Enhanced visualization of acute macular neuroretinopathy by infrared imaging and spectral optical coherence tomography

Dear Editor,

Acute macular neuroretinopathy (AMNR) is a rare condition of unknown etiology, first described in 1975 by Bos and Deutman.[1] It is characterized by subacute visual impairment that can either be transient or permanent, generally seen in young women, unilaterally or bilaterally.[1,2] Ophthalmoscopically round or oval parafoveal, brown-reddish lesions are typical. Red-free photography, scanning laser ophthalmoscopy (SLO), and optic coherence tomography (OCT) have been introduced for better evaluation of AMNR lesions.[3-5]

A 22-year-old female patient complained of visual impairment in her right eye for 2 days. She was diagnosed with familial mediterranean fever 14 years ago. Best corrected visual acuity was 2/10 OD and 10/10 OS. Anterior segment examination and intraocular pressure were unremarkable. The pupillary reflex was normal and afferent pupillary defect was not detected. Results of color vision testing with Ishihara plates were 3/15 in the right eye and 15/15 in the left eye. Fundoscopy revealed no obvious changes [Fig. 1a]. However, there was a dark macular lesion in 1 disc diameter on the infrared reflectance (IR) image [Fig. 1b]. Fundus autofluorescence findings were unremarkable for both eyes [Fig. 1c]. Fundus fluorescein angiography (FFA) with Heidelberg retinal angiography-2 showed subtle retinal pigment epithelium defect [Fig. 1d]. Visual field analysis with Goldmann perimetry showed paracentral scotoma. Macular evaluation with spectral OCT revealed disruption of IS/OS junction and thinning of outer nuclear layer with increased reflectivity [Fig. 1e].

On the first month of presentation, best corrected visual acuity (BCVA) was 3/10, color vision testing was 15/15 with Ishihara plates in the affected eye; there was neither pupillary reflex abnormality nor afferent pupillary defect. OCT showed thinning of the outer nuclear layer with subfoveal IS/OS disruption.

After 3 months, BCVA was 3/10 in the right eye. IR imaging showed attenuation of the lesion [Fig. 2a]. Spectral OCT demonstrated realignment of the outer photoreceptor layer and the IS/OS junction besides focal thinning of the outer nuclear layer [Fig. 2b].

AMNR is a rare condition that had been reported primarily among young women in their child-bearing ages.[1] Several risk factors were defined like hypertension, acute inflammatory processes, oral contraceptive use, eclampsia, heavy caffeine consumption, trauma, postpartum hypotension, and hypotensive shock.[1,2] The diagnosis was complicated in our patient because the typical lesion was not evident biomicroscopically. Fluorescein angiography was almost normal. However, with the IR imaging mode of high resolution angiography (HRA) 2, the lesion became evident.

While AMNR was first believed to involve the inner retina, later studies using OCT demonstrated that the outer retina is primarily affected.[4,5] High-speed ultrahigh resolution OCT images showed focal depression of the external limiting membrane, IS/OS junction, photoreceptors, and retinal pigment epithelium. The inner retina appeared normal.[4]

In our case, spectral OCT demonstrated disruption of IS/OS junction and thinning of the outer nuclear layer with increased reflectivity at initial examination. During follow-up, hyper-reflectivity disappeared, but the visual acuity remained unchanged and a focal thinning of the outer nuclear layer persisted. In conclusion, the diagnosis of AMNR remains a challenge due to subtlety in retinal changes and rarity of the disease.

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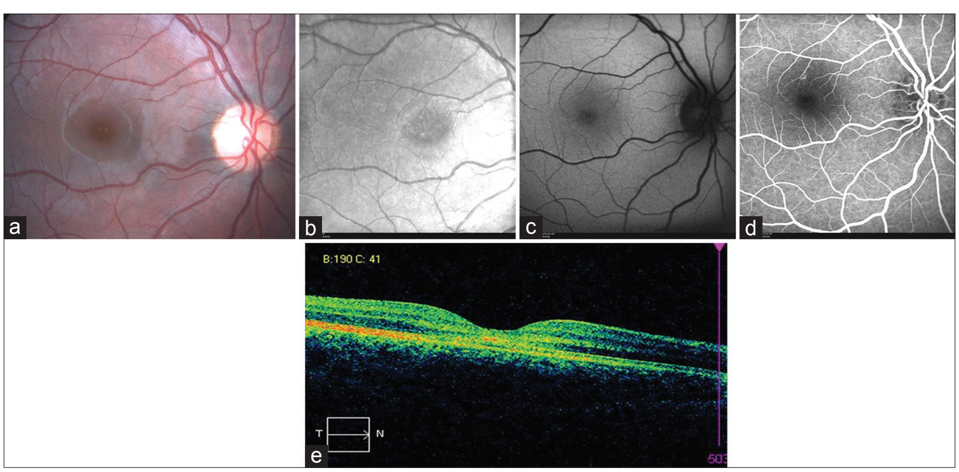

Figure 1: (a) Fundus examination showed normal macular appearance. (b) The infrared imaging demonstrated sharply demarcated dark macular lesion. (c) Fundus autofluorescence imaging was normal. (d) Fundus fluorescein angiography was almost normal except a subtle RPE defect; (e) Spectral OCT showed disruption of IS/OS junction and thinning of outer nuclear layer and increased reflectivity

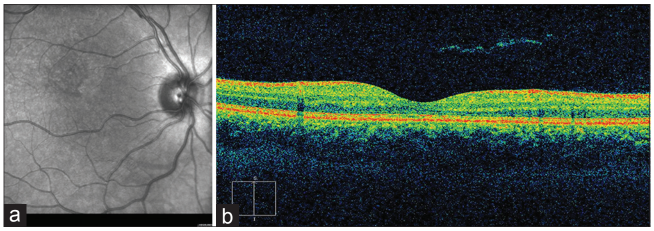

Figure 2: (a) The infrared imaging demonstrated attenuation of the dark lesion 3 months after onset. (b) Spectral OCT showed realignment of IS/OS junction but a focal thinning of outer nuclear layer

References

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