5 Mckenzie WH. Tuberculosis of the conjunctiva. Report of a case. *Am J Ophthalmol* 1939; **22**: 744–749.
6 Salas D, Murthy S, Champ C, Hawksworth N. Primary tuberculosis of the conjunctiva. *Eye* 2001; **15**(Part 5): 674–676.
7 Cameron JA, Nasr AM, Chavis P. Epibulbar and ocular tuberculosis. *Arch Ophthalmol* 1996; **114**(6): 770–771.
8 Flinnoff WC. Ocular tuberculosis, experimental and clinical. *Arch Ophthalmol* 1924; **53**: 130–136.
9 Grunert C. A contribution to the subject of tuberculosis of the conjunctiva. *Arch Ophthalmol* 1899; **28**: 540–556.
10 Eyre JWH. Tuberculosis of the conjunctiva: its etiology, pathology and diagnosis. *Lancet* 1912; **1**: 1319–1328.
11 Aclimandos WA, Kerr-Muir M. Tuberculous keratoconjunctivitis. *Br J Ophthalmol* 1992; **76**: 175–176.

AG Zaborowski[1], BN Gundry[1], ME Masenya[2] and L Visser[1]

[1]Department of Ophthalmology, Nelson R Mandela School of Medicine, Durban, South Africa

[2]Department of Anatomical Pathology, Nelson R Mandela School of Medicine, 52 Umbilo Road, Congella, Durban, KwaZulu-Natal 4013, South Africa

Correspondence: A Zaborowski,
Tel: +27 31 3603450;
Fax: +27 31 5727634.
E-mail: azaborowski@mweb.co.za

Eye (2006) **20,** 978–979. doi:10.1038/sj.eye.6702090; published online 9 September 2005

Sir,

An atypical presentation of lattice corneal dystrophy in a patient with juvenile glaucoma

Lattice corneal dystrophy (LCD) is a bilateral, local amyloidosis, characterized by variation in the corneal manifestations, clinical course, and genetics.[1] LCD has been attributed to TGFBI (transforming growth factor, beta-induced) gene on human chromosome 5q31 (OMIM No. 601692). LCD type II is associated with secondary open-angle glaucoma in the context of familial amyloidosis, Finnish (FAF).[2] We report a case with the corneal findings of LCD type II but without any systemic involvement, in a patient with juvenile glaucoma.

Case report

A 26-year-old woman was referred to the glaucoma clinic because of elevated intraocular pressure (IOP) found in a routine eye examination. The patient was otherwise healthy with no significant family history, no visual complaints, or any history of recurrent erosions.

Her best-corrected visual acuity was 20/20 in both eyes. Corneal sensitivity was normal. Slit-lamp examination revealed refractile, radial filamentary lines in the anterior corneal stroma creating a lattice-work pattern, with sparing of the central cornea (Figure 1). No opacification or corneal haze was noted.

Gonioscopy showed high iris insertion and prominent iris processes in both eyes (Figure 2). IOP was 34 mmHg in the right eye and 29 mmHg in the left eye. Fundus examination revealed an arcuate nerve fibre loss between the first-order inferior temporal vessels of the left eye. Visual fields were normal. No change in the findings of the cornea, fundus, and visual fields was noted within the 5 years of follow-up. IOP remained controlled under antiglaucoma treatment.

Familial and systemic amyloidosis were ruled out by extensive physical examination and laboratory work-up. The patient did not present any skin lesions, macroglossia, arrhythmias or megalocardy, hepatosplenomegaly, arthritis, and cranial or peripheral neuropathy. There was no evidence of pulmonary, haematopoetic, or renal involvement. ESR, CRP, and immunoglobulins levels were within normal limits. Echocardiography was normal. Skin biopsy was negative

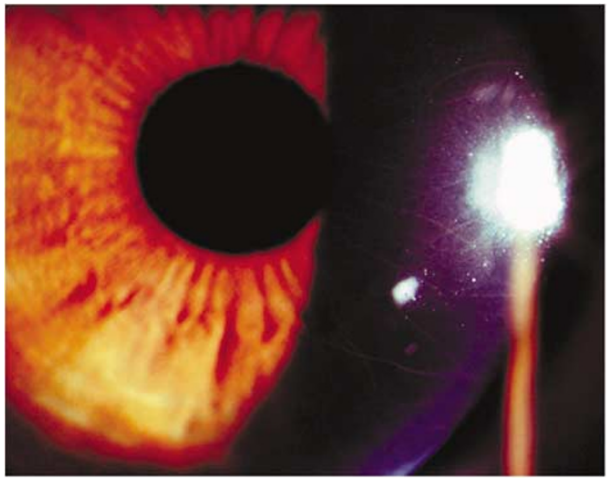

Figure 1 The patient's cornea is shown with refractile and discrete filamentary lines in the anterior stroma creating a lattice-work pattern. A white round dot in the anterior corneal stroma corresponding to an epithelial irregularity is remarkable. Note the absence of opacification, as well as the sparing of the central cornea.

Figure 2 High iris insertion and prominent iris processes seen on gonioscopy.

for amyloid deposition. Blood samples were taken for DNA analysis after voluntary informed signed consent was obtained using the Thessaloniki IRB consent. Screening for the two mutation hotspots within the TGFB1 gene using sequencing did not reveal the common mutations present in the majority of patients with LCD (R124L, R124C, R124H, R555W, R555Q).

Comment

Despite the typical for FAF corneal picture, vision preservation and age of onset in that patient, the absence of systemic involvement within the 5 years of follow-up is indicative of a localized form of LCD. Furthermore, our patient suffered from juvenile and not secondary glaucoma. These clinical findings are not representative of any of the common isolated forms reported in the past.[1,3] This is suggestive of a more pleomorphic appearance of the isolated LCDs, and it is in agreement with conclusions made by Stewart *et al*[4] on the validity of LCDs' current classification.

References

1 Afshari NA, Mullally JE, Afshari MA, Steinert RF, Adamis AP, Azar DT *et al*. Survey of patients with granular, lattice, avellino, and Reis–Bucklers corneal dystrophies for mutations in the BIGH3 and gelsolin genes. *Arch Ophthalmol* 2001; **119**(1): 16–22.
2 Starck T, Kenyon KR, Hanninen LA, Beyer-Machule C, Fabian R, Gorn RA *et al*. Clinical and histopathologic studies of two families with lattice corneal dystrophy and familial systemic amyloidosis (Meretoja Syndrome). *Ophthalmology* 1991; **98**(8): 1197–1206.
3 Mannis MJ, De Sousa LB, Gross RH. The stromal dystrophies, Lattice dystrophy. In: Krachmer JH, Mannis MJ, Holland, Palay (eds). *Cornea*, Section 3. Mosby Inc.: St Louis MO, 1998.
4 Stewart H, Black GC, Donnai D, Bonshek RE, McCarthy J, Morgan S *et al*. A mutation within exon 14 of the TGFBI (BIGH3) gene on chromosome 5q31 causes an asymmetric, late-onset form of lattice corneal dystrophy. *Ophthalmology* 1999; **106**(5): 964–970.

E Tsina[1], N Udar[2], K Small[2] and F Topouzis[1]

[1]B′ Department of Ophthalmology, Aristotle University of Thessaloniki, General Hospital 'Papageorgiou', Periferiaki odos Thessalonikis, N. Efkarpia 56403, Thessaloniki, Greece

[2]Jules Stein Eye Institute, David Geffen School of Medicine, UCLA, Los Angeles, CA, USA

Correspondence: F Topouzis,
Tel: +30 2310 994920;
Fax: +30 2310 839497.
E-mail: ftopouzis@otenet.gr

Eye (2006) **20,** 979–980. doi:10.1038/sj.eye.6702092; published online 16 September 2005