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Serial Changes in Multimodal Imaging and Electrophoretographic Findings in Acute Unilateral Outer Retinopathy Presenting With Blind Spot Enlargement

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Abstract

Acute blind spot enlargement can arise from either optic nerve or outer retinal disease. We report an acute outer retinopathy of the left eye in a 60-year-old man with bilateral high myopia. He was referred after three weeks of photopsia and blurred vision in the left eye. Best-corrected visual acuity was -0.08 logMAR in the right eye and 0.15 logMAR in the left eye (0.05 logMAR before onset), and axial lengths were 29.59 mm and 30.30 mm, respectively, with a relative afferent pupillary defect in the left eye. There was no anterior chamber or vitreous inflammation, and no white dots or foveal granularity. Widefield fundus autofluorescence showed sharply demarcated peripapillary hyperautofluorescence and patchy midperipheral hyperautofluorescence in the left eye alone, whereas fluorescein and indocyanine green angiography showed no significant findings. Optical coherence tomography showed attenuation of the ellipsoid zone extending from nasal to the fovea toward the disc, and Humphrey 30-2 perimetry showed blind spot enlargement. Multifocal electrophoretography showed reduced amplitudes in the left eye corresponding to the outer retinal and field abnormalities, while mean full-field amplitudes were within reference limits in both eyes. By about 20 weeks, the symptoms had resolved, left eye acuity had returned to 0.05 logMAR, and the outer retinal change, the multifocal amplitudes, and the field defect had all improved. The phenotype most closely resembled acute idiopathic blind spot enlargement, but findings overlapping with multiple evanescent white dot syndrome precluded a single diagnosis. Where the two overlap, serial multimodal imaging and electrophoretography were useful in characterizing the process.

Categories: Ophthalmology

Keywords: acute idiopathic blind spot enlargement, enlarged blind spot, fundus autofluorescence, high myopia, multifocal electrophoretography, multiple evanescent white dot syndrome, optical coherence tomography, outer retinopathy

Introduction

Blind spot enlargement arises not only from optic nerve disease but also from disorders affecting the peripapillary outer retina. Acute unilateral blind spot enlargement without disc edema was described in 1988 as acute idiopathic blind spot enlargement (AIBSE) [1] and was subsequently regarded as a spectrum that includes multiple evanescent white dot syndrome (MEWDS) [2]. The two are separated mainly by the appearance of the fundus and by the fate of the visual field. AIBSE involves the peripapillary retina, usually without ophthalmoscopically visible white dots; photopsia is frequent, a relative afferent pupillary defect is common, and the field defect characteristically persists after the photopsia has subsided [3,4]. MEWDS is defined by multifocal gray-white lesions with foveal granularity, together with wreath-like hyperfluorescence on fluorescein angiography or hyperreflective lesions extending from the retinal pigment epithelium into the outer nuclear layer on optical coherence tomography (OCT), and by absent to mild intraocular inflammation; it resolves spontaneously and completely [5]. Photopsia, blind spot enlargement, and outer retinal dysfunction are common to both. High myopia and previous retinal detachment surgery have been reported as settings in which a secondary, MEWDS-like reaction occurs [6], and the eye described here has both. Multifocal electrophoretographic abnormalities have been reported to persist, often bilaterally, after clinical recovery [4], and OCT has established that the lesion in AIBSE lies in the outer retina [7,8], yet misdiagnosis as optic neuritis remains common [9]. We present a case of acute unilateral outer retinopathy in which features of AIBSE and MEWDS overlapped, with serial multimodal imaging and electrophoretography, and set out the similarities to and differences from both entities.

Case Presentation

History and findings at presentation

A 60-year-old man had undergone surgery seven years earlier for a single localized rhegmatogenous retinal detachment in the superotemporal quadrant of the left eye, treated with cryopexy and a single radial scleral buckle; the retina had remained attached since. Three weeks before presentation to our hospital, he noticed photopsia and blurred vision in the left eye and consulted a local ophthalmologist, who identified outer

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retinal disruption in the left macula and referred him. Best-corrected visual acuity was -0.08 logMAR in the right eye and 0.15 logMAR in the left (0.05 logMAR in the left eye before onset), and axial lengths were 29.59 mm and 30.30 mm, respectively. Both eyes were phakic without cataracts. There was no pain on eye movement, but a relative afferent pupillary defect (RAPD) was present in the left eye. No anterior chamber or vitreous inflammation was seen. Both eyes showed a peripapillary staphyloma and a peripapillary conus (Figure [1](#)). No white dots or foveal granularity were present.

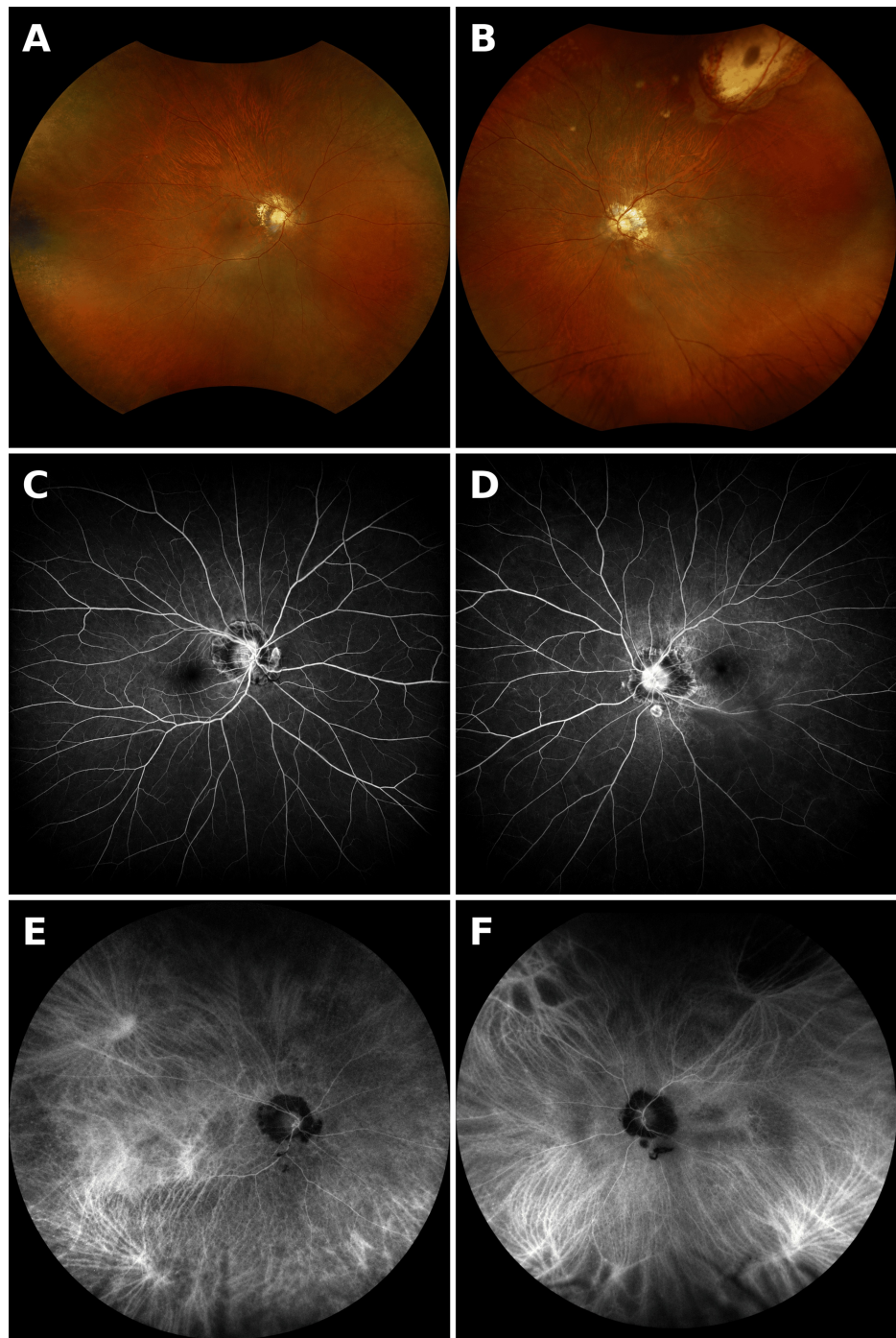


FIGURE 1: Baseline ultra-widefield fundus photography and angiography

Ultra-widefield images at presentation. (A) Right eye and (B) left eye, fundus photographs; (C) right eye and (D) left eye, fluorescein angiography; (E) right eye and (F) left eye, indocyanine green angiography. All angiographic images are late-phase. Both eyes show a peripapillary staphyloma and a peripapillary conus, and no white dots are seen in the left eye; the pale chorioretinal lesion superotemporally in the left eye corresponds to the site of the previous cryopexy and scleral buckle. Neither eye shows definite leakage, hyperfluorescence, or hypofluorescence, and the left eye shows no wreath-like hyperfluorescence, no peripapillary zonal hypofluorescence, and no angiographic correlate of the hyperautofluorescent area. All images were acquired with an ultra-widefield scanning laser ophthalmoscope (Optos California).

Imaging, systemic workup, and electrophysiology

Widefield fundus autofluorescence (Optos California, Optos plc, Dunfermline, UK; 532 nm green excitation) showed a sharply demarcated area of hyperautofluorescence around the left optic disc together with patchy midperipheral hyperautofluorescence (Figure 2).

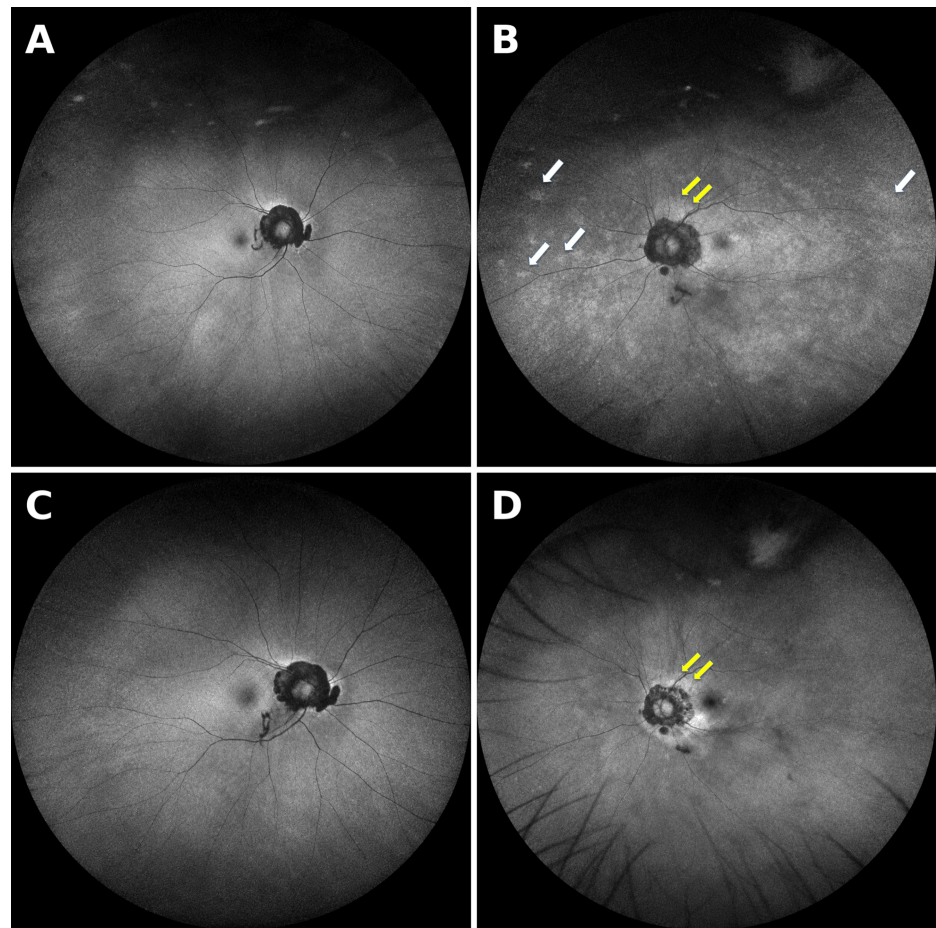


FIGURE 2: Serial fundus autofluorescence findings in both eyes

Ultra-widefield fundus autofluorescence (Optos California; 532 nm green excitation). (A) Right eye at presentation; (B) left eye at presentation; (C) right eye at about 20 weeks; (D) left eye at about 20 weeks. In the left eye alone, a sharply demarcated area of hyperautofluorescence surrounds the optic disc (yellow arrows), and patchy midperipheral hyperautofluorescence is present (white arrows) at presentation. At about 20 weeks, the patchy midperipheral hyperautofluorescence has resolved, and the peripapillary hyperautofluorescence has contracted to a residual rim adjacent to the conus (yellow arrows). The right eye is normal at both time points.

Fluorescein angiography and indocyanine green angiography showed no significant findings in either eye; in particular, there was no wreath-like hyperfluorescence and no peripapillary zonal hypofluorescence (Figure 1). OCT (Spectralis, Heidelberg Engineering, Heidelberg, Germany) showed attenuation of the ellipsoid zone in the left eye extending from the nasal side of the fovea toward the optic disc, with no hyperreflective lesion extending from the retinal pigment epithelium into the outer nuclear layer (Figure 3).

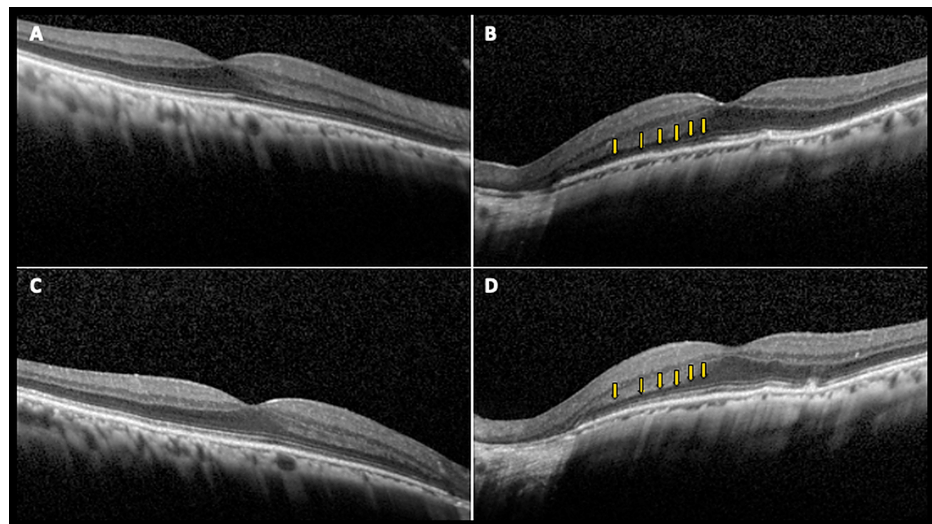


FIGURE 3: Serial optical coherence tomography findings in both eyes

Optical coherence tomography (Spectralis, Heidelberg Engineering). (A) Right eye at presentation; (B) left eye at presentation; (C) right eye at about 20 weeks; (D) left eye at about 20 weeks. In the left eye at presentation the ellipsoid zone is attenuated from nasal to the fovea toward the optic disc (segment marked by yellow vertical bars). At about 20 weeks the ellipsoid zone is better delineated over the same segment (yellow vertical bars). In the right eye the ellipsoid zone is continuous and unchanged at both time points. All scans are horizontal sections through the fovea, acquired without eye-tracked follow-up registration to the baseline scan, so that the locations sampled at the two visits correspond closely but are not identical. The scale is the calibrated display of the instrument.

Humphrey 30-2 perimetry showed blind spot enlargement corresponding to the area of outer retinal disruption, with a mean deviation of -8.53 dB in the left eye (Figure 4).

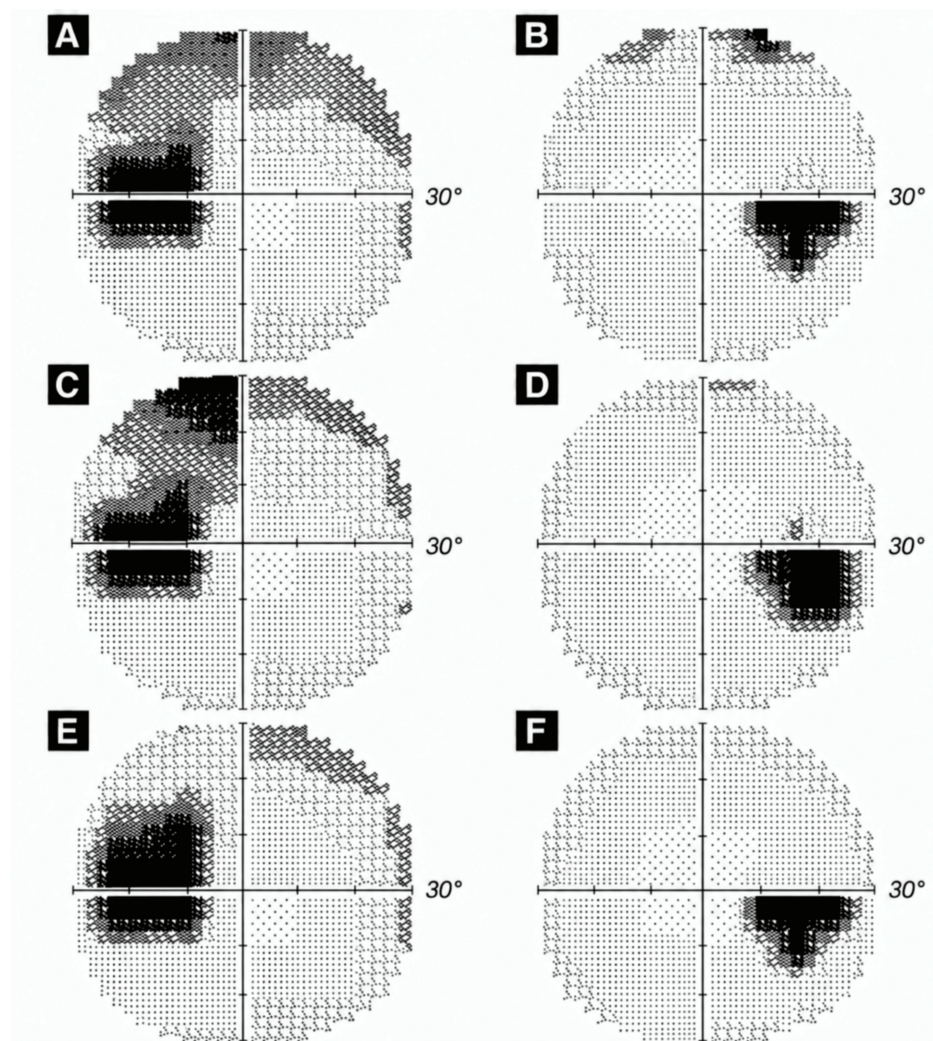


FIGURE 4: Serial Humphrey perimetry findings in both eyes

(A, C, E) Left eye and (B, D, F) right eye, Humphrey 30-2 (SITA Fast), at presentation and at about 7 and 19 weeks. All panels are grayscale displays. The left eye shows blind spot enlargement at presentation corresponding to the area of outer retinal disruption, with contraction of the defect by about 19 weeks; the mean deviation improved from -8.53 dB to -6.75 dB while the pattern standard deviation increased. The right eye shows a stable area of reduced sensitivity around the blind spot, consistent with the peripapillary conus of high myopia, with a mean deviation of -3.0 dB at presentation and -2.9 dB at about 19 weeks.

Blood tests for syphilis, tuberculosis, and sarcoidosis and an anti-recoverin antibody assay were all negative, and noncontrast computed tomography (CT) from head to pelvis showed no evidence of malignancy.

Full-field electroretinography (RETeval, LKC Technologies, Gaithersburg, Maryland) was recorded with the five-step protocol of the International Society for Clinical Electrophysiology of Vision (ISCEV) [10], using the skin-electrode sensor strip supplied with the device; the mean amplitudes were within the reference limits of the manufacturer's normative database in both eyes. Multifocal electroretinography (VERIS Science 6.0.10, Electro-Diagnostic Imaging, Redwood City, California) showed reduced amplitudes in the left eye corresponding to the areas of outer retinal disruption and field loss (Figure 5).

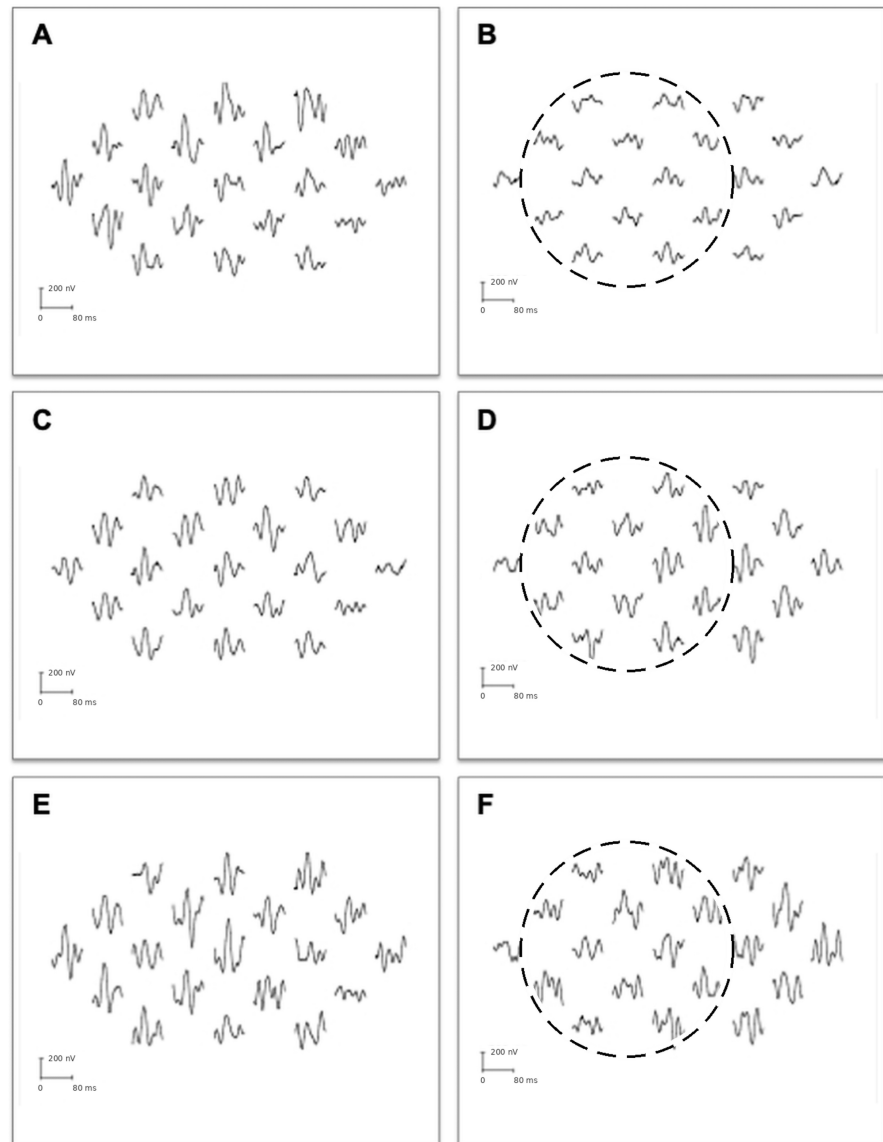
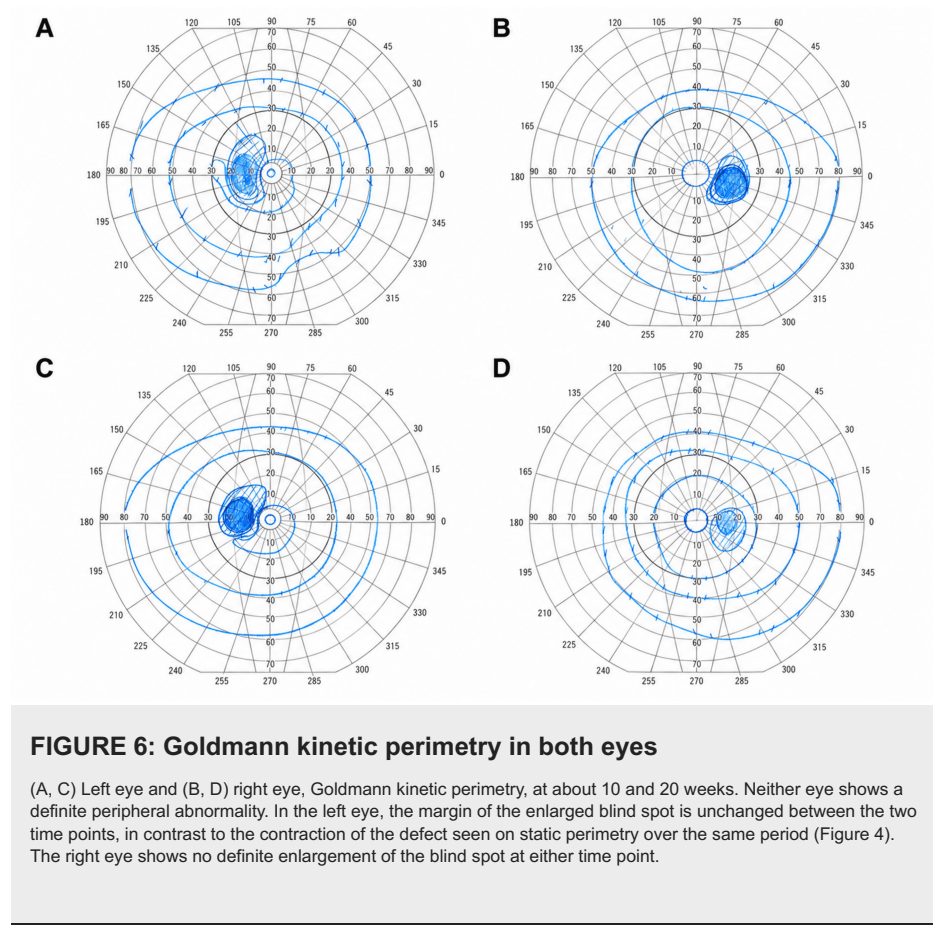


FIGURE 5: Serial multifocal electroretinogram trace arrays in both eyes

(A, C, E) Right eye and (B, D, F) left eye at about 2, 10 and 20 weeks. All panels show raw trace arrays in visual field coordinates, with identical time base, amplitude scale, orientation and magnification. In the left eye the amplitudes are reduced at about 2 weeks in the region corresponding to the outer retinal disruption and the field defect, improve by about 10 weeks and are maintained at about 20 weeks; the right eye shows no definite change at any time point. The circle encloses the analysis region corresponding to the site of structural abnormality on optical coherence tomography and was selected post hoc; no statistical test was applied. Recording conditions: VERIS Science 6.0.10, 19 scaled hexagons over a stimulus field 30 degrees in diameter centered on fixation, recording time about 4 minutes, skin electrodes.

Clinical course

The patient was observed without treatment. On fundus autofluorescence, the patchy midperipheral hyperautofluorescence had resolved by about 11 weeks, and the peripapillary hyperautofluorescence had contracted to a residual rim adjacent to the conus; the appearance was similar at about 20 weeks (Figure 2). Left best-corrected visual acuity improved to 0.05 logMAR at about 10 weeks, and the RAPD resolved. Multifocal amplitudes improved at about 10 weeks and were maintained at about 20 weeks. Humphrey 30-2 perimetry of the left eye also showed contraction of the field defect at about 19 weeks, with the mean deviation improving from -8.53 dB at presentation to -8.19 dB at about seven weeks and -6.75 dB at about 19 weeks. Goldmann kinetic perimetry, performed in both eyes at about 10 and 20 weeks, showed no definite peripheral abnormality and no clear reduction in the left blind spot enlargement between the two time points (Figure 6).



By about 20 weeks, the symptoms had resolved, and the outer retinal disruption had clearly improved compared with presentation (Table 1).

Time point (approximate weeks from presentation)	Main events and examinations
About 3 weeks before presentation	Onset (photopsia and blurred vision in the left eye)
About 1 week before presentation	Seen by referring ophthalmologist; outer retinal disruption identified in the left eye; no white dots
Presentation	BCVA (logMAR) -0.08 right/0.15 left. Quantitative pupillometry. Widefield fundus autofluorescence, fluorescein angiography, and indocyanine green angiography. OCT. Humphrey 30-2 (left MD -8.53 dB). Axial length. Blood tests and non-contrast CT from head to pelvis
About 2 weeks	Full-field electroretinography. Multifocal electroretinography (reduced amplitudes in the left eye)
About 5 weeks	Quantitative pupillometry
About 7 weeks	BCVA (logMAR) 0.15 left. Quantitative pupillometry. Humphrey 30-2 (left MD -8.19 dB)
About 10 weeks	BCVA (logMAR) 0.05 left. Quantitative pupillometry. Multifocal electroretinography (improved). Goldmann perimetry
About 11 weeks	BCVA (logMAR) 0.05 left. Fundus autofluorescence (improved)
About 15 weeks	BCVA (logMAR) 0.10 left
About 19 weeks	Humphrey 30-2 (left MD -6.75 dB)
About 20 weeks	Final assessment. BCVA (logMAR) -0.08 right / 0.05 left. RAPD resolved. Fundus autofluorescence. OCT (improved). Multifocal electroretinography. Goldmann perimetry

TABLE 1: Clinical course and timing of ophthalmic examinations

Time points are given as approximate weeks from the first visit to our hospital. Examinations listed within the same period were not necessarily performed on the same day.

BCVA: best-corrected visual acuity; MD: mean deviation; OCT: optical coherence tomography; CT: computed tomography; RAPD: relative afferent pupillary defect.

Discussion

Localization of the lesion

Reduced multifocal amplitudes and blind spot enlargement corresponded to the area of outer retinal attenuation on OCT. The midperipheral autofluorescence abnormality resolved during follow-up, so the structural change was not confined to the peripapillary retina. Goldmann perimetry showed no definite peripheral abnormality, and mean full-field amplitudes remained within reference limits, placing the functional center of the process in the peripapillary region. An RAPD also occurs in outer retinal disease [3], so its presence does not indicate optic nerve involvement. An optic nerve lesion was considered unlikely given the absence of pain on eye movement and of disc swelling, together with the combination of preserved mean full-field amplitudes and a localized reduction of multifocal amplitudes matching the area of ellipsoid zone attenuation on OCT.

The two perimetric methods gave apparently discordant results. Humphrey 30-2 perimetry measures differential light sensitivity at fixed points on a 6-degree grid, so a partial recovery of sensitivity within the scotoma registers as contraction of the defect and as an improvement in mean deviation, which rose from -8.53 dB at presentation to -6.75 dB at about 19 weeks. Goldmann kinetic perimetry maps the border at which a moving target of fixed intensity becomes visible, so a scotoma that becomes shallower without retreating at its margin appears unchanged. The fact that the pattern standard deviation increased while the mean deviation improved fits the same reading: the depth of the localized loss decreased while its distribution remained irregular. The two findings are therefore complementary rather than contradictory.

Similarities to and differences from AIBSE

Acute unilateral blind spot enlargement without disc edema, photopsia, an RAPD, and reduced local retinal function with preserved mean full-field amplitudes together reproduce the AIBSE phenotype [1,3]. Sharply demarcated peripapillary hyperautofluorescence with matching attenuation of the ellipsoid zone and recovery of the outer retinal structure over follow-up are also shared with descriptions from the OCT era

[7,8].

Several differences from the published series exist. All 27 patients in one series were women aged 19 to 53 years [3], and the prospectively followed patients in the OCT era were in their twenties [8], whereas the present patient is a 60-year-old man. Visual acuity is usually preserved, but here it fell from a premonitory 0.05 logMAR to 0.15 logMAR. Published series report either that the field defect does not improve even as photopsia subsides [3] or that it contracts but never returns to an unequivocally normal-sized blind spot [4]; here the defect contracted on Humphrey perimetry while blind spot enlargement persisted on Goldmann perimetry. AIBSE is defined by peripapillary localization, yet the autofluorescence abnormality extended into the midperiphery. These are differences from the reported series rather than grounds for exclusion.

Similarities to and differences from MEWDS

Acute unilateral onset, photopsia, blind spot enlargement, multifocal hyperautofluorescence, and spontaneous resolution are equally compatible with MEWDS. The distribution is not atypical either: lesions were centered on or around the disc in 17 of 29 episodes (59%) in one series [11]. Cases without ophthalmoscopically visible white dots have been reported [12], so their absence alone does not exclude MEWDS. A secondary, MEWDS-like reaction has also been described in eyes with pre-existing disruption of the choriocapillaris-Bruch membrane-retinal pigment epithelium complex, with high myopia and previous retinal detachment surgery being among the associated conditions [6]; this eye has both.

Two of the three key classification criteria were, however, unmet: there were no multifocal gray-white lesions with foveal granularity, and neither wreath-like hyperfluorescence on fluorescein angiography nor hyperreflective lesions extending from the retinal pigment epithelium into the outer nuclear layer were present. Only the third criterion, absent to mild anterior chamber and vitreous inflammation, was met [5]. Indocyanine green angiography in MEWDS shows multifocal hypofluorescent spots that are often confluent and form broad hypofluorescent zones [13], whereas no definite hypofluorescence was seen in the late phase here. This dissociation between a clear autofluorescence abnormality and the lack of any angiographic correlate is the most distinctive feature of the case.

An overlapping phenotype

AIBSE was described from the outset not as a single disease but as a spectrum containing MEWDS as a subset [2]. The present case is best read as an AIBSE phenotype on which a MEWDS-like multifocal autofluorescence pattern is superimposed. Acute zonal occult outer retinopathy is unlikely: neither the demarcating line nor the trizonal pattern of the multimodal imaging definition was present [14], and the course differs from the slowly progressive phenotype involving both the peripapillary and the far peripheral retina [15]. We therefore describe the case as an overlap rather than assign a single diagnosis. The secondary MEWDS framework [6] could account for the predisposing background, but the lack of angiographic findings makes this speculative.

Other differential diagnoses

Paraneoplastic retinopathy is clinically unlikely given the unilateral disease, the preserved mean full-field amplitudes, the improvement without treatment, and the absence of malignancy on imaging. Only the anti-recoverin antibody was measured, however, and other antiretinal antibodies were not; since antiretinal antibodies other than recoverin are frequently positive in subjects without autoimmune retinopathy and require cautious interpretation [16], the diagnosis cannot be excluded outright.

Limitations

This single observational case supports no conclusion about diagnosis or prognosis, and the alternatives above were judged unlikely rather than excluded. The multifocal recording departed from the ISCEV standard in electrode type, element number, and field size [17]. Skin electrodes give smaller signals and a lower signal-to-noise ratio than corneal electrodes, so the absolute amplitudes cannot be compared with normative data acquired in the standard configuration; 19 scaled hexagons over a 30-degree field correspond to several degrees of visual angle per element, which is coarse relative to the extent of the lesion on OCT; and no normative database exists for this configuration at our institution. Serial change was therefore judged qualitatively, against the fellow eye and against the same eye at earlier time points, rather than against reference values, and the correspondence with structure and visual field is approximate.

Several data that would have strengthened the interpretation are missing. No kinetic field was obtained at presentation, so the two Goldmann examinations compare time points after most of the recovery had already occurred. Visual evoked potentials and orbital magnetic resonance imaging were not obtained, so the localization to the outer retina rests on the correspondence between the imaging, perimetric, and electroretinographic findings rather than on neuro-ophthalmic testing. Quantitative pupillometry was performed at the time points shown in Table 1, but its numerical output is not available to us, and the RAPD and its resolution are therefore reported as clinical observations; the reliability indices of the perimetric examinations are likewise not reported. Age, high myopia, the peripapillary staphyloma, and the previous buckling procedure are confounders: the myopic changes degrade the visibility of the peripapillary outer

retinal bands and raise the background autofluorescence, and all of them may depress field sensitivity and multifocal amplitudes independently of the present process. The fact that the fellow eye showed a comparable and stable reduction of sensitivity around the blind spot indicates that only the component of the affected eye's findings that appeared acutely and then improved can be attributed to this episode. Follow-up of 20 weeks is too short to assess residual dysfunction, recurrence, or involvement of the fellow eye.

Conclusions

In a 60-year-old man with bilateral high myopia and axial lengths of about 30 mm, acute unilateral blind spot enlargement corresponded to a structural change in the outer retina on OCT and to a localized loss of function on multifocal electroretinography, and both improved without treatment. The clinical phenotype most closely resembled AIBSE, but the midperipheral autofluorescence abnormality and the overlap with related outer retinal disease precluded a single diagnosis. In cases such as this, where features of AIBSE and MEWDS overlap, serial multimodal imaging and electroretinography are useful in characterizing the process. These observations come from a single patient followed for 20 weeks; they describe the course in one eye and do not establish a diagnostic or prognostic rule.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

Concept and design: Ryusuke Hashimoto

Acquisition, analysis, or interpretation of data: Ryusuke Hashimoto, Takayuki Baba

Drafting of the manuscript: Ryusuke Hashimoto

Critical review of the manuscript for important intellectual content: Ryusuke Hashimoto, Takayuki Baba

Supervision: Takayuki Baba

Disclosures

Human subjects: Informed consent for treatment and open access publication was obtained or waived by all participants in this study. Chiba University Hospital issued approval NA. **Conflicts of interest:** In compliance with the ICMJE uniform disclosure form, all authors declare the following: **Payment/services info:** All authors have declared that no financial support was received from any organization for the submitted work. **Financial relationships:** All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. **Other relationships:** All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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