venlafaxine (Effexor; Pfizer, Mission, Kans.) increase the risk for cataract formation in adults,[3] raising the possibility that closer follow-up for patients is needed. High levels of serotonin have been shown to cause cataracts in rats,[4] and high levels have been found in the aqueous humour of patients with cataracts. The effect of SSRIs on levels of serotonin in the eye and aqueous humour and the implication of SSRI use on adolescents was never pursued. Currently, there is no indication for regular ophthalmologic examinations for patients receiving fluvoxamine treatment. A review of the literature shows little is known about the correlation of SSRIs and cataract formation and its mode of action in the eye. Our case demonstrates potential association of rapid deterioration of visual acuity as a result of SSRI treatment in a teenager.

A 19-year-old female with a history of depression and anxiety presented with bilateral white cataracts. For the past 7 months the patient was taking 150 mg fluvoxamine daily and has been previously taking 75 mg quetiapine daily. The patient denies the use of any other medication in the past 10 years or any previous ocular symptoms.

The patient noticed deteriorating visual acuity in both eyes 3 weeks before presenting for an initial eye examination. She was diagnosed with decreased visual acuity to finger counting in each eye caused by bilateral cataracts and was referred urgently to our clinic. At presentation a week later, uncorrected distance visual acuity decreased to hand motion in each eye. Anterior segment examination revealed white mature cataracts bilaterally and no other abnormalities. Posterior segment ultrasound showed no retinal detachment and an axial length of 23.31 mm OD and 22.87 mm OS. The patient underwent phacoemulsification with posterior chamber intraocular lens implantation first on the right eye and 1 month later on the left eye. Both surgeries were uneventful. Intraocular lenses (Abbott Medical Optics, Santa Ana, Calif.) were implanted with no complications. Postoperative uncorrected distance visual acuity was 20/20 OU. The patient was discharged with yearly follow-up.

Serotonin is a monoamine neurotransmitter found primarily in the gastrointestinal tract and the central nervous system. Its function as a neurotransmitter is to regulate mood, appetite, and sleep. Serotonin is also found in the aqueous humour of patients with cataracts and glaucoma,[4,5] and serotonin binding proteins are found in the lens of animals.[6,7]

Although the mechanism of serotonin in relation to cataract formation is not fully understood, the involvement of serotonin in cataractogenesis has been demonstrated in animal models both at high concentrations and when inhibited.[8–10] It has been shown that high concentrations of serotonin can cause cataracts in rat eyes,[4] whereas several serotonin antagonists including RG 12915[9] and ICT 322[10] also have been shown to cause oxidative stress-induced cataractogenesis in rats. In the toad lens, serotonin inhibits an ATPase of the lens epithelium shown to contribute to lens transparency.[11] Serotonin has also been found in human aqueous humour at high levels in patients with cataracts and glaucoma,[4–6] but it was never measured in healthy individuals. Although the levels of serotonin are similar in patients with glaucoma and cataract, the metabolism and turnover of serotonin differs between those patients.[5] Melatonin, one of the downstream products of serotonin, also plays a role in cataract formation, but a protective one. Melatonin is sequentially synthesized from serotonin in the pineal gland and functions in the regulation of the circadian system. Melatonin also acts in the ocular lens as an antioxidant and radical scavenger, and has been shown to have protective properties against formation of cataracts in rats.[12,13] Although SSRIs have a relatively safe profile and few reported ophthalmologic sequelae resulting from their use, several studies have demonstrated that serotonin, melatonin, and their receptors play an important role in cataractogenesis.[4–13] Thus, the possibility of SSRIs affecting the concentration of serotonin and melatonin in the aqueous humour and the risk for cataract formation should be considered.