four periocular, and one brow haemangioma). We used a starting dose of 1 mg/kg per day in three divided doses, increasing it to 2 mg/kg per day after 1 week if tolerated. Six of our patients have responded very well to the treatment and have had no side effects to date. The propranolol was stopped in one case owing to loss of appetite, which coincided with the commencement of propranolol.

One point we wished to make is to raise awareness of the fact that propranolol elixir can be dispensed in four concentrations (5 mg/5 ml, 10 mg/5 ml, 40 mg/5 ml and 50 mg/5 ml). We have had one drug error where the prescription was correctly written, but the dispensing community pharmacist gave the 50 mg/5 ml propranolol. This resulted in the infant being given 10 times the prescribed dose. He was admitted for monitoring with no ill effect. We feel it is important that parents are made aware of the different formulations available so that similar possible errors are prevented.

Conflict of interest

The authors declare no conflict of interest.

Reference

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Sir,

A case of African crystalline maculopathy

We read with great interest the report[1] describing an isolated case of crystalline maculopathy in a North-East African individual from Egypt. West African Crystalline Maculopathy (WACM) has only been seen in a single population isolate to date (The Yoruba and Igbo tribes of West Africa), making a genetic predisposition likely. While the genetic makeup of Egyptians is interesting, genetically, they are very different to the West Africans; a combination of geographical location and history of modern and ancient migration have resulted in a complex and diverse gene pool of European, Middle Eastern, and African genetic characteristics.[2]

The central foveal crystalline deposits in this case are indeed interesting; however, we believe it differs from the described features of WACM. Firstly, the appearance and distribution of the crystals are different from that seen with published cases of WACM. Our previously published series, as well as of other authors have shown WACM to consist of intra-retinal crystals (Figure 1),[3–5] whereas, in this case, the crystals appear very localised to the inner retina at the foveola. Commonly in WACM, there is also concurrent co-pathology that affects the blood-retinal barrier such as diabetes.

The authors have described and excluded other causes of crystalline deposits in this report; therefore, although we believe this case does not exhibit the recognised changes seen in WACM, it could be a different phenotype. However, it is difficult to make further comment from just a single index case. We now have accumulated an unpublished case series of over 50 patients with WACM, who all, without exception, originate from West Africa, and exhibit the described features. It is premature to consider a new label for this condition from just a single albeit interesting case.

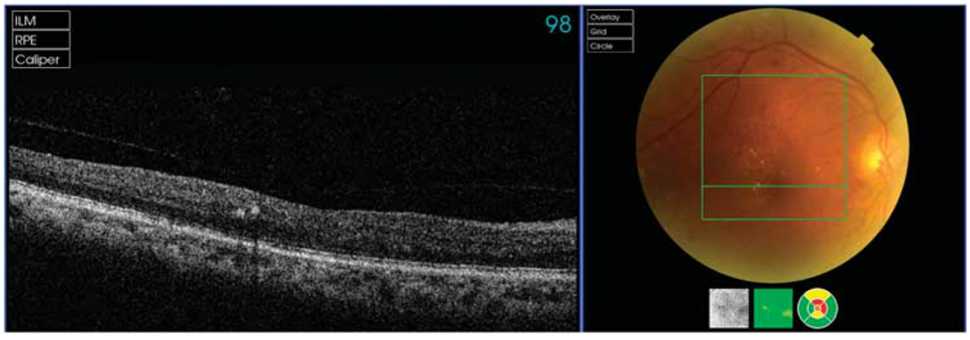

Figure 1 Fundus photograph demonstrating West African Crystalline Maculopathy, with OCT image showing retinal localisation of crystals.

Conflict of interest
The authors declare no conflict of interest.

References

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Sir,
Reply to 'a case of African crystalline maculopathy'

We thank Drs A Dhital and M Mohamed[1] for their interest in our report[2] and for their stimulating comments.

The distribution of the crystals in our case exactly matches that described by Sarraf *et al*[3] in their original series first describing West African crystalline maculopathy (WACM) in 2003. They describe the crystals as superficial, refractile, yellow or green in appearance, bilateral and asymmetric in distribution, and focally deposited within the fovea.

Two published reports to date have documented the location of the crystals using optical coherence tomography (OCT). The first revealed multiple discrete hyperreflective lesions in the innermost retinal layers in the region of the fovea (Figure 1).[4] The second report visualized the crystals in the layer of Henle, the foveal portion of the outer plexiform layer of the retina (Figure 2).[5] Similarly, our report localized the crystals to Henle's layer in the fovea (Figure 3).[2] Given that the fovea lacks the inner retinal layers, the layer of Henle represents the innermost layer in the fovea, which explains the superficial location of the crystals.[6] In the authors' letter, the crystals appear to be located, at least in part, in Henle's layer. However, their scan appears to be through a perifoveal location where the inner retinal layers are still present, which explains the deeper location of the crystals.

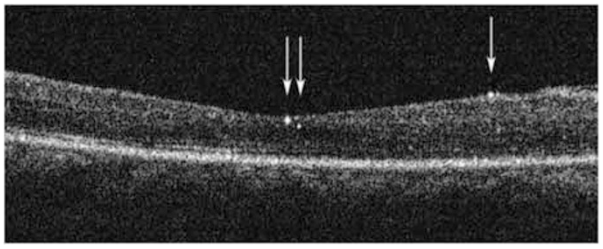

Figure 1 Optical coherence tomography of fovea of patient with WACM showing refractile crystals (arrows) in inner foveal layers, reproduced from Baker *et al*.[4] Copyright 2009 Wolters Kluwer Health. All rights reserved.

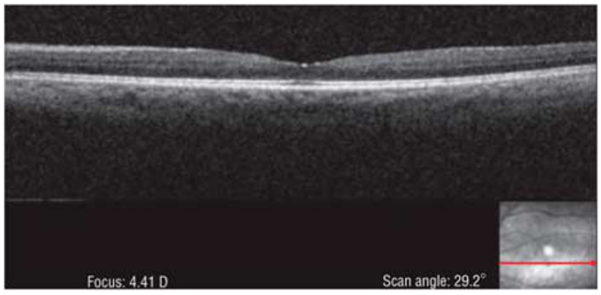

Figure 2 Optical coherence tomography of fovea of patient with WACM showing refractile crystals in inner foveal layers, reproduced from Rajak *et al*.[5] Copyright 2009 American Medical Association. All rights reserved.

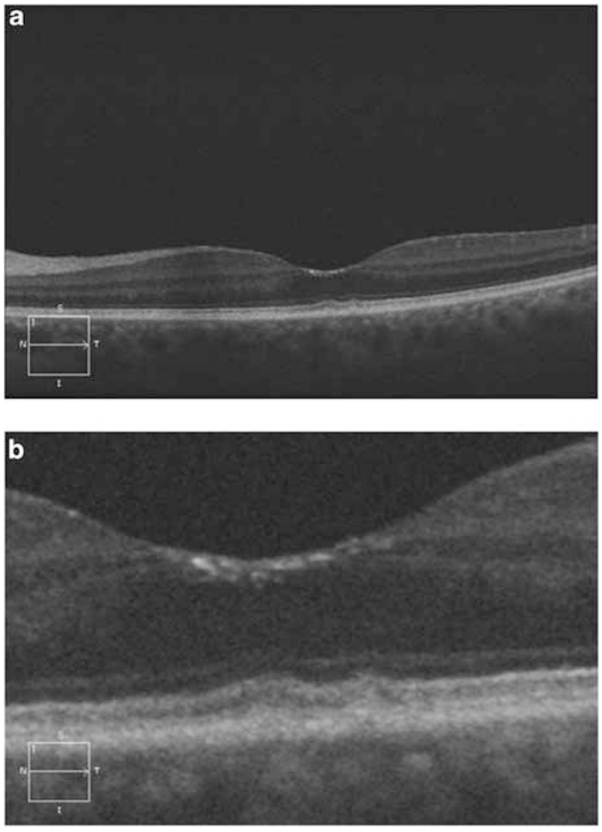

Figure 3 (a and b) Optical coherence tomography of fovea of patient with WACM showing refractile deposits in inner foveal layers, reproduced from our original article.[2]