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Sir,

Acute macular neuroretinopathy: anatomic localisation of the lesion with high-resolution OCT

Acute macular neuroretinopathy has a poorly understood pathology and pathogenesis. Recent evidence has indicated a lesion in the outer retina which is now confirmed in this report using high resolution optical coherence tomography.

Case report

A 27-year-old woman presented with a 6-day history of acute painless blurring of vision in both eyes commencing 2 days after a flu-like illness. She described discrete paracentral scotomas in both eyes and was able to draw these precisely on an Amsler chart (Figure 1). Visual acuities were 6/9 bilaterally with normal pupils and clear ocular media. There was subtle, patchy red discolouration at the macula bilaterally, and a solitary haemorrhagic cotton wool spot in the right eye. Scanning laser ophthalmoscopic (SLO) infra-red imaging showed dark areas corresponding to the abnormalities on Amsler (Figure 2). High-resolution optical coherence tomography (OCT) showed disruption of the photoreceptor inner /outer segment junction in the same areas, with associated focal thinning of the outer nuclear layer (Figure 3). After 8 months, the patient still complained of paracentral scotomas, which were less dense and slightly more diffuse, corresponding to improved OCT appearance (Figure 3) and less discreet changes on the infra-red images (Figure 4). Visual acuities remained 6/9 bilaterally.

Comment

Acute macular neuroretinopathy (AMNR) is a rare and poorly understood condition. The original description by

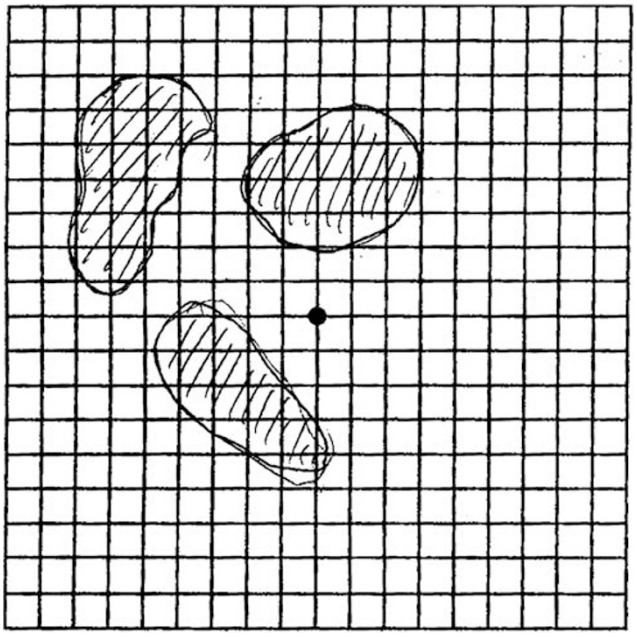

Figure 1 Left eye Amsler grid showing scotomas at presentation.

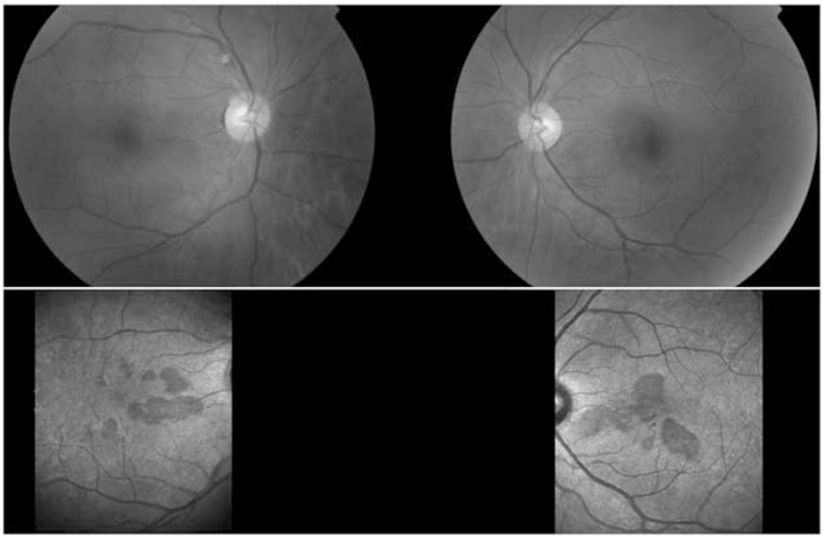

Figure 2 Colour fundus photographs and infra-red images at presentation, 2 weeks after the onset of symptoms. Subtle red perifoveal lesions are easily visualised on infra-red imaging. A haemorrhagic cotton wool spot is seen on the right superotemporal arcade.

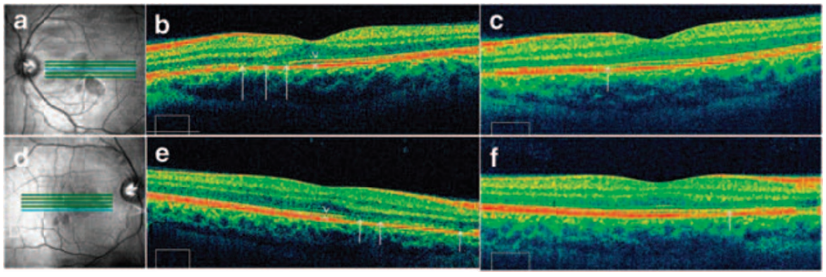

Figure 3 High-resolution OCT images (Cirrus HDOCT, Carl Zeiss Meditec, Jena, Germany). (a–c) Right macula. A 6-mm horizontal raster scan, as illustrated by the blue line on the red-free scout image (a), is shown at presentation (b) and after 8 months (c). An area of focal disruption of the photoreceptor inner/outer segment junction is seen (arrows) with thinning of the outer nuclear layer, corresponding to the lesion seen on red-free imaging. The normal reflectivity of the inner/outer segment junction in an unaffected area is indicated by the arrowheads. Partial reconstitution of this layer is seen after 8 months. (d–f) Left macula. The same abnormality is shown (arrows) with arrowheads again showing the normal photoreceptor inner/outer segment junction. After 8 months, only a small area of residual abnormality is seen.

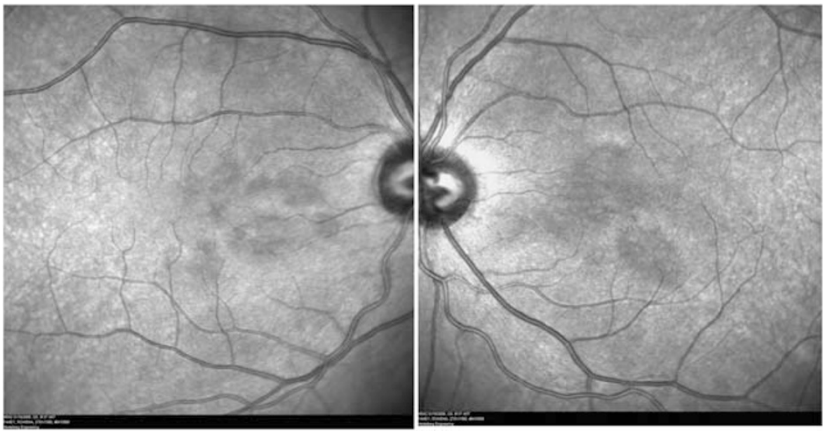

Figure 4 : Infra-red reflectance images 8 months after the onset of the symptoms showing partial resolution of the abnormalities.

Bos and Deutman[1] has been followed by numerous reports implicating precipitants including, most commonly, an acute viral illness[1,2] but also the administration of vasoconstricting drugs[3] and acute systemic hypotensive episodes.[4] Although Bos and Deutman[1] originally proposed a lesion in the inner retina,

subsequent observations have suggested the outer retina to be the location of the pathology.[2,5,6] One or two retinal flame-shaped haemorrhages are also often seen.[2] A recent report showed disturbance of the inner/outer segment junction on ultra-high-resolution OCT in a proposed case of AMNR,[7] but the features of the case were not typical (age over 50 years, unilateral non-classical lesion, and no predisposing event). The high-resolution OCT images in our patient clearly show focal abnormality in the photoreceptor layer, corresponding to the lesions on infra-red imaging, with preservation of inner retinal architecture. Whether a direct viral effect or immunologic phenomenon is responsible remains unclear, although abnormalities of choroidal perfusion may be responsible for cases associated with the use of adrenaline or noradrenaline (with or without antecedent acute systemic hypotension). This report is further evidenced that the pathology in this unusual condition is located in the outer retina/photoreceptors and not, as was originally thought, in the inner retina. The condition may perhaps be more accurately described as 'acute macular outer retinopathy'.

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Sir,

Flecked retina associated with ring 17 chromosome

Ring chromosome 17 was first described in 1970. There have been 4 previous reports of flecked retina associated with ring chromosome. We report another case of ring chromosome 17 with flecked retina.

Case report

We present a 25-year-old boy, who presented 23 years ago with myoclonic seizure, learning disability, and developmental delay. There were no signs of dysmorphism or any skin lesions. CT scan of the head showed no definite abnormalities. No family history of seizures or visual problems were reported.

Ocular examination at the age of 16 years revealed VA of 6/6 in both eyes. Low-frequency jerk nystagmus was apparent on dextroversion. Fundoscopy revealed well-defined white foveal flecks at the level of retinal pigment epithelium in both eyes (Figure 1).

Karyotype analysis (Figure 2) showed 46 XY, r(17)(p13.3q25) with no obvious loss of genetic material. Fluorescence *in situ* hybridisation studies (Figure 2) using the Oncor D17s379 probe from the Miller–Dieker chromosome region on 17p13.3, revealed no deletion. Probes within 300 kb of each telomere showed signals on normal chromosome 17 only, suggesting that at least 300 kb of material has been lost from each arm.

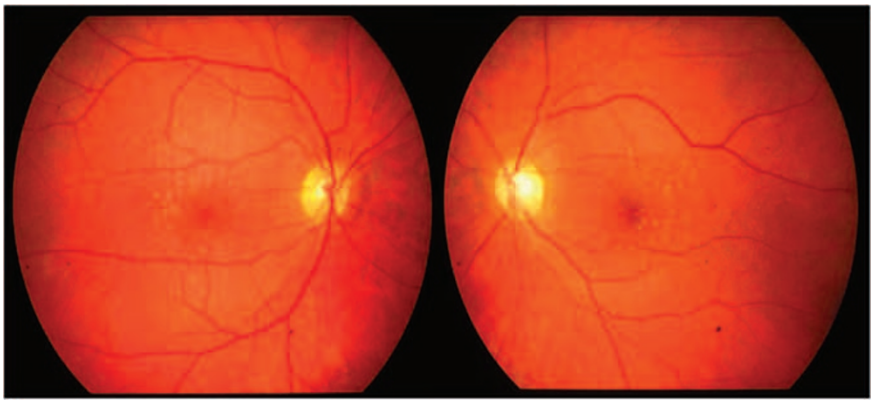

Figure 1 Fundus photographs of reported patient showing well-defined white foveal flecks at the level of the retinal pigment epithelium.