

A Subtle Case of Type 2 Acute Macular Neuroretinopathy

JAMES O. ROBBINS, MD; DAVID N. BOOY, MD

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INTRODUCTION

Acute macular neuroretinopathy is a poorly understood disruption of the outer retinal layers leading to central visual deficits. Here, we present a case of a young woman with undiagnosed vision loss, found to have acute macular neuroretinopathy after a detailed history and by subtle findings on ophthalmic testing.

CASE

A 28-year-old female was referred to clinic for an “avocado-shaped” blind spot in her right eye that began nine months prior. She had seen both her primary optometrist and ophthalmologist with no etiology identified. She denied preceding illness or significant ocular history. Her daily

medications included fluoxetine and combination oral contraceptive pills (OCPs).

10-2 Humphrey visual field (HVF) demonstrated a right-side paracentral scotoma, superonasal to fixation (**Figure 1**). Color, infrared, and autofluorescence fundus photos were unrevealing for a focal lesion (**Figures 2,3**). Spectral-domain ocular coherence tomography (SD-OCT) of the right macula was significant for focal disruption of the ellipsoid zone (EZ) layer (**Figure 4**).

The patient was diagnosed with acute macular neuroretinopathy (AMN) based on these findings.

Figure 2. SD-OCT of the macula of right eye with a focal disruption of the outer plexiform layer and corresponding outer nuclear layer atrophy.

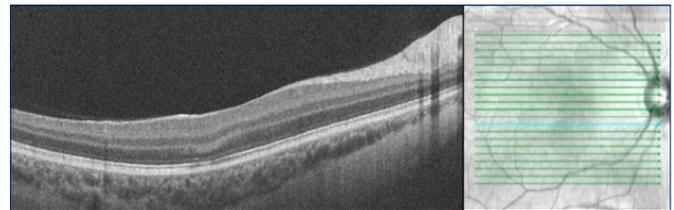


Figure 1. Reliable 10-2 Humphrey visual field of right eye, demonstrating a paracentral scotoma superonasal to fixation.

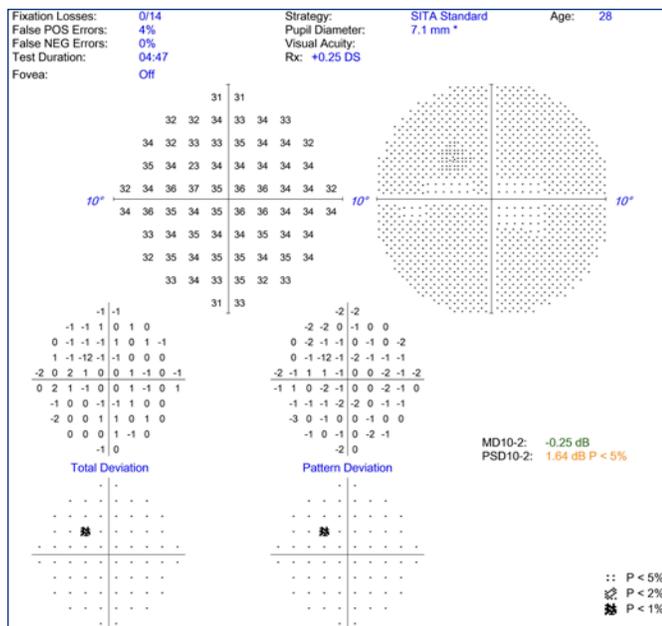


Figure 3. Color fundus photograph of right eye unrevealing for a focal lesion.



Figure 4. Autofluorescence photograph of right eye unrevealing for a focal lesion.



DISCUSSION

AMN classically presents with complaint of a paracentral, petaloid scotoma in otherwise healthy young women.¹ With AMN, OCT typically demonstrates focal ellipsoid zone disruption correlating with the scotoma. This case can be further categorized as Type 2, involving the outer plexiform layer with outer nuclear layer atrophy.² In certain cases, petaloid lesions can be observed on fundus examination within days to months after symptom onset and may be best visualized with near infrared photography.

While the pathogenesis of AMN is poorly understood, several strong associations suggest that microvascular ischemia plays a role. Causes for ischemia may include inflammation, secondary to autoimmune processes or recent infection, hypotension, or catecholamine-induced vasoconstriction from sympathomimetics like epinephrine and caffeine.³ Of note, a strong risk factor for AMN is OCP use; however, there is no evidence to suggest that discontinuation of OCPs is beneficial to prognosis. Therefore, this patient was not recommended to stop her medication or switch to another form of contraception. There are currently no effective interventions for AMN and symptoms can be expected to remain stable or improve gradually over time.

References

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Authors

James O. Robbins, MD, Department of Ophthalmology, Duke University, Durham, NC.

David N. Booy, MD, Division of Ophthalmology, Alpert Medical School of Brown University, Providence, RI.

Disclosures

None

Correspondence

James Robbins, MD
 Department of Ophthalmology, Duke University
 2351 Erwin Rd, Durham, NC 27705
 802-249-2538
james.o.robbs@duke.edu