Acute macular neuroretinopathy (AMNR) is a rare retinal condition that features the sudden onset of bilateral central scotomata, which may be preceded by a viral proadrome in some patients. Acute macular neuroretinopathy may be difficult to diagnose because of the subtle or even absent findings on funduscopic examination and fluorescein angiography. Bos and Deutman initially described AMNR in 4 patients with paracentral scotomata, slightly decreased visual acuity, and reddish, wedge-shaped intraretinal lesions directed toward the fovea. Because of the acute onset of the symptoms and based on their theory that the more superficial layers of macular retina were involved, the term AMNR was adopted.

Herein, we describe 2 patients with features of AMNR in whom Amsler grid testing, infrared imaging, spectral-domain optical coherence tomography (SD-OCT), fundus autofluorescence, and multifocal electroretinogram (mERG) were useful in characterizing the precise structural and functional deficits of this condition. The localization of structural deficits to the outer retina and photoreceptor layer using SD-OCT and evidence of depressed cone amplitudes using mERG in both patients were more consistent with photoreceptor and outer retinal dysfunction. Hughes et al previously reported 2 cases of AMNR that featured outer retinal structural changes using SD-OCT. The functional and anatomic deficits observed in our patients using multimodality diagnostically testing, combined with the previous structural characterization of AMNR,²,³ are supportive of the proposed nomenclature acute macular outer retinopathy (AMOR) to more accurately describe this unique clinical entity.²

Report of Cases.  

**Case 1.** A 24-year-old woman had severe fatigue, headaches, and intermittent photopsias, followed by bilateral central scotomata 1 week later. Visual acuities were 20/25 OD and 20/20 OS. Amsler grid testing showed a well-defined boot-shaped scotoma in the right eye and a wedge-shaped scotoma in the left eye (Figure 1). Color vision with Ishihara plate testing was normal in both eyes. No relative afferent pupillary defect was seen. Slitlamp examination showed no anterior chamber or vitreous cells. Dilated funduscopic examination and fluorescein angiography were unremarkable.

Infrared imaging showed a well-defined boot-shaped image in the right eye and a wedge-shaped image in the left eye, which corresponded precisely to the scotomata defined by Amsler grid testing. Fundus autofluorescence imaging showed subtle hypoautofluorescence corresponding to these affected areas. The mfERG showed subnormal amplitudes of the central responses with normal implicit times consistent with a regional abnormality of local retinal responses in the form of central cone dysfunction (Figure 1). Spectral-domain optical coherence tomography showed thinning of the photoreceptor outer segments and irregularities of the outer retinal architecture (Figure 2). Fluorescein and high-speed indocyanine green angiography were unremarkable. The findings were most consistent with AMNR and no treatment was recommended.

At the final follow-up visit 4 months later, the patient’s symptoms were improved and visual acuities were 20/20 OU. Funduscopic examination showed brown discoloration of the parafoveal region and mild retinal pigment epithelial irregularities, which were more prominent than the findings on initial examination. Spectral-domain optical coherence tomography showed persistent abnormalities of the outer retina.

**Case 2.** A 17-year-old healthy girl had fevers and flulike symptoms for 3 days followed by visual photop-
Sias and 2 discrete shadows in the left eye. She was asymptomatic in the right eye.

Visual acuities were 20/20 OU. Amsler grid testing showed 2 areas of visual blur; both areas were temporal to the fovea in the left eye. No relative afferent pupillary defect was observed. The anterior segment examination was unremarkable in both eyes. Dilated funduscopic examination was normal in the right eye. A subtle, slightly abnormal foveal reflex was seen in the left eye, but the funduscopic examination was otherwise unremarkable.

The infrared reflectance image highlighted 2 discrete abnormal images nasal to the fovea, which corresponded precisely to the patient’s scotomata (Figure 3). Spectral-domain optical coherence tomography revealed attenuation of the inner segment–outer segment junction in the abnormal parafoveal area identified by infrared imaging. A fluorescein angiogram showed subtle hypofluorescence in these areas in the late frames of the angiogram. The mfERG showed subnormal amplitudes of the cone responses in the left eye to a greater degree than that observed in the right eye, with normal implicit times. At the 4-month follow-up, the patient’s symptoms remained stable. Her visual acuities were 20/20 OU and the funduscopic lesions in the left eye were more easily seen than on her prior examination. Specifically, there were 2 reddish-brown lesions nasal to the fovea, similar in size and character to the images previously seen by infrared imaging (Figure 4).

Comment. Our patients’ characteristic histories, clinical features, and findings were most consistent with the entity described as AMNR. Recent reports have suggested that infrared imaging is valuable in highlighting the macular lesions of AMNR and SD-OCT has facilitated identification of the anatomical abnormalities present.

In our 2 patients, multimodality diagnostic testing allowed a precise diagnosis of AMNR and provided additional insight into the disease process. Infrared imaging and SD-OCT provided information about the structural defects corresponding to the patients’ scotomata. Fundus autofluorescence showed subtle areas of hypoautofluorescence in both patients. Although these changes were not as prominent as the infrared and SD-OCT abnormalities, the re-

Figure 2. Elevation map of the right eye using spectral-domain optical coherence tomography shows inverted boot-shaped central area (A) with a focal outer nuclear layer and photoreceptor inner segment–outer segment junction attenuation (B) on the horizontal raster scan (inset, B). Elevation map of the left eye (C) shows thinning of the nasal parafoveal region in a wedge-shaped distribution with corresponding outer nuclear layer and photoreceptor inner segment–outer segment junction changes (D) on the horizontal raster scan (inset, D).

Figure 3. Infrared imaging of patient 2’s left eye shows 2 discrete areas of decreased reflectance, which corresponded to her symptoms (A). Spectral-domain optical coherence tomography shows attenuation of the inner segment–outer segment junction and outer retinal architectural abnormalities including focal thinning of the outer nuclear layer (B). The abnormal inferonasal parafoveal region (C) also demonstrated similar outer retinal architectural changes (D). Fundus autofluorescence showed mild hyperautofluorescence surrounding hypoautofluorescent areas corresponding to the lesions nasal to the fovea (white arrows, E). A venous laminar phase angiogram showed patchy choroidal filling (F) and a subtle area of hypofluorescence corresponding to the lesions in the late venous frames of the angiogram (yellow arrows, G).
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observed in the outer retina. Al-
deficit to the structural changes
provided a correlation of a functional
photoreceptor dysfunction pro-
mfERG to precisely identify cone
prominent in the region between the disc and the fovea in the left eye (D).
subnormal amplitudes with normal implicit times in the right eye (C), but these changes were more
eye (A) but showed reddish, oval lesions in the left eye (B). Multifocal electroretinogram demonstrated
sponses of outer retinal origin with
lateral reduction of central re-
discussed in both patients. This was es-
logic changes at the level of the
accumulation with resultant over-
ence from a focal inflammatory
or blockage of choroidal fluores-
sing outer retinal architectural
mation caused by either infect-
and potentially destructive ocular in-
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Figure 4. Fundus photographs of patient 2 at the final 4-month follow-up were unremarkable in the right eye (A) but showed reddish, oval lesions in the left eye (B). Multifocal electroretinogram demonstrated subnormal amplitudes with normal implicit times in the right eye (C), but these changes were more prominent in the region between the disc and the fovea in the left eye (D).

gional decrease in intrinsic tissue au-
tofluorescence suggested patho-
logic changes at the level of the retinal pigment epithelium and pos-
sibly involving the outer retina. The additional use of mfERG high-
lighted the functional deficits of these patients despite better than 20/25 visual acuity. Specifically, bi-
lateral reduction of central re-
sponses of outer retinal origin with normal implicit times was identi-
fied in both patients. This was es-
pecially helpful in identifying ab-
normal photoreceptor function in the asymptomatic eye of patient 2.

Our findings are consistent with prior reports of outer retinal archi-
tectural changes observed with SD-
OCT in AMNR. The addition of mfERG to precisely identify cone photoreceptor dysfunction pro-
voked a correlation of a functional deficit to the structural changes observed in the outer retina. Al-
though we did not identify a cho-
oroidal or retinal vascular perfusion defect by fluorescein angiography or indocyanine green angiography
testing, focal hypofluorescence in the region of the macular lesions was observed on fluorescein angi-
ography of patient 2. This could represent inner choroidal ischemia or blockage of choroidal fluores-
cence from a focal inflammatory accumulation with resultant over-
laying outer retinal architectural disruption.

The term AMNR was originally applied to this condition because of the acute onset of presentation and the theory that the superficial macular retina was involved; given our observations correlating func-
tional and structural aspects of this disease, the term acute macular outer retinopathy may be more appropriate.

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Interferon-γ Release Assay in Tuberculous Scleritis

Scleritis is a painful, often chronic, and potentially destructive ocular in-
fammation caused by either infec-
tious agents or noninfectious im-
mune reactions. Tuberculosis (TB) is one possible infectious cause of scleritis. In this report, we describe 3 patients in whom use of an inter-
feron (IFN)–γ release assay as-
sisted in the diagnosis of tubercu-
lar scleritis.

Report of Cases. Case 1. A 29-year-
old woman was referred for bilat-
eral anterior scleritis refractory to topical corticosteroids. On exami-
nation, corrected visual acuities were 1.2 with normal intraocular pressure in both eyes. The sclera was
markedly hyperemic in all 4 quad-
trants bilaterally (Figure 1A). Mild inflammatory cells were present in the anterior chambers in both eyes but the fundi were unremarkable. Laboratory investigations revealed

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