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Sir,

Foveal schisis with Mizuo phenomenon: Etio-

pathogenesis of tapetal reflex in X-linked retinoschisis Mizuo phenomenon has been rarely reported in juvenile X-linked retinoschisis (XLR).¹ We report a case of XLR with peripheral Mizuo phenomenon² which presented as isolated foveal schisis.

Case report

A 16-year-old male presented with diminution of vision OS since 1 year. He had no history of nyctalopia. Best-corrected visual acuity was 6/9 OD and 6/24 OS. Colour vision was normal. Fundus evaluation showed dull foveal reflex and a tapetal reflex in the periphery. After 5 h of dark adaptation, the tapetal reflex disappeared (Mizuo phenomenon) (Figure 1), which reappeared within a minute of light exposure. Full-field electroretinogram (Metrovision, France) showed reduction of 'b'-wave amplitude of combined maximal scotopic response and reduced cone ON–OFF responses (Figure 2). Optical Coherence Tomography (OCT) showed intraretinal foveal schisis (Figure 3).

Comment

Muller cells regulate extracellular K^+ ion concentration in the retina.³ In XIS, Muller cell dysfunction occurs secondary to the splitting of the nerve fibre layer. This causes defective inward transportation of K^+ ions from the inner and outer plexiform layers. The excess of extracellular K^+ ions causes Mizuo phenomenon.¹ But the fact that most patients with XLR do not demonstrate tapetal reflex is intriguing. The nonuniform distribution of K^+ channels across muller cell in retina⁴ and

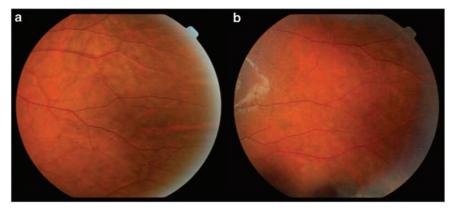


Figure 1 Fundus photograph of the left eye. (a) Tapetal reflex in the temporal periphery in the left eye. (b) Disappearance of the reflex following prolonged dark adaptation.

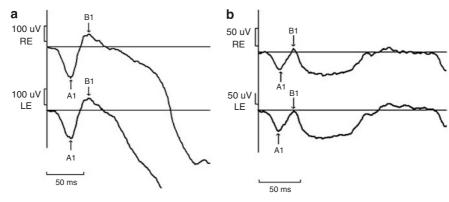


Figure 2 Full field electroretinography recordings. (a) Forty percentage reduction in 'b' wave amplitude of combined maximal scotopic response was noted in both eyes. (b) Reduced cone ON and OFF bipolar response in both eyes.

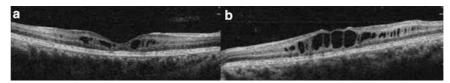


Figure 3 OCT from both eyes. (a) OCT from the right eye showing foveal schitic changes with macular atrophy. (b) OCT from the left eye shows intraretinal bridging columns in between hyporeflective spaces classical of foveal schisis.

differential affection of muller cell segment in XLR are possible explanations. This suggests that if adequate numbers of K^+ channels are intact, the reflex is absent. As greater numbers of K^+ channels are affected, Mizuo phenomenon becomes demonstrable wherein excessive \mathbf{K}^+ ions are cleared during dark adaptation. When most of the K^+ channels are affected, the tapetal reflex becomes constant, as there is persistent K⁺ ion backlog. Previous reports of increased tapetal reflex with time¹ suggest progressive K⁺ channel damage in XLR. Tapetal reflex disappeared following vitrectomy and posterior hyaloid peeling⁵ in XLR. The disruption of K⁺ channels at muller cell foot plate during the surgical procedure could have increased the inward conductance of $K^{\scriptscriptstyle +}$ ions trapped within dysfunctional muller cells causing disappearance of the reflex. The selective reduction of 'b' wave of combined maximal ERG and foveal schisis on OCT confirmed diagnosis of XLR in the absence of peripheral retinoschisis and presence of Mizuo phenomenon.

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Sir,

Comment on central retinal artery and vein collapse pressure in giant cell arteritis *vs* non-arteritic anterior ischaemic optic neuropathy

Jonas and Harder¹ measured the central retinal artery and vein collapse pressure in two groups of patients with anterior ischaemic optic neuropathy (AION). The aim of the study was to evaluate the role of these ophthalmodynamometric measures in differentiating an arteritic from a non-arteritic aetiology.

The giant cell AION group entry criteria need some clarification. Did all patients exhibit the classic symptoms of giant cell arteritis (GCA) as well as elevated inflammatory markers and a positive temporal artery biopsy? When a patient presents with an AION and the classical features of GCA, the diagnosis is not usually challenging.

The issue is whether this change in central retinal artery collapse pressure has the same sensitivity in patients where there are no other clinical features of GCA. In occult GCA, the underlying diagnosis of vasculitis of an AION can be challenging² and it is in these circumstances where the clinical value of this ophthalmodynamometric test would be useful.

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Reply to Dr Aristodemou

The authors would like to thank Dr Aristodemou for his interest in our study.¹ All patients with giant cell arteritis who were included into the study fulfilled the inclusion criteria for acute arteritic anterior ischaemic optic neuropathy, which were a positive temporal artery biopsy showing the typical granulomatous inflammation with giant cells, an erythrocyte sedimentation rate markedly above the age-related normal value, an elevated C-reactive protein level in the serum, clinical symptoms with a sudden and marked loss in vision, headache often persisting for weeks and months, an acute optic disc swelling with an unsharp and prominent optic disc border, few haemorrhages, and often a rather whitish ischaemic appearance of the swollen neuroretinal rim. The authors agree with Dr Aristodemou that when a patient presents with these typical features of arteritic anterior ischaemic optic neuropathy, the diagnosis is usually not very challenging.

Since the study included only patients with acute arteritic anterior ischaemic optic neuropathy exhibiting the typical ophthalmoscopic and laboratory findings, the authors have unfortunately no ophthalmodynamometric data of patients with occult giant cell arteritis, although without doubt, it would clinically be helpful.² As