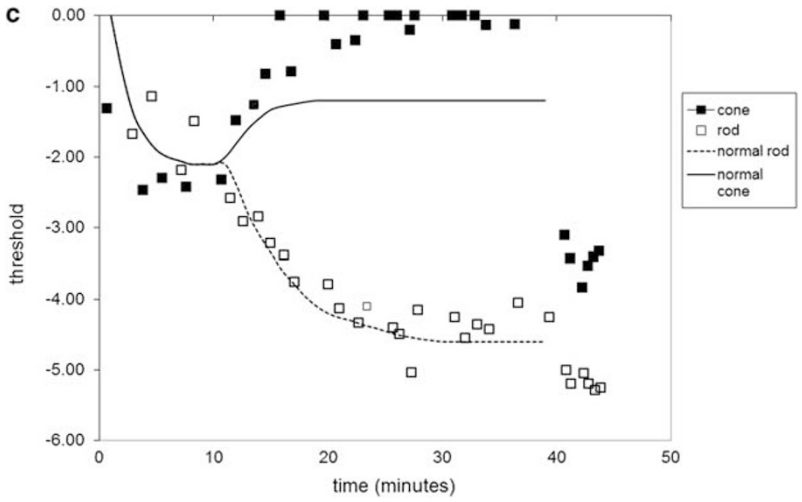

Figure 1 Continued.

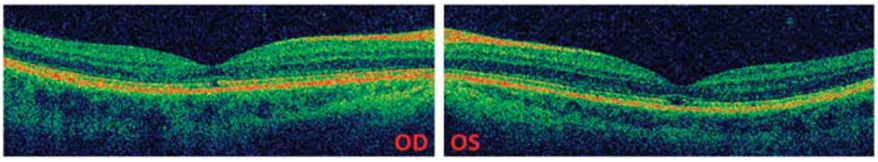

Figure 2 The 3D-OCT showing bilateral disruption of the photoreceptor inner segment/outer segment layer and intact outer limiting membrane.

thresholds by dark adapting rods and established the diagnosis of E-RCI. The condition is usually benign and previous reports have not shown progression.[3–5] The continuing reduction in VA in the present case and the markedly reduced PERG and mfERG suggested progressive macular dysfunction, which was confirmed by 3D-OCT demonstration of bilateral disruption of the photoreceptor IS/OS layer. To our knowledge, this is the first case of progressive macular dysfunction in association with E-RCI. It demonstrates the value of electrophysiology, psychophysics, and OCT in revealing functional and structural abnormalities despite the presence of a normal fundus exam.

Conflict of interest

The authors declare no conflict of interest.

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Sir,

A case of African crystalline maculopathy

We present the first reported case of West African crystalline maculopathy in an East African patient from Egypt.

Case report

A 67-year-old woman was referred for evaluation of bilateral retinal crystalline deposits. She had no systemic or ocular complaints. Past history revealed osteoarthritis and hypertension. Her medications were ramipril, atorvastatin, diclofenac, alendronate, and multivitamins. The patient was born in Egypt and moved to Canada 22 years prior to presentation. She reported a typical North American diet and denied ever eating kola nuts or other West African foods, such as cassava greens, cassava roots, afanga greens, plantains, or palm oil.

On examination, best-corrected visual acuity was 20/50 OD and 20/60 OS. Intraocular pressures were 17 mm Hg OU. Anterior segments were normal OU. Ophthalmoscopy showed bilateral iridescent yellow-green crystals in the inner retinal layers close to the fovea and retinal pigment epithelial changes in the macula (Figure 1). Fluorescein angiography demonstrated focal staining of drusen and no fluorescein leakage. Optical coherence tomography (Cirrus HD-OCT; Carl Zeiss Meditec Inc., Dublin, CA, USA) showed the crystalline deposits to be at the level of Henle's layer in the foveal region OU (Figure 2).

A pedigree analysis was not performed, but there was no family history of West African heritage or consanguinity. Select family members including two of the patient's sisters were examined and had no evidence of a crystalline maculopathy. The patient's husband was also examined because he shared a similar dietary history and had no macular crystals.

Toxic, genetic, and degenerative causes of crystalline maculopathy were considered and excluded based on history and examination.[1] There was no history of tamoxifen, canthaxanthine, or nitrofurantoin use, methoxyfluorane exposure or intravenous drug abuse. The patient was negative for diabetes and renal impairment, eliminating hyperoxaluria, cystinosis, methoxyfluorane, and Sjogren–Larsson syndrome as possible causes.[1] Retinal detachment and telangiectasis were absent on ophthalmoscopic and angiographic examination. Retinal pigment epithelial abnormalities characteristic of tamoxifen retinopathy, Bietti dystrophy, and cystinosis were also notably absent.[1]

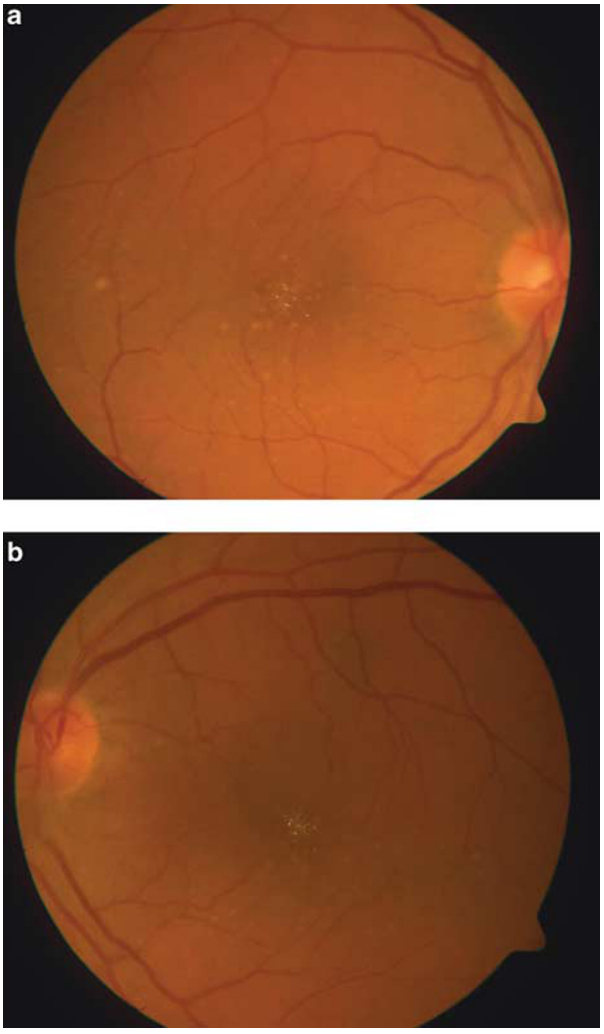

Figure 1 (a) Colour fundus photograph of the right eye. (b) Colour fundus photograph of the left eye.

Comment

West African crystalline maculopathy (WACM) was first reported in 2003 in a series of elderly members of the Nigerian Igbo Tribe. These West African patients had asymptomatic yellow-green refractile crystals

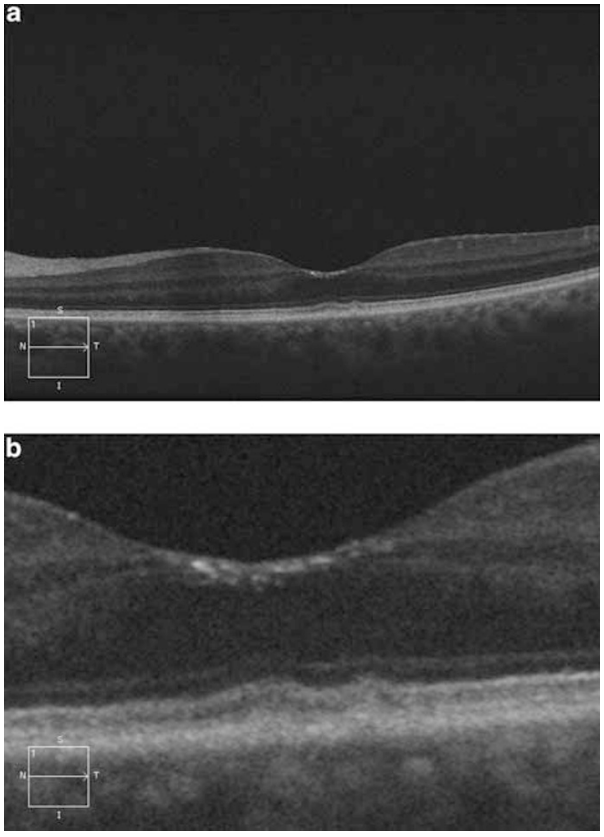

Figure 2 Optical coherence tomography of the left eye showing refractile deposits in Henle's layer.

in the superficial fovea.[2] Kola nut ingestion was suggested as a potential cause. Subsequent reports of patients with similar clinical findings have expanded to other West African countries, including Liberia, Cameroon, Ghana, and Sierra Leone.[1,3,4] A recent report showed the crystals to be in Henle's layer using optical coherence tomography.[3] The pathogenesis of these crystals is unknown, although retinal vascular disease, with breakdown of the blood–retinal barrier, has been suggested as a possible cause.[3,4] Regional food and diet peculiarities have also been considered as possible aetiologic agents.[2,3]

In contrast to previously published cases of WACM, our patient originated from East Africa, in Egypt, a country which shares ethnic and historical connections with the Middle East and is culturally distinct from West Africa.[5] Given the similarities of our East African patient to previously reported cases of WACM, we believe the more appropriate term for this condition may be African crystalline maculopathy.

Conflict of interest
The authors declare no conflict of interest.

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Sir,

Reply: the report 'Spontaneous dislocation of an Artisan phakic IOL causing corneal decompensation requiring an endothelial graft' by Harsum *et al*

We read with great interest this report describing spontaneous dislocation of an Artisan IOL.[1] As the authors suggest, this is a rare event and normally occurs following direct or indirect trauma. We were curious about their comments regarding the contralateral unaffected eye. The assertion has been made that there is decentration of this IOL and atrophic iris at the points of enclavation. Are the authors suggesting that this IOL is unstable?

Having used a large number of these IOLs in aphakic eyes, we would argue that the position of this IOL, although not perfectly centred, is completely acceptable. This IOL is unlikely to have moved since implantation. Artisan implantation is a fairly complex bimanual procedure. The centration of the Artisan IOL during insertion can be slightly variable and is influenced by several factors, including the effect of viscoelastic on anterior chamber depth, pupil size, and position. However, we have never known these small variations to affect final visual outcome. The enclaved iris in Figure 1b shows no clearly visible signs of atrophy as suggested.[1] On the right fixation point, the amount of enclaved iris seems just about adequate. The left fixation point I would describe as abundant!

True decentration of Artisan IOLs has been described involving a downward translation of the entire IOL haptic through the stroma of the enclaved iris.[2] The incidence has been reported as 2.5% over approximately 5 years. It was postulated that this might be secondary to stromal atrophy at the fixation points, which was quantified using anterior segment OCT. It appears this process can occur despite generous fixation, however, re-fixation of the haptics is not always necessary.

Conflict of interest
The authors declare no conflict of interest.

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