Ultra-Wide-Field Fundus Autofluorescence in Multiple Evanescent White Dot Syndrome

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• PURPOSE: To observe the progression of affected lesions using ultra-wide-field fundus autofluorescence (FAF) in multiple evanescent white dot syndrome.
• DESIGN: Retrospective, observational case series.
• METHODS: SETTING: Institutional. PATIENT POPULATION: 14 eyes of 13 patients (mean age, 35.8 years) with acute disease unilaterally. OBSERVATION PROCEDURES: Patients underwent ultra-wide-field FAF, spectral-domain optical coherence tomography (SD OCT), multifocal electroretinography (mfERG), and Goldmann or automated perimetry; the best-corrected visual acuity (BCVA) and refractive error were measured. MAIN OUTCOME MEASURE: Ability of ultra-wide-field FAF to detect lesions with greater sensitivity compared with color fundus photography.
• RESULTS: Ultra-wide-field FAF imaging enabled improved visualization of the affected lesions and showed that the core lesion was in the posterior fundus involving the peripapillary retina and posterior pole and surrounded by hyper-autofluorescent spots outside the vascular arcade. The posterior lesions expanded rapidly and peripheral spots spread farther peripherally and reached a maximal extent during the acute stage. During follow-up, the peripheral hyper-autofluorescent spots resolved and then hyper-autofluorescence of the posterior fundus gradually faded. SD OCT showed diffuse disruption of the photoreceptor inner segment/outer segment junction (IS/OS) in the posterior fundus during the acute stage. The correlation between the IS/OS abnormality and hyper-autofluorescent areas was unclear. The disrupted IS/OS was restored with normalization of the FAF.
• CONCLUSIONS: Ultra-wide-field FAF showed that the lesions arise from the peripapillary retina and the posterior pole and spread peripherally in a centrifugal manner during the acute stage. The hyper-autofluorescent spots faded from the periphery in a centripetal manner. (Am J Ophthalmol 2015;159:698–706. © 2015 by Elsevier Inc. All rights reserved.)

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MULTIPLE EVANESCENT WHITE DOT SYNDROME (MEWDS), first reported by Jampol and associates in 1984,[1] is an acute, generally unilateral retinopathy that develops predominantly in young adults. Patients report blurred vision, photopsia, or visual field defects and have multiple yellow or white spots in the fundus. Myopia was highly prevalent in Japanese patients with MEWDS.[2] Although the clinical features of MEWDS have been well described, the cause is unknown. Fluorescein angiography showed multiple hyperfluorescent spots, but late-phase indocyanine green angiography (IA) showed more pronounced hypofluorescent spots. Full-field electroretinography (ffERG) and multifocal electroretinography (mfERG) showed dysfunctional photoreceptors. Spectral-domain optical coherence tomography (SD OCT) showed a disrupted or irregular photoreceptor inner segment/outer segment (IS/OS).[3,4]

Fundus autofluorecence (FAF) imaging is a noninvasive method of retinal imaging. The FAF signal initially was thought to originate predominantly from lipofuscin in the retinal pigment epithelium (RPE).[5] Material containing N-retinylidene-N-retinylethanolamine (A2E)[6] is another source of FAF.

It is difficult to determine the affected lesions in the MEWDS because of faint and transient white dots in the peripheral fundus and diffuse faint retinal opacities in the posterior fundus. In the current study, we used an ultra-wide-field FAF camera to investigate the evolution of the affected lesions in acute MEWDS.

METHODS

THE INSTITUTIONAL REVIEW BOARD/ETHICS COMMITTEE OF Gunma University School of Medicine (Gunma, Japan) approved the retrospective review of patient data for multiple evanescent white dot syndrome. This study complied with the tenets of the Declaration of Helsinki. All patients provided informed consent for participation in this research.

The study included 14 eyes of 13 patients (4 men, 9 women; mean age, 35.8 years; range, 17–50 years) with MEWDS at Gunma University Hospital. One patient developed the MEWDS bilaterally at a different time. All patients reported unilateral blurred vision and spotty visual field defects at the first examination. The fundi in the affected eyes all had faint white spots of varying diameters

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TABLE 1. Patient Characteristics and Changes Over Time in Multiple Evanescent White Dot Syndrome

Case	Sex/Age	Affected Eye/BCVA		Refractive Error	Follow-up Period (Months)	FAF Pattern		Resolution of Peripheral Spots (Weeks)	IS/OS Status of Posterior Pole At First Visit	Restoration of IS/OS (Months)
		First Visit	Last Visit			First Visit	Last Visit			
1	F/39	OS/0.3	OS/1.2	−1.25	16	Posterior pole + peripheral spots	Normalized	2	Diffuse disruption	3
2	M/27	OD/1.2	OD/1.2	−5.25	18	Posterior pole	Normalized	4	Blurred IS/OS	3
3	M/49	OD/1	OD/1	−6.5	16	Posterior pole + peripheral spots	Normalized	3	Diffuse disruption	12
4	F/50	OD/1.2	OD/1.2	−8.5	16	Posterior pole	Hyper-autofluorescence	4	Blurred IS/OS	7
5	F/43	OD/0.9	OD/1.2	−0.5	25	Posterior pole + peripheral spots	Normalized	6	Blurred IS/OS	12
6	F/28	OS/1.2	OS/1.2	−3	16	Posterior pole	Hyper-autofluorescence	20	Blurred IS/OS	4
7	F/28	OD/1.2	OD/1.2	−3	16	Posterior pole	Normalized	4	Blurred IS/OS	4
8	F/43	OD/1.2	OD/1.2	−9.5	14	Posterior pole	Normalized	8	Diffuse disruption	5
9	M/17	OD/0.4	OD/1.2	−7	3	Posterior pole + peripheral spots	Normalized	3	Diffuse disruption	3
10	F/26	OD/1.2	OD/1.2	−7	36	Posterior pole	Normalized	4	Blurred IS/OS	3
11	F/18	OD/1.2	OD/1.2	−1.75	7	Posterior pole	Hyper-autofluorescence	4	Diffuse disruption	6
12	F/41	OS/0.1	OS/0.9	−8.75	6	Posterior pole + peripheral spots	Normalized	4	Diffuse disruption	4
13	M/33	OS/1	OS/1.2	−0.5	4	Posterior pole	Normalized	4	Diffuse disruption	3
14	F/28	OS/0.2	OS/1	−11.25	57	Posterior pole	Normalized	4	Diffuse disruption	18
Mean	35.8	0.76	1.16	−5.27	13.7			5.3		6.2

BCVA = best-corrected visual acuity; FAF = fundus autofluorescence; IS/OS = inner segment/outer segment junction.

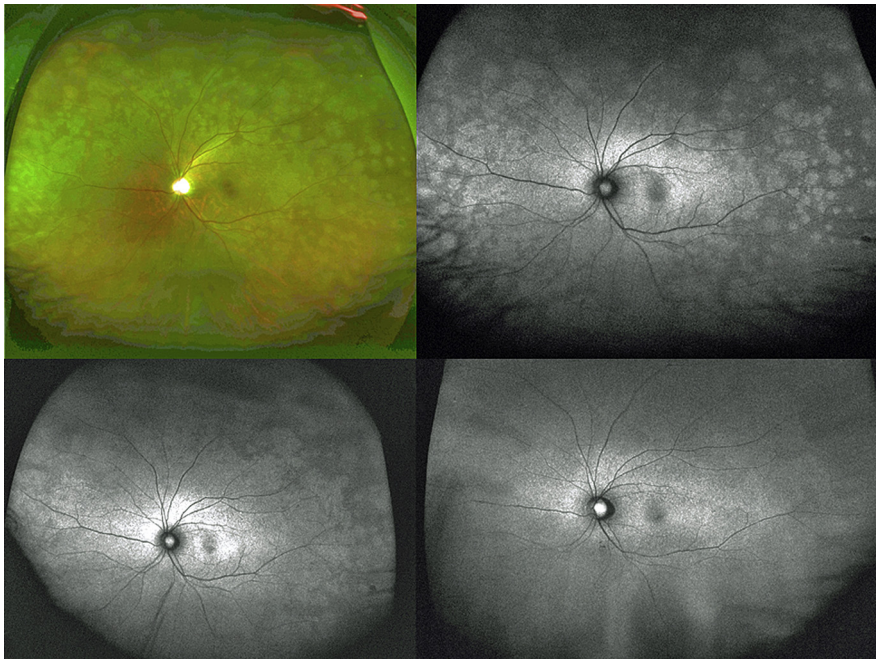

FIGURE 1. Case 1. The patient is a 41-year-old woman with multiple evanescent white dot syndrome in her left eye. (Top left) A color fundus photograph obtained during the first examination shows foveal granularity and scattered yellowish spots in the posterior pole and nasal midperiphery (best-corrected visual acuity [BCVA], 0.1). (Top right) At the first examination, the ultra-wide-field fundus autofluorescence image shows diffuse hyper-autofluorescence in the peripapillary retina and posterior pole and numerous hyper-autofluorescent spots in the periphery. (Bottom left) After 1 week, diffuse hyper-autofluorescence in the posterior fundus expanded and the peripheral hyper-autofluorescent spots fused, resulting in a geographic pattern in the peripheral fundus (BCVA, 0.15). (Bottom right) After 4 months, the fundus autofluorescence is normal (BCVA, 0.9).

mainly in the midperiphery to equatorial region. The included patients had typical MEWDS with numerous transient white spots and diffuse disruption of the photoreceptor outer segment on SD OCT images, which was later restored. All patients were healthy except for the ocular symptoms. No women were pregnant. The mean refractive error of the affected eyes was −5.3 diopters (D) (range, −0.5 to −11.25 D). The patients were followed untreated for a mean of 13.7 months (range, 3–57 months). In addition to routine ocular examinations, we performed color fundus photography with a 50-degree angle camera (Topcon TRC-50DX; Topcon Corporation, Tokyo, Japan), Goldmann perimetry (GP) or Humphrey field analyzer (HFA), ffERG, mfERG, and SD OCT (Cirrus HD-OCT; Carl Zeiss Meditec, Inc, Dublin, California, USA). We evaluated the FAF using a wide-field scanning laser ophthalmoscope (OPTOS 200Tx; Optos PLC, Dunfermline, UK), which allows nonmydriatic FAF imaging of up to 200 degrees of the fundus. OPTOS fundus photograph with pseudo-color images were obtained at each examination. During the acute 3-week stage, we performed OCT and wide-field FAF weekly. At the 3- to 8-week intermediate stage, we performed those test every 4 weeks. At the late stage after 9 weeks, we performed the tests once or twice in 2-month intervals. Two independent retinal specialists (H.H., S.K.) evaluated all photographs. The agreement in the grading of the FAF, color fundus, and OCT images exceeded 90%.

RESULTS

ULTRA-WIDE-FIELD FAF DETECTED THE AFFECTED LESIONS with greater sensitivity than conventional color fundus photography. The affected lesions appeared as diffuse hyper-autofluorescence in the posterior pole and involved the peripapillary retina and macular area and surrounding hyper-autofluorescent spots outside the vascular arcades in all 14 eyes of 13 patients. During the first 2 weeks of observation, diffuse hyper-autofluorescence in the posterior pole expanded to the midperiphery and the peripheral hyper-autofluorescent spots spread farther to their maximal extent. The peripheral hyper-autofluorescent spots faded first and the posterior diffuse autofluorescence then faded. SD OCT showed diffuse disruption or blurring of the IS/OS line in the posterior fundus. The disrupted IS/OS then aggregated in nodules on the RPE and the IS/OS was restored. As the abnormal hyper-autofluorescence in the periphery and the posterior fundus faded to the level

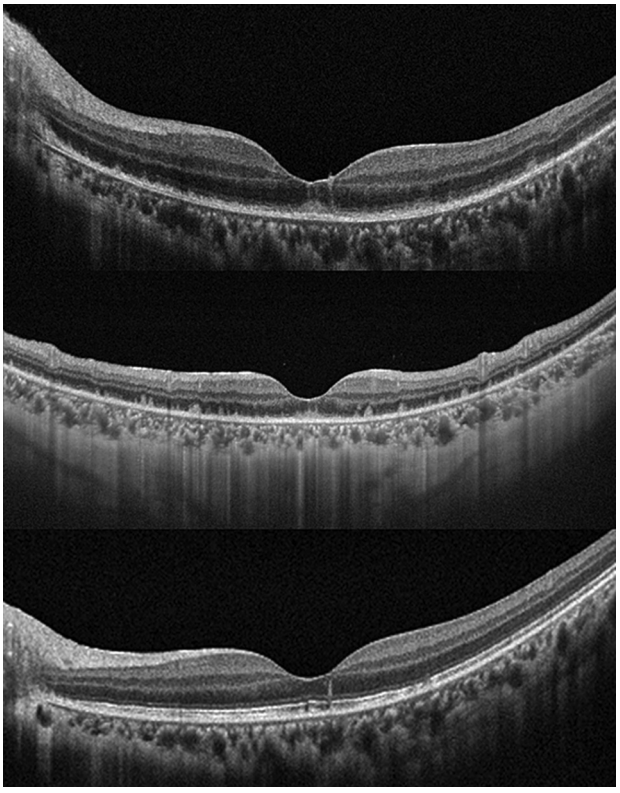

FIGURE 2. Case 1. Spectral-domain optical coherence tomography of the left fundus in a patient with multiple evanescent white dot syndrome. (Top) Diffuse disruption of the photoreceptor layer, including the inner segment/outer segment junction (IS/OS), cone outer segment tips (COST), and external limiting membrane (ELM), are seen in the posterior fundus at the first examination. (Middle) After 1 week, multiple drusenoid nodules are seen at the level of the disrupted IS/OS. (Bottom) At 4 months, the IS/OS and COST are generally restored, with an occasional disruption.

of the unaffected fellow eyes, the continuity of the disrupted IS/OS was restored. Table 1 shows the patient characteristics.

• **ACUTE STAGE (WITHIN 3 WEEKS AFTER MEWDS ONSET):** We observed the acute stage in all 14 eyes of 13 patients. At the first evaluation, the best-corrected visual acuity (BCVA) levels ranged from 0.2 to 1.2 (mean, 0.76) in the affected eyes and 1.0 to 1.2 (mean, 1.16) in the fellow eyes. In all eyes, the posterior fundus had faint white discoloration with multiple white spots in the midperiphery; in 5 eyes the spots spread to the far periphery. Ultra-wide-field FAF showed diffuse hyper-autofluorescence in the posterior pole that involved the peripapillary and macular areas with numerous hyper-autofluorescent spots in the midperiphery or far periphery in all 14 eyes. During the acute stage, the diffuse hyper-autofluorescence in the posterior pole expanded and the peripheral hyper-autofluorescent spots spread farther peripherally. SD OCT showed diffuse disruption of the IS/OS in 8 eyes with various BCVA levels or blurring of the IS/OS in 6 eyes with good BCVA in the posterior fundus. The fellow eyes appeared normal on the SD OCT images. Visual field tests were performed in 14 eyes. All 14 affected eyes had an enlarged blind spot; the macula was involved in 3 eyes. The correlation between the visual field abnormality and the white spots was unclear. GP of the unaffected fellow eyes showed that the blind spot enlarged in 2 patients and showed peripheral depression in 2 patients.

• **INTERMEDIATE STAGE (3–8 WEEKS):** Ultra-wide-field FAF showed gradual fading of the peripheral hyper-autofluorescent spots within 4 weeks in 11 affected eyes, but the spots remained for 6–20 weeks in 3 eyes. In contrast, the diffuse hyper-autofluorescence persisted in the posterior pole in all 14 eyes. SD OCT showed multiple nodules at the level of the disrupted IS/OS in 8 eyes and the IS/OS was blurred in 6 eyes. Restoration of the IS/OS started at around 2 weeks, but the disruption of the IS/OS frequently persisted.

• **LATE STAGE (AFTER 9 WEEKS):** After resolution of the peripheral hyper-autofluorescent spots, the diffuse hyper-autofluorescence in the posterior pole gradually decreased while slight hyper-autofluorescence remained in the posterior fundus over 12 weeks in 11 eyes. The IS/OS became continuous in 11 eyes by 12 weeks but was not restored completely in 3 eyes for more than 1 year.

• **CASE 1:** A 41-year-old woman reported a central scotoma in the left eye 9 days before the first examination. The scotoma expanded gradually and caused generally blurred vision. At the first examination, the vision was 0.1×-9.5 D in the left eye. Biomicroscopy showed cells in the anterior chamber in the left eye. A mild vitreous opacity was seen in the left eye. The right eye appeared normal. The left fundus had a slight whitish discoloration in the posterior fundus and numerous white spots of 1–2 disc diameters spreading in the midperiphery and equatorial regions (Figure 1). Ultra-wide-field FAF showed diffuse hyper-autofluorescence in the peripapillary and macular regions. A hyper-autofluorescent spot was peripheral to the posterior lesion (Figure 1). SD OCT showed diffuse disruption of the photoreceptor layer including the IS/OS, cone outer segment tips (COST), and external limiting membrane (ELM) in the posterior fundus. The RPE appeared irregular (Figure 2). Cells were seen in the posterior vitreous. The photoreceptor layer was intact in the right eye. GP showed an enlarged blind spot that involved the macula in the left eye and peripheral depression in the right eye (Figure 3). ffERG showed a nonrecordable rod response and low amplitudes of the maximal and cone responses in the affected eye and a normal response in the unaffected fellow eye. One week after the first examination, the whitish spot became faint but ultra-wide-field FAF showed diffuse hyper-autofluorescence that involved the posterior

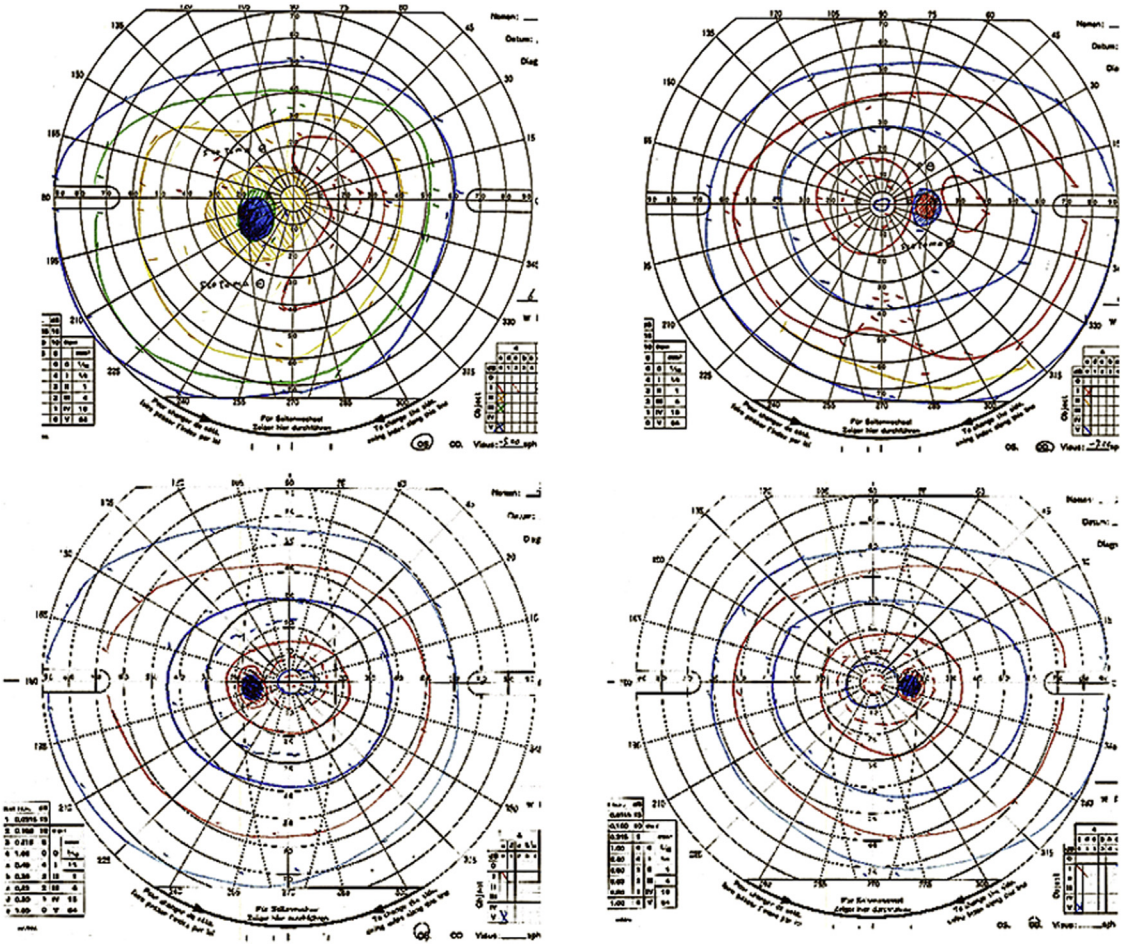

FIGURE 3. Case 1. Goldmann perimetry results from a patient with multiple evanescent white dot syndrome. (Top, left and right) At the first examination, the blind spot is enlarged and a relative scotoma involves the macula in the left eye (Top left) (best-corrected visual acuity, 0.1). There is an inferior peripheral depression in the right eye (Top right). (Bottom, left and right) After 4 months, Goldmann perimetry shows that both eyes are normal.

fundus and midperiphery. Multiple hyper-autofluorescent spots fused together in the peripheral fundus (Figure 1). The BCVA of the left eye was 0.15. SD OCT showed multiple drusenoid nodules at the level of the disrupted IS/OS (Figure 2). At 4 weeks, the left fundus appeared normal except for mottling in the equatorial region. Ultra-wide-field FAF showed irregular hyper-autofluorescence in the equatorial region but the retina was otherwise normal. The BCVA was 0.4 in the left eye. SD OCT showed that the IS/OS was restored except at the fovea. At 16 weeks, the BCVA of the left eye was 0.9. The left fundus appeared normal on ultra-wide color fundus photographs and FAF images (Figure 1, Bottom right). SD OCT showed that the IS/OS and COST were restored in the posterior pole, but the IS/OS was blurred and the COST was unidentifiable peripheral to the posterior pole (Figure 2). ffERG showed slight recovery of the rod response, but the amplitudes of the maximal and cone responses were low. The GP became normal bilaterally (Figure 3).

• **CASE 2:** An 18-year-old woman reported a central scotoma in the right eye 4 days before the first examination. The scotoma expanded gradually and resulted in general blurring of vision. At the first examination, vision was 1.2 × −1.75 D in the right eye. The right fundus was mottled in the posterior fundus (Figure 4). Ultra-wide-field FAF showed diffuse hyper-autofluorescence around the optic disc and the macula and hyper-autofluorescent spots temporal to the macula (Figure 4). SD OCT showed a blurred IS/OS line and ELM and loss of the COST in the posterior fundus (Figure 5). The left eye was normal on ophthalmoscopy and SD OCT. HFA showed decreased sensitivity (mean deviation [MD], −32.33 decibels) in mostly all areas of the central 30-2 in the right eye and a pericentral depression (MD, −6.10 decibels) in the left eye (Figure 6). Two weeks after the first examination, the whitish lesions became faint, but ultra-wide-field FAF showed diffuse hyper-autofluorescence that extended to the midperiphery with a surrounding

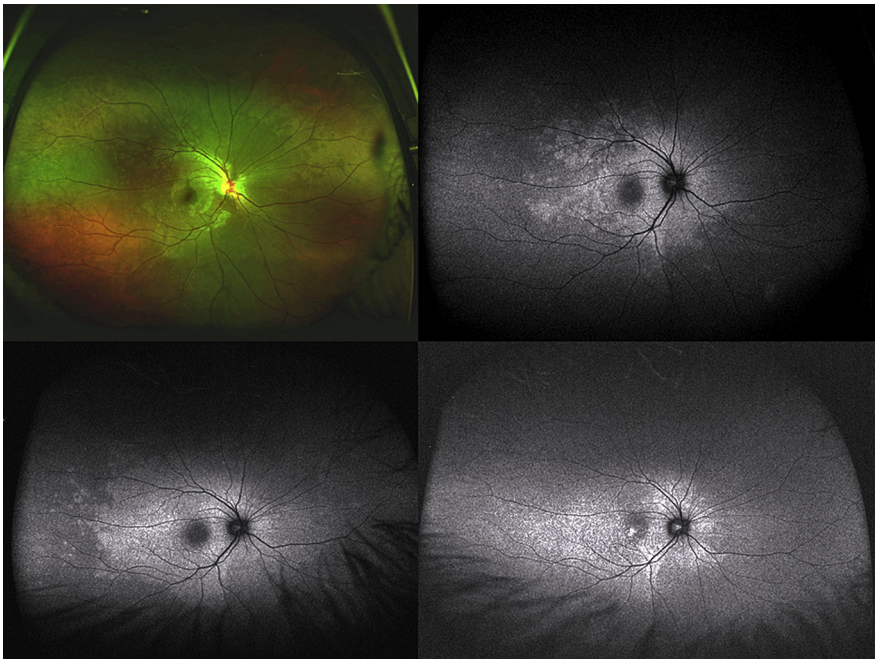

FIGURE 4. Case 2. The patient is an 18-year-old woman with multiple evanescent white dot syndrome in the right eye. (Top left) A color fundus photograph obtained at the first examination shows foveal granularity and yellowish spots in the posterior pole and nasal midperiphery in the right eye (best-corrected visual acuity [BCVA], 1.2). (Top right) At the first examination, a fundus autofluorescence image shows diffuse hyper-autofluorescence in the peripapillary retina and macular area, which is surrounded by numerous hyper-autofluorescent spots. (Bottom left) After 2 weeks, the diffuse hyper-autofluorescence has expanded into the posterior fundus and numerous spots have spread to the periphery (BCVA, 1.2). (Bottom right) After 12 weeks, the area of hyper-autofluorescence has diminished and remains around the optic disc (BCVA, 1.2).

hyper-autofluorescent spot in the periphery (Figure 4). The IS/OS became increasingly blurred in the posterior pole on the SD OCT images. The COST was not visible (Figure 5). Twelve weeks after the first examination, the BCVA was 1.2 in the right eye. The right fundus appeared normal, but diffuse hyper-autofluorescence remained around the optic disc (Figure 4). SD OCT showed that the IS/OS was restored in the posterior pole, but the COST remained blurred (Figure 5). HFA showed an enlarged blind spot in the right eye and a normal left eye (Figure 6). Table 2 shows the changes of visual field in MEWDS patients.

DISCUSSION

USING ULTRA-WIDE-FIELD FAF, WE OBSERVED CENTRIFUGAL spread of the affected lesions in eyes with MEWDS. In the acute disease stage, diffuse hyper-autofluorescence was always present in the peripapillary area and the posterior pole, which was not well observed by ophthalmoscopy. In addition to the diffuse posterior lesions, FAF showed multiple hyper-autofluorescent spots outside the vascular arcades. During the acute stage, the diffuse hyper-autofluorescent lesions in the posterior fundus expanded and multiple peripheral hyper-autofluorescent lesions rapidly spread farther into the periphery. All patients reported an enlarged blind spot during the acute stage that corresponded to the peripapillary hyper-autofluorescent lesions. In the intermediate stage, the peripheral hyper-autofluorescent spots faded first but the posterior hyper-autofluorescent lesions persisted. In the late stage, the posterior lesions returned to normal in 10 of the 14 eyes, but faint or granular hyper-autofluorescence persisted in the posterior pole in 3 eyes. These findings suggested that the affected lesions in MEWDS originated from the peripapillary retina and the macula and spread to the periphery. Because the discoloration of the posterior fundus was faint compared with the white spots, it was easily overlooked. Except for the scattered white spots, the MEWDS appeared similar to acute zonal occult outer retinopathy (AZOOR), although the damage in the photoreceptor outer segment is reversible in MEWDS but irreversible in AZOOR.[7]

On the OCT images, diffuse disruption of the IS/OS was seen in the posterior pole in the acute stage. In the intermediate stage, the aggregated destroyed outer segment formed nodules on the RPE and the restoration of the IS/OS began. In the late stage, the IS/OS was restored completely. In our previous study,[3] the characteristic

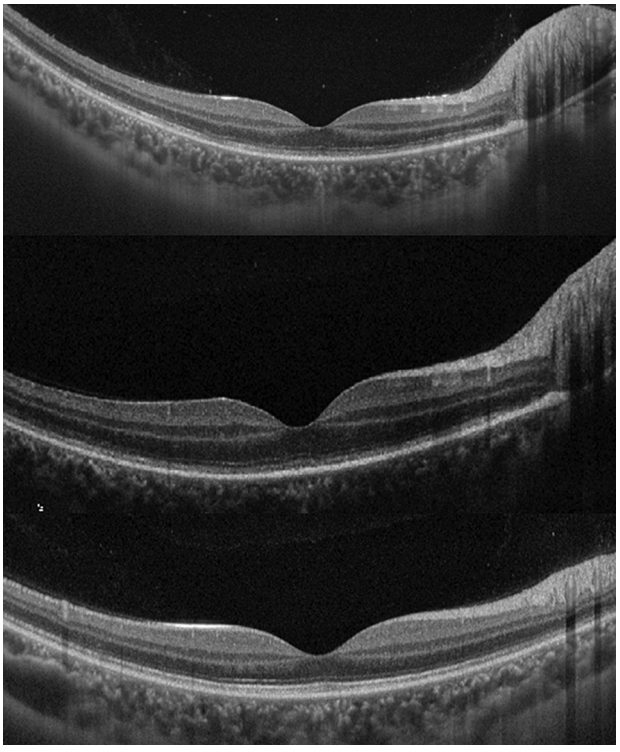

FIGURE 5. Case 2. A spectral-domain optical coherence tomography (SD OCT) image of the right eye of a patient with multiple evanescent white dot syndrome. (Top) At the first examination, SD OCT shows a blurred inner segment/outer segment junction (IS/OS) and external limiting membrane (ELM) and loss of the cone outer segment tips (COST) in the posterior fundus. (Middle) After 2 weeks, the IS/OS is more blurred in the posterior pole. The COST is not visible. (Bottom) After 12 weeks, SD OCT shows that the IS/OS and ELM are restored in the posterior pole, but the COST remains blurred.

MEWDS OCT finding was diffuse disruption of the IS/OS in the acute stage, which was restored from 1.5 weeks to 6 months. In the current study, we observed similar findings on the SD OCT images. In the intermediate stage, the disrupted photoreceptor outer segment aggregated and formed nodules on the RPE that resembled reticular pseudodrusen.[8]

What is the source of the increased FAF in MEWDS? The FAF signal originates predominantly from lipofuscin in the RPE. The material containing A2E, such as the destructive products of the photoreceptors and phagocytosed photoreceptor debris, is another source of FAF. Since FAF increased in the acute stage of MEWDS, the diffusely destroyed photoreceptor outer segment appeared to be the source of the increased FAF, which later normalized along with absorption of debris and restoration of the IS/OS. The ultra-wide-field FAF camera (Optos 200Tx) is a scanning laser ophthalmoscopy camera that facilitates clear focusing of the entire fundus. The Optos 200Tx uses green laser as excitation light for FAF, which spares the effect of the macular pigment. Furthermore, an ultra-wide field of 200 degrees is a great advantage when evaluating pan-fundus lesions.

The increased FAF signal observed in inflammatory diseases may result from RPE cellular alterations, that is, specific activation of pro-oxidative pathways leading to an increase in the size and number of fluorophores in these cells.[5] Accordingly, a previous study[5] attributed the hyper-autofluorescent lesions in MEWDS to accumulated lipofuscin granules, either from hypertrophy and hyperplasia of the RPE with increased production of lipofuscin or from impaired function of the RPE with impaired clearance of lipofuscin.

Joseph and associates suggested that photoreceptor loss causes unmasking of normal underlying RPE autofluorescence.[9] In their cases, SD OCT showed clear ellipsoid zone (IS/OS) attenuation with registration to the hyper-autofluorescent lesions, which was further reinforced by the photoreceptor bleaching effect seen with successive FAF images captured during the same session.

IA showed that hypofluorescent lesions corresponded to the white spots on late-phase color fundus. These angiographic hypofluorescent spots disappeared during the recovery stage of MEWDS. Hangai and associates reported a correlation between the area of IS/OS disruption and hypofluorescence in the late phase of IA.[4] This suggested that MEWDS affects the choriocapillaris or precapillary arterioles and the RPE and photoreceptors. Obana and associates reported that hypofluorescent dots were clustered in the posterior pole and sporadic in the peripheral region and appeared to radiate away from the optic disc or fovea.[10] The angiographic pattern seen on IA images was similar to the hyper-autofluorescent pattern seen on ultra-wide-field FAF images.

We assumed that the destroyed photoreceptor outer segment was the source of the hyper-autofluorescence. The inflammation may involve the RPE and choriocapillaris, which may lead to occlusion of the choriocapillaris. As the photoreceptors are restored, the RPE and focal occlusion of the choriocapillaris may recover.

AZOOR, MEWDS, blind spot enlargement, and punctate choroiditis may share similar morbidity; thus they are referred to as the AZOOR complex,[11] which is suspected to result from a viral infection, possibly with an immune-mediated mechanism in a genetically susceptible person, but its precise pathogenesis is unknown.[12] For example, MEWDS has been reported after *Varicella* infection[13] or vaccination.[14]

The current study showed that posterior lesions involving the peripapillary retina and the posterior pole share similar patterns with AZOOR.

Although the causes of AZOOR and MEWDS have not yet been determined, some inflammatory agents may enter the subretinal space from the subarachnoid space through the margin of the optic disc. The pattern of the

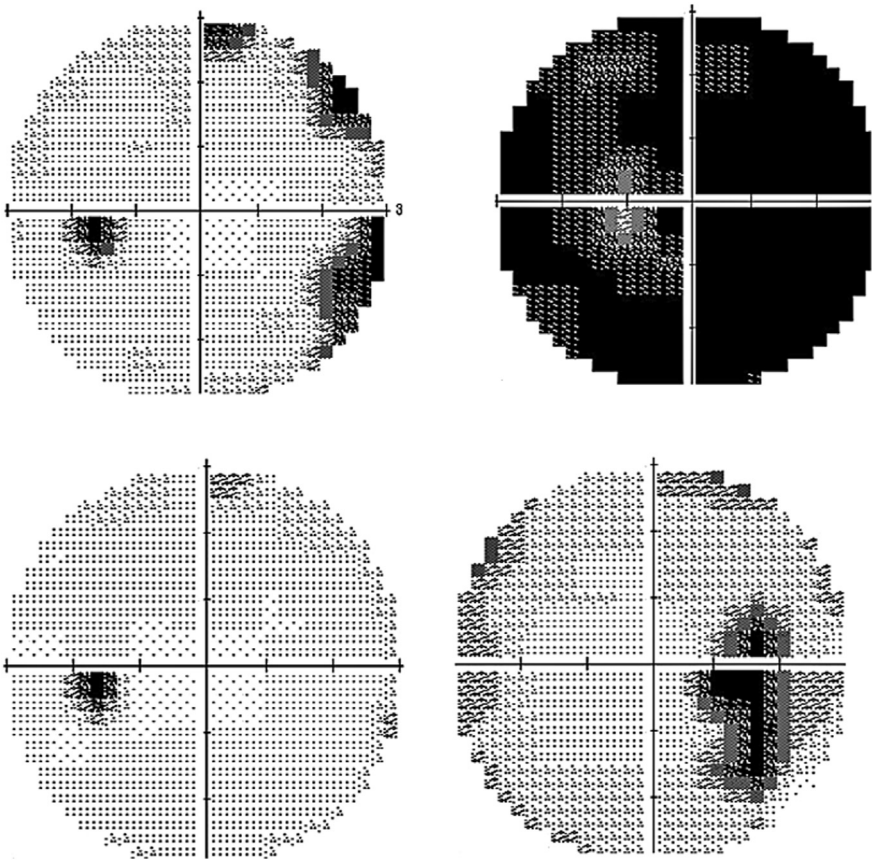

FIGURE 6. Case 2. Humphrey field analyzer results in a patient with multiple evanescent white dot syndrome. (Top, left and right) At the first examination, decreased sensitivity (mean deviation [MD], -32.33 decibels) is seen in almost all areas of the central 30-2 in the right eye and pericentral depression (MD, −6.10 decibels) in the left eye. (Bottom, left and right) After 12 weeks, an enlarged blind spot remains in the right eye but the left eye is normal.

centrifugally expanding lesion from the optic disc to the peripheral fundus supports this assumption. As we previously reported,[3] visual field defects were detected frequently in the unaffected fellow eyes. In the current study, GP showed enlargement of a blind spot in the unaffected eyes in 4 of the 14 eyes. Because FAF and OCT showed no fundus abnormalities in the unaffected eyes, the responsible lesion may be in the retrobulbar optic nerve. If inflammation occurs in the subarachnoid space of the optic nerve, a visual field abnormality in the unaffected eye can be explained.

The current study had some limitations. Although FAF images have a 200-degree angle, the OCT images were taken using a 9-millimeter B-scan in the posterior pole. We could not compare the OCT and FAF images outside of the posterior fundus surrounded by a vascular arcade. The 2 modalities were compared only in the posterior fundus.

In conclusion, ultra-wide-field FAF showed centrifugal spreading of the affected lesions in MEWDS. The lesions appear to arise from the optic disc and radiate to the peripheral fundus.

BOTH AUTHORS HAVE COMPLETED AND SUBMITTED THE ICMJE FORM FOR DISCLOSURE OF POTENTIAL CONFLICTS OF Interest and none were reported. No sponsor or funding organization was involved in the study. Contributions of authors: design of the study (S.K., H.H.); conduct of the study (S.K., H.H.); collection, management, analysis, and interpretation of the data (S.K., H.H.); and manuscript preparation (S.K., H.H.), review (S.K., H.H.), or approval (S.K., H.H.).

TABLE 2. Changes of Visual Field in Multiple Evanescent White Dot Syndrome

Case	Affected Eye	Visual Field (First Visit)
1	OS	OD normal/OS enlarged blind spot
2	OD	OD enlarged blind spot/OS normal
3	OD	OD enlarged blind spot, temporal, inferior, and superior scotoma/OS slight superior scotoma
4	OD	OD enlarged blind spot, temporal, inferior, and superior scotoma/OS normal
5	OD	OD enlarged blind spot/OS inferior arcuate scotoma
6	OS	OD slight enlarged blind spot/OS enlarged blind spot
7	OD	OD enlarged blind spot/OS enlarged blind spot
8	OD	OD enlarged blind spot and central scotoma/OS normal
9	OD	OD enlarged blind spot/OS normal
10	OD	OD enlarged blind spot/OS normal
11	OD	OD enlarged blind spot and inferior scotoma/OS normal
12	OS	OD enlarged blind spot and central scotoma/OS normal
13	OS	OD normal/OS enlarged blind spot and superior nasal depression
14	OS	OD normal/OS enlarged blind spot and central scotoma

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Biosketch

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