coaugulation.[1–3, 7] To the best of our knowledge, our patient is the first reported case of leukemia causing spontaneous suprachoroidal haemorrhage and acute angle closure glaucoma. The ocular haemorrhage is most probably explained on the basis of thrombocytopenia. The anterior segment inflammation or iritis that was evident on initial presentation may have been secondary to leukemic infiltration of his anterior segment. We would like to stress the importance of performing routine blood tests in patients presenting with unexplained ocular haemorrhage and the importance of determining the exact cause of an acute elevation of intraocular pressure.

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Sir,

Unilateral lattice dystrophy in an elderly patient
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Lattice corneal dystrophy is an inherited, primary, usually bilateral corneal amyloidosis characterised by refractile lattice lines with a double contour in the corneal stroma.[1] LCD is classified into at least four clinical subtypes.[2] LCD type I (Biber-Haab-Dimmer) is an autosomal dominant, bilaterally symmetrical corneal disorder that is characterised by numerous translucent fine lattice lines that are associated with white dots and faint haze in the superficial and middle layers of the central stroma. The symptoms appear during the first or second decades of life.[1,3] Visual acuity becomes progressively impaired and most patients require a corneal transplant by 40 years of age.[4] LCD type II is associated with systemic amyloidosis (Meretoja's syndrome).[5] The lattice lines in LCD type II are thicker and more peripheral than those of LCD type I. They extend from the limbus towards the central cornea involving the mid-stroma at the periphery to more superficial stroma centrally.[6] The visual acuity is good until later in life. LCD type III is a late onset LCD that appears with decreased vision in the fifth to seventh decades of life. Asymmetrical findings are common. Lattice lines extend from limbus to limbus, are thicker and are more easily seen with direct illumination than that of LCD type I.[4,7] LCD type III A differs from the type III by the presence of erosions, the occurrence in whites and the autosomal dominant inheritance pattern.

Though generally considered bilateral and symmetrical, unilateral and asymmetrical cases of LCD type I are reported.[8] We report a case of lattice dystrophy the lesions of which clinically appeared like LCD type I and predominantly involved one eye.

Case report

An 85-year-old male presented to us on 20th July, 2001 with the complaint of diminution of vision of the left eye of 3 years duration. He gave a history of undergoing cataract surgery in the right eye with good visual recovery. On examination, his visual acuity was 6/6 in the right eye and 6/36 in the left eye. The corneal examination in both eyes revealed climatic droplet keratopathy lesions. In the left eye, multiple lattice lesions were seen which were prominent on retroillumination (Figure 1). The lattice lines seen in the nasal cornea involved the anterior to midstroma of the cornea, whereas the lesions involving the middle of

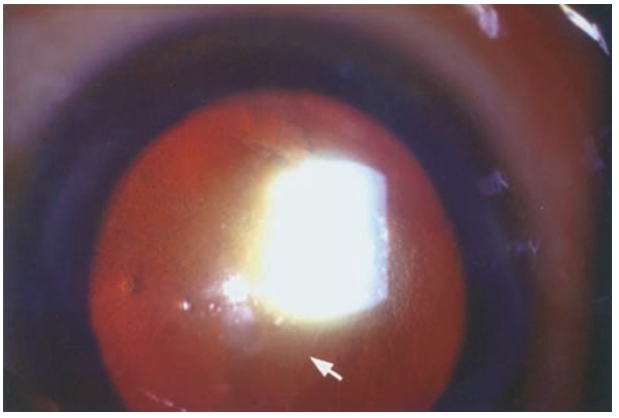

Figure 1 Retroillumination of left eye showing typical lattice changes.

cornea were mid-stromal. The lesions did not involve the limbus. In the right eye, faint lines were seen temporally, though they were not classical lattice changes. The anterior chamber was deep and quiet. There was pseudophakia in the right eye and 2+ nuclear sclerosis and 1+ posterior subcapsular cataract in the left eye. Fundus examination was within normal limits. The patient underwent cataract surgery in the left eye. The surgery was uneventful. One week after surgery, the eye was quiet and the vision in the left eye was 6/12.

Comment

Lattice corneal dystrophy is classically a bilateral condition. Isolated case reports of unilateral corneal dystrophy have been reported.[9–12] Raab *et al* reported a series of six cases of unilateral lattice corneal dystrophy classifying them as a sub-group of lattice corneal dystrophy.[8] All cases involved men between 31–80 years with an average age of 59 years. The patients were asymptomatic or had minimal symptoms. The visual acuity in the affected eye was 20/80 or better. Unilaterality, late onset, minimum symptomatology and preservation of relatively good visual acuity distinguish these patients from those with classical lattice dystrophy.

Sridhar *et al* reported three cases of unilateral lattice dystrophy.[13] Two cases presented in the early fourth decade and another in the early third decade of life. One of the patients was female and the other two were males. Clinically the lattice lesions were similar to classic lattice dystrophy of cornea (LCD type I). Two of the patients required penetrating keratoplasty because of poor vision. In one patient, the lattice changes developed in the other eye nearly 15 years after he was first seen. This case was a sixth case of the unilateral lattice series of Raab *et al*. The authors concluded that lattice changes can be rarely unilateral. The lattice even in unilateral cases may cause significant visual loss warranting penetrating keratoplasty. Lattice lesions may develop in the other eye many years later and this should be explained to all patients with apparent unilateral lattice corneal dystrophy.

Several reports have linked granular, lattice, granular-lattice (Avellino dystrophy) and Reis–Bucklers dystrophy to chromosome 5q31.[14–17] Missense mutations in BIGH3 gene in family members of these dystrophies mapping to 5q have been reported.[18] Mutations at codon 124 have been associated with LCD (arginine to cysteine). Some, but not all mutations occuring at the codon 124 hot spot appear to induce structural changes that favour amyloid formation. Mutations at eight other sites have also been responsible.[19]

Hirano *et al* reported two Japanese patients who were clinically diagnosed with late onset and sporadic lattice corneal dystrophy.[20] In one patient, the changes were seen only in one eye. Corneal opacity with gray colored nodular deposits in the central cornea and relatively thick lattice lines that extend from limbus to limbus in the mid-stromal layer were observed only in the right eye. These two cases were caused by the Leu 527 Arg mutation of TGFBI gene.

The case reported presented in the ninth decade of life with lattice changes predominantly in one eye. The lattice lesions were similar to those seen in LCD type I. In the other eye, faint lines were seen, but they were not classical lattice changes. The eye with lattice had an associated cataract. The patient regained good visual acuity after cataract surgery. The clinical features of this patient are similar to that of unilateral lattice dystrophy reported by Raab *et al*.

Based on these cases and cases reported by Sridhar *et al*,[13] we feel that unilateral lattice dystrophy with lesions resembling LCD type I can occur in two clinical types. The first type occurs predominantly in males and the patient presents late in life, has minimal symptoms and good visual acuity. The second type occurs early in life (third and fourth decade) and the visual acuity is impaired warranting penetrating keratoplasty. In these patients, lattice changes may develop in the other eye, later in life. Further genetic studies of unilateral lattice dystrophy patients with lesions resembling LCD type I are warranted.

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Sir,

Chiasmal cavernous angioma. a rare cause of progressive visual loss

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Intracranial cavernous angiomas are not uncommon.[1,2] Only 5% are supratentorial, most of which are intraparenchymal and are located in the cortical and subcortical ganglia. Infratentorial angiomas may be located within the brainstem, cerebellum, third or fourth ventricles or the posterior fossa.

Angiomas can very rarely develop within the substance of the optic nerve, optic tract and optic chiasm, where they produce acute or progressive loss of vision in one or both eyes. We report a case of chiasmal angioma presenting with progressive visual loss.

Case report

A 42-year-old man presented with a history of blurred vision in the right eye for 4 days. He had an unaided visual acuity of 6/4 in both eyes. Pupillary reactions and fundus examination were normal. He returned a week later with a history of decrease in colour vision in his right eye. Again, apart from mild colour desaturation, there was nil of note. In particular, he had no afferent pupillary defect or optic disc swelling. There was no pain on ocular movements. As he demonstrated Uhtoff's phenomenon, he was thought to have mild optic neuritis.

Visual fields demonstrated central loss in the right eye. Electrodiagnostic tests demonstrated a complete loss of pattern visual evoked potential (VEP) with preserved flash VEP response from the right eye. His right visual acuity subsequently dropped to 1/60 and he developed a right afferent pupillary defect and optic disc pallor. Magnetic resonance imaging (MRI) scan demonstrated a lesion at the right optic chiasm, which was thought to be a meningioma or a craniopharyngioma (Figure 1). Magnetic resonance angiography (MRA) was reported to be normal. The patient was referred to the neurosurgeons. He