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A case of secondary multiple evanescent white dot syndrome in a patient with preexisting wet age-related macular degeneration

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ABSTRACT

Purpose: To report a case of secondary Multiple Evanescent White Dot Syndrome in a patient with preexisting wet age-related macular degeneration.

Observation: A 75-year-old male on treat and extend regimen for wet age-related macular degeneration (AMD) presented with a sudden loss of vision and saw central dark shadow in the right eye (RE) for a duration of 1 week. There was no significant history preceding the visual loss. Examination showed a visual acuity (VA) of counting fingers at 1 meter in the right eye and 20/25 in the left eye. Anterior segment examination was unremarkable with dilated fundus examination showing a clear vitreous, tortuous blood vessel, a hyperemic disc and fibrosis at the macula. The left eye (LE) examination was unremarkable. Optical Coherence Tomography (OCT) showed fibrosis due to the previous wet AMD and hyperreflective excrescences projecting from the retinal pigment epithelium (RPE) outside of the old area of wet AMD. Fundus Fluorescein Angiogram (FFA) showed hyperfluorescent spots in a wreath-like pattern increasing in intensity in the early phase and showing late staining towards the late phase while Indocyanine green angiography (ICGA) did not clearly delineate the lesions. Fundus autofluorescence (FAF) revealed hyper Autofluorescence (AF) at the posterior pole. Optical Coherence Tomography Angiography (OCTA) revealed a flow reduction in the choriocapillaris of the affected area. Basic blood investigations with Venereal Disease Research Laboratory (VDRL), syphilitic IgM and IgG antibodies, Quantiferon TB gold test, complete renal function tests and liver function tests were performed. All the blood investigations were within normal limits and the workup for syphilis and tuberculosis was negative. The patient was started on 1mg/kg body weight of oral prednisolone (after the non-response to low dose of oral steroids) with the diagnosis of secondary multiple evanescent white dot syndrome (MEWDS) secondary to wet AMD. The patient was followed up every weekly and the last visit showed improvement in visual acuity to 20/50 with resolution of lesions on FAF and OCT macula.

Conclusion and importance: Secondary MEWDS is extremely rare and unique in terms of its presentation and its association with preexisting chorioretinal disease where there is damage to the choriocapillaris- Bruch's membrane-RPE complex. This case report highlights one such rare case scenario and how multimodal imaging helps in the diagnosis, management and follow-up of patients with secondary MEWDS.

1. Introduction

Multiple Evanescent White dot syndromes (MEWDS) is often preceded by a flulike illness particularly in healthy young females between the second and fourth decades of life and resolves without any treatment. Clinically, they present as grey-white lesions extending from the posterior pole to the equator and at the level of retinal pigment epithelium or outer retina with the most characteristic finding of granularity at the fovea and disruption and attenuation of the ellipsoid zone on optical coherence tomography (OCT).[1–3] Fundus Fluorescein angiography (FFA) shows a classical wreath-like pattern of punctate hyperfluorescent spots in the early phase followed by late staining[4] while

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Indocyanine green angiography (ICGA) shows hypocyanescent spots. Hypocyanescence is due to the inflammatory debris affecting the outer blood-retinal barrier and reduced uptake by the RPE.[4,5] Fundus Auto Fluorescence (FAF) shows hyper autofluorescence (AF) that is believed to be due to inflammation causing increased phagocytosis of the photoreceptor outer segments and increased production of lipofuscin.[4–7] It affects the outer layer of the photoreceptors (neuroretinitis) causing loss of the ellipsoid zone which recovers in most cases.[8] The hypothesized mechanism is that the interaction of genetics, inflammation, autoimmune mechanisms and environmental triggers could cause MEWDS.[8] MEWDS has been associated with various infections and following vaccination.[9–15]

Secondary MEWDS is seen in association with diseases that cause disruption of the Bruch's membrane – Retinal Pigment Epithelium – Choriocapillaris complex. The lesions in secondary MEWDS are reported to have a course similar to that of primary MEWDS except that they have the additional clinical features of underlying triggering retinal disease.[16] There are not many cases that are reported in the literature regarding secondary MEWDS. We would like to report one such rare case of secondary MEWDS where the patient had a pre-existing wet AMD with disruption of the Bruch's membrane – Retinal Pigment Epithelium – Choriocapillaris complex. Our patient did not have much clinical findings but multimodal imaging greatly helped us in the diagnosis and monitoring of our patient.

2. Case report

A 75-year-old male with secondary MEWDS secondary to neovascular AMD with polypoidal choroidal vasculopathy (PCV) subtype presented with a sudden drop in vision and subjective complaint of a central dark shadow in the right eye for a duration of 1 week. There was no flu-like illness before the onset of symptoms. There was no redness, pain or photophobia before the onset of vision loss. He was not a known hypertensive or diabetic patient and did not have any other underlying systemic disorder.

This acute event was not associated with choroidal neovascularization (CNV) recurrence as the underlying neovascular AMD (nAMD) was well-controlled with photodynamic therapy (PDT) and intravitreal ranibizumab and aflibercept injections (7 aflibercept and 4 ranibizumab). He was on a 12 weekly treat and extend regimen before the onset of the present complaints. Fig. 1 shows the FFA, ICG and OCT of the PCV lesion and the area of involvement of PCV and MEWDS on FAF. Examination showed best corrected visual acuity (BCVA) of counting fingers at 1 m in the RE and 20/25 in the LE. BCVA in the RE prior to the onset of vision loss was 20/32. Anterior segment examination of the RE was unremarkable. Dilated fundus examination revealed subretinal fibrosis and a quiescent PCV lesion. The vitreous was clear but optic disc was hyperemic with burring of disc margins and tortuosity of blood vessels. There were no dots or spots or any granularity at the fovea (apart from the scar due to the previous PCV) that were evident on clinical examination. LE examination was unremarkable.

FFA and ICGA showed changes consistent with MEWDS and were observed outside of the original PCV lesion. These changes included hyperfluorescent spots increasing in size and intensity from the early phase towards the late phase concentrated at the posterior pole. ICGA did not delineate the lesions clearly, however, showed hypocyanescent area between the optic disc and the macula throughout various phases of the angiogram. Similarly, FAF showed a diffuse hyper AF area concentrated at the posterior pole extending beyond the margins of the original lesion. OCT macula of the RE showed well-defined sub retinal hyperreflective material corresponding to fibrosis from the treated PCV lesion. Hyperreflective excrescences were projecting from the RPE into the inner retinal layers and ellipsoid zone loss extending outside of the area of the old PCV lesion. Fig. 2 shows the secondary MEWDS lesions in the color fundus photo, FFA, ICGA, FAF and OCT. Optical coherence tomography angiography (OCTA) of the affected area showed areas of signal reduction in the choriocapillaris (seen after segmentation) which could have been due to reduced flow or masking.

Full blood count with erythrocyte sedimentation rate and C-reactive protein done was within normal limits. VDRL was non-reactive and syphilitic IgM and IgG antibodies were negative. Quantiferon TB gold was negative, and liver function tests and renal function tests were normal. The patient was negative for Covid and did not give any recent h/o Covid vaccination. Table 1 gives the details of the investigations done for the patient and their respective results.

The patient was provisionally diagnosed as secondary MEWDS on the basis of clinical presentation and investigations of MEWDS and pre-existing wet AMD (PCV in our patient). The patient was initially

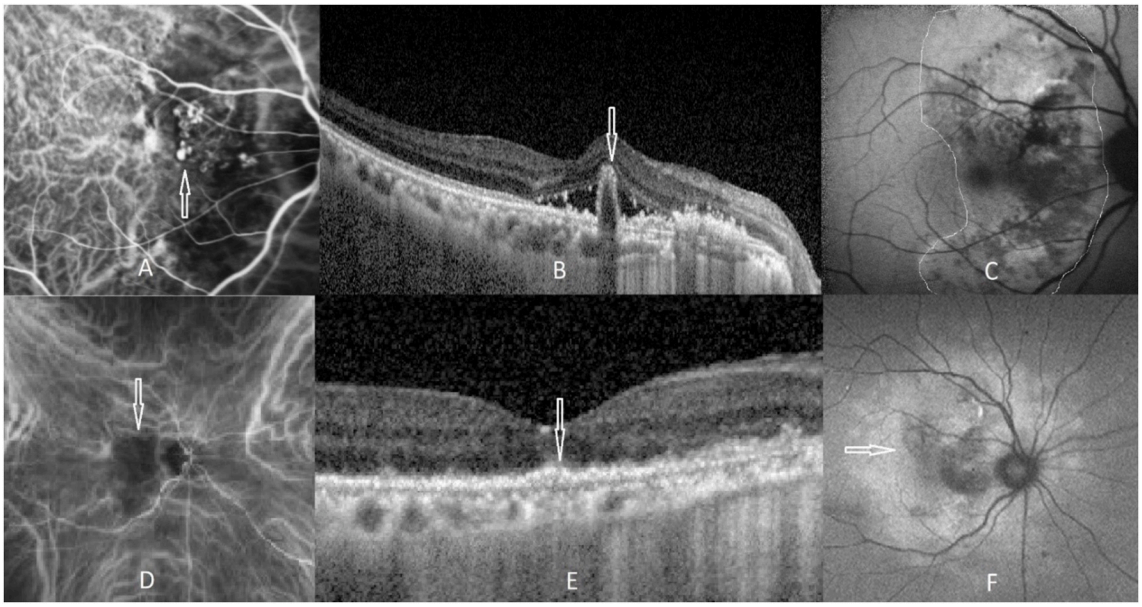

Fig. 1. shows the initial PCV on (A) ICG, (B) OCT as tall tented PED with sub RPE ring, double layer sign and subretinal fluid, (C) FAF delineating the area of involvement of the previous PCV (D) ICG showing hypocyanescence of the MEWDS area between the optic disc and the macula (E) OCT showing RPE excrescences in MEWDS (F) FAF of the MEWDS affected area all in white arrows.

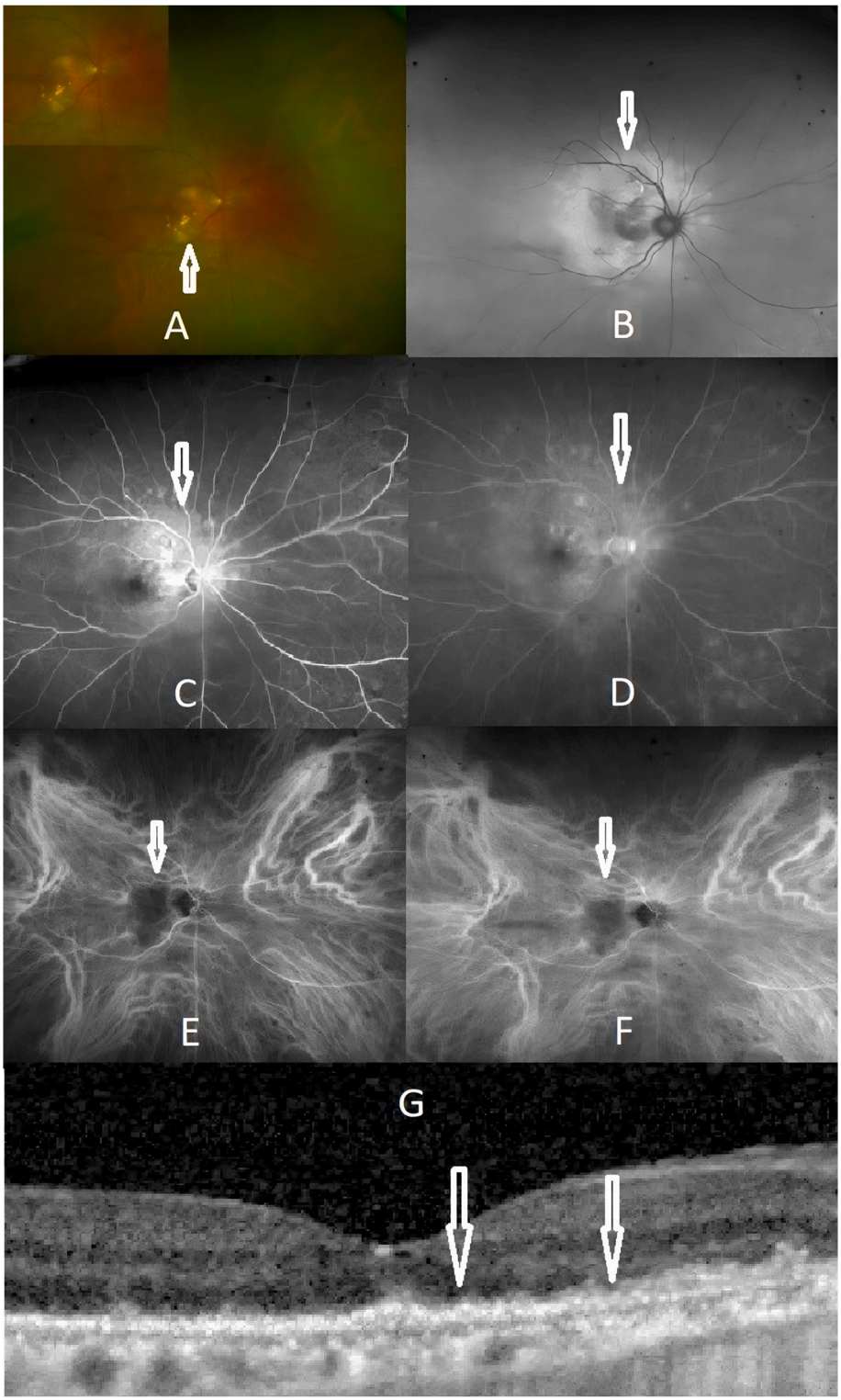

Fig. 2. shows the MEWDS lesions on (A) color fundus photograph with inset, (B) FAF showing hyper AF spots, (C) and (D) early and late phases of FFA showing early punctate hyperfluorescent spots and late staining in MEWDS, (E) and (F) early and late phases of ICGA showing area of hypocyanescence between the optic disc and macula and (G) showing OCT of the RE with RPE excrescences all in white arrows. (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

started on 60 mg of oral prednisolone and was reviewed after 5 days. BCVA in the RE was the same as before but OCT showed subfoveal hyperreflective lesion extending from the RPE into the inner retinal layers. FAF showed an increase in the hyper AF to the periphery that was not present earlier. The dosage of oral prednisolone was increased to 80 mg once daily (1mg/kg body weight) in view of subjective non-improvement of vision and worsening OCT and FAF findings. He was regularly followed up every week and the dosage of oral prednisolone was tapered by 10 mg every 2 weeks as there was an improvement in visual acuity and OCT and FAF findings. The last visit showed an

Table 1
List of blood investigations done for the patient.

Hematology	
Hemoglobin	17.0 gm/dl
WBC count	6.64 cells/mm^3
Differential count	N - 53.9%, L - 34.6%, Monocyte −7.4%, Eosinophil - 3.3%, Basophil - 0.8%
Erythrocyte sedimentation rate	3 mm/h
C-Reactive protein	4.8 mg/L
Biochemistry	
Serum urea	5.5 m mol/L
Serum creatinine	78 u mol/L
Serum Chloride	102 m mol/L
Serum Bicarbonate	27.2 m mol/L
Serum Glucose	4.6 u mol/L
Liver function tests	
Total serum protein	74 G/L
Serum albumin	43 G/L
Serum alkaline phosphatase	92 U/L
Serum alanine transferase	28 U/L
Serum aspartate transaminase	23 U/L
Total serum bilirubin	14 u mol/L
Microbiology	
Serum VDRL Syphilis antibody	Non-reactive
Syphilis blood IgG Immunoassay	Negative
Syphilis blood IgM Immunoassay	Negative
Quantiferon TB Gold test	Negative

improvement in the BCVA to 20/50. The RPE excrescences had reduced markedly and so as the subfoveal hyperreflective lesion and FAF findings. However, the EZ loss still persisted accounting for the non-recoverable pre-MEWDS VA to20/32 (Fig. 3). OCTA showed improvement in the choriocapillaris flow after starting the patient on oral steroids (Fig. 4).

3. Discussion

Our patient was diagnosed with MEWDS based on the unilaterality of the clinical findings, classic wreath-like pattern on FFA, hyper AF lesions on FAF at the posterior pole, subfoveal hyperreflective lesion on OCT with RPE excrescences and EZ loss. FAF has a crucial role to play in such cases as it can delineate the MEWDS lesions from the original pathological lesion (PCV in our patient) and its origin and distribution form the primary lesion. Careful observation is however necessary in such cases as the old lesion from wet AMD can mask the signs of secondary MEWDS on FAF.

Secondary MEWDS is believed to be triggered by macular disease or iatrogenic retinal injury. MEWDS has been seen in association with Best disease with secondary CNV, toxoplasma chorioretinitis, acute zonal occult outer retinopathy, pseudoxanthoma elasticum with angioid streaks, retinal trauma, retinal detachment, retinopexy, multifocal choroiditis, subretinal hemorrhage, atrophic scars (idiopathic or secondary to causes other than Multifocal choroiditis/Punctate Inner choroidopathy without CNV), CNV where there is disruption of the choriocapillaris-Bruch's membrane-RPE complex.[16,17] Though, type 4 hypersensitivity reaction has earlier been speculated, disruption of the choriocapillaris- Bruch's membrane-RPE complex leading to compromise of the posterior retinal-blood barrier and subsequent loss of immune privilege by the outer retina/EZ has also been hypothesized. Retinal autoantibodies are assumed to be produced due to interaction between the immune system and immune-privileged retinal antigens. These in turn trigger changes predominantly in the outer retina with lesions appearing close to the preexisting chorioretinal lesions and spreading later to the periphery which is classical of secondary MEWDS. This is otherwise described as an epiphenomenon wherein a secondary pathology is seen aside from a primary pathological manifestation. The lesions in the secondary MEWDS follow an independent path of progression and resolution independent of the associated primary disease.[16–18] Our patient belongs to this category of secondary MEWDS where there is a pre-existing wet AMD lesion with disruption of the choriocapillaris-Bruch's membrane-RPE complex and secondary MEWDS lesions starting close to the area of the primary chorioretinal lesion before spreading to the periphery.

Histopathological changes of the PDT-treated areas in AMD have shown uniform occlusion of the choriocapillaris layer that is evidenced by swelling of the vascular endothelial cells, their detachment from the basement membrane and progressing to rupture, fragmentation, and complete degeneration. Focal areas of vacuolar degeneration of RPE have been observed along with the bullous separation of individual epithelial cells from Bruch's membrane.[19] The combined mechanism of occlusion of choriocapillaris and RPE-Bruch's membrane disturbance following PDT might have led to the formation of MEWDS in an already

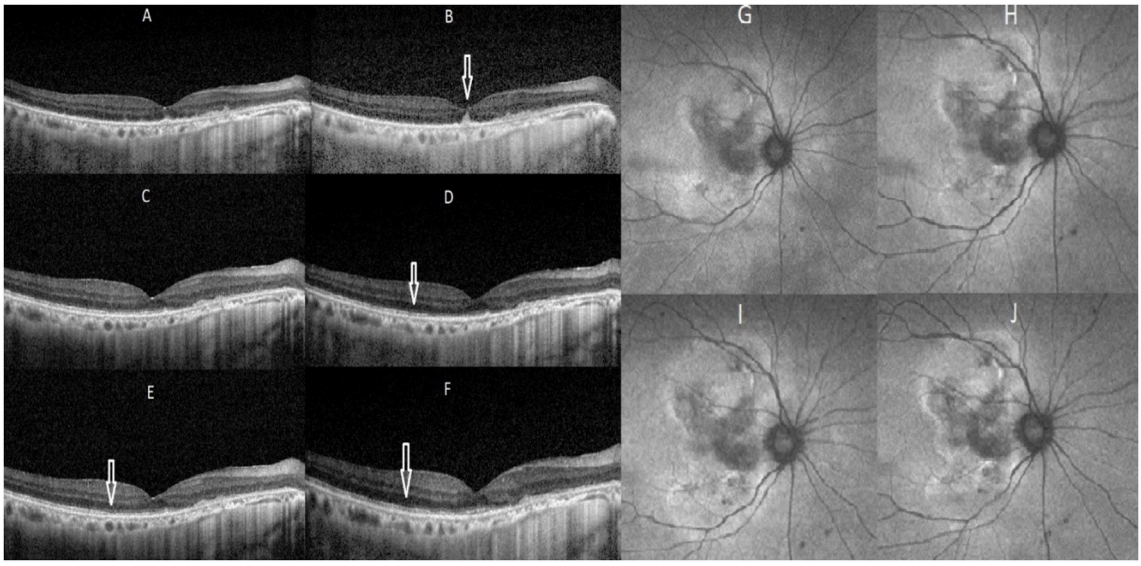

Fig. 3. Overview of the OCT lesions from the RPE to resolution of lesions with EZ granularity and disruption after starting on oral steroids all in white arrows (A to F). Overview of FAF lesions showing resolution with oral steroids (G-J).

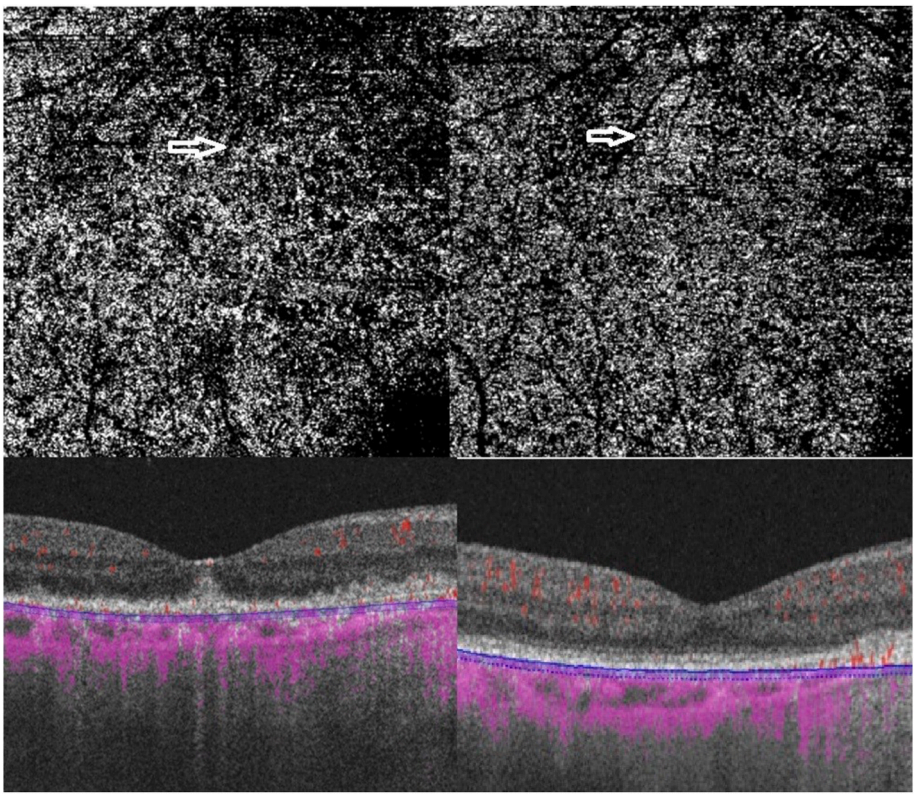

Fig. 4. OCTA at the choriocapillaris level showing reduction of flow before starting oral steroids and improvement of flow after treatment with oral steroids (white arrow).

compromised choriocapillaris-RPE-Bruch's membrane complex due to AMD.

Secondary MEWDS has been shown to have bilaterality and recurrences because of the persistence of the underlying primary pathology and the possibility of an underlying systemic etiology.[16,17] Our patient did not show either of these was similar to the findings of Serrar et al. where the recurrences and bilateral involvement was less frequent.[17] The usual classical feature of hypocyanescent lesions was less extensive when compared to that of FFA findings in secondary MEWDS and that was consistent with the findings of Serrar et al.[17]

The demographics of secondary MEWDS is slightly older than the primary with a young mean age. Our patient was an elderly non-myopic male. The clinical presentation of the lesions in our patient was minimal at the baseline but showed an increase in the foveal involvement and periphery in the second visit (1 week later) that was not comparable to the findings of Serrar et al.[17] Similarly, those with secondary MEWDS had worse baseline VA that might be attributed to the underlying macular pathology. Secondary MEWDS has been shown to have subclinical inflammation compared to primary MEWDS but our patient did not have anterior chamber inflammation or vitreous cells. The choroidal thickness (CT) of our patient was 231μm at baseline which did not reduce over the course of the disease and that might be attributed to the underlying macular scar from previous wet AMD. This is in contrast to Serrar et al. where there were variations in CT during the active stages and stages of resolution in both primary and secondary MEWDS.[17]

Gal-or et al.[20] and Perriera et al. did not observe any flow alteration in the retinal vasculature and choriocapillaris on OCTA.[20,21] These findings were consistent with the findings of Pichi et al.,[22] where the authors hypothesized that MEWDS is a disease of photoreceptors (photoreceptoritis) and not a disease of the choriocapillaris and hence normal flow in the choriocapillaris is justified. Lages et al. hypothesized that OCTA may not pick up transient flow changes in the choriocapillaris as it is a disease of the end capillaries with very low flow.[23] However, Kochthali et al. reported flow reduction in the choriocapillaris of the affected area using OCTA that resolved with the resolution of the disease as seen in our patient.[24]

MEWDS is considered a self-limiting condition and does not warrant treatment with steroids. However, oral or pulse steroid therapy with methylprednisolone has been reported in the literature. Systemic steroids have been shown to reduce the duration of disease and hasten visual recovery.[24–26] Noroozneshad et al. reported reduction of lesions in the fundus with restoration of EZ in OCT after 3 weeks of starting the patient on oral prednisolone and, absence of recurrence until 9 months of follow-up with tapering dosage of oral steroids.[24] Sheng et al. reported remarkable improvement in visual acuity, photopsia, resolution of fundus lesions, improvement of central scotoma and resolution of lesions in OCT within 1 week of starting the patient on 40 mg of methylprednisolone who had poor vision in the other eye.[25] Takahashi et al. reported improvement in VA and resolution of visual field defect and no recurrence with pulse steroid therapy for 3 days. Our patient was started on 60 mg of oral steroids initially because of poor visual acuity of counting fingers on presentation (in contrast to 20/32, 1 month before the onset of secondary MEWDS) and the patient was very anxious to get it treated at the earliest. The dosage was increased to 1mg/kg body weight subsequently because of increase in the lesions on OCT and FAF with non-improvement in VA. Visual acuity improved thereafter and the final VA was 20/50 during the last visit with OCT and FAF showing resolution of the lesions.

4. Conclusion

This case report highlights a case of secondary MEWDS about which not much has been reported in the literature. This case report also emphasizes the importance of multimodal imaging in the diagnosis and monitoring of patients with central scotoma and minimal clinical findings. Also, MEWDS should be suspected in patients with an unusual

presentation with an underlying chorioretinal pathology that might act as a triggering agent.

Patient consent

Written consent to publish this case has not been obtained. This report does not contain any personal identifying information.

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Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

CRediT authorship contribution statement

Priya R. Chandrasekaran: Writing – original draft, Methodology, Formal analysis, Data curation, Conceptualization. **Hiok Hong Chan:** Validation, Supervision. **Tien-En Tan:** Validation, Supervision. **Farah N.I. Ibrahim:** Validation, Supervision. **Jinzhi Zhao:** Validation, Supervision. **Kelvin Y.C. Teo:** Writing – review & editing, Validation, Supervision, Methodology, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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References

1. Jampol LM, Sieving PA, Pugh D, Fishman GA, Gilbert H. Multiple evanescent white dot syndrome. I. Clinical findings. *Arch Ophthalmol*. 1984;102(5):671–674. https://doi.org/10.1001/archopht.1984.01040030527008.
2. Polk TD, Goldman EJ. White-dot chorioretinal inflammatory syndromes. *Int Ophthalmol Clin*. 1999;39(4):33–53. https://doi.org/10.1097/00004397-199903940-00005.
3. Raven ML, Ringeisen AL, Yonekawa Y, Stem MS, Faia LJ, Gottlieb JL. Multi-modal imaging and anatomic classification of the white dot syndromes. *Int J Retina Vitreous*. 2017;3:12. https://doi.org/10.1186/s40942-017-0069-8.
4. Dell'Omo R, Pavesio CE. Multiple evanescent white dot syndrome (MEWDS). *Int Ophthalmol Clin*. 2012;52(4):221–228. https://doi.org/10.1097/IIO.0b013e31826647ed.
5. Tavallali A, Yannuzzi LA. MEWDS, common cold of the retina. *J Ophthalmic Vis Res*. 2017;12(2):132–134. https://doi.org/10.4103/jovr.jovr_241_16.
6. Hua R, Chen K, Liu LM, Liu NN, Chen L, Teng WP. Multi-modality imaging on multiple evanescent white dot syndrome-A Spectralis Study. *Int J Ophthalmol*. 2012;5(5):644–647. https://doi.org/10.3980/j.issn.2222-3959.2012.05.21.
7. Marsiglia M, Gallego-Pinazo R, Cunha de Souza E, et al. Expanded clinical spectrum of multiple evanescent white dot syndrome with multimodal imaging. *Retina*. 2016;36(1):64–74. https://doi.org/10.1097/IAE.0000000000000685.
8. Pichi F, Srvivastava SK, Chexal S, et al. En face optical coherence tomography and optical coherence tomography angiography of multiple evanescent white dot syndrome: new insights into pathogenesis. *Retina*. 2016;36(Suppl 1):S178–S188. https://doi.org/10.1097/IAE.0000000000001255.
9. Yang CS, Hsieh MH, Su HI, Kuo YS. Multiple evanescent white dot syndrome following acute Epstein-Barr virus infection. *Ocul Immunol Inflamm*. 2019;27(2):244–250. https://doi.org/10.1080/09273948.2017.1371763.
10. Fine L, Fine A, Cunningham Jr ET. Multiple evanescent white dot syndrome following hepatitis a vaccination. *Arch Ophthalmol*. 2001;119(12):1856–1858. https://doi.org/10.1001/archopht.119.12.1870.
11. Baglivo E, Safran AB, Borruat FX. Multiple evanescent white dot syndrome after hepatitis B vaccine. *Am J Ophthalmol*. 1996;122(3):431–432. https://doi.org/10.1016/s0002-9394(14)72074-4.
12. Ogino K, Kishi S, Yoshimura N. Multiple evanescent white dot syndrome after human papillomavirus vaccination. *Case Rep Ophthalmol*. 2014;5(1):38–43. https://doi.org/10.1159/000358870. Published 2014 Feb 1.
13. Haw YL, Yu TC, Yang CS. A CARE-compliant article: a case report of possible association between recurrence of multiple evanescent white dot syndrome and the Herpesviridae family. *Medicine (Baltim)*. 2020;99(15), e19794. https://doi.org/10.1097/MD.0000000000019794.
14. Peck T, Moushmoush O, Venkat A. A Case of MEWDS Following COVID-19 Infection. https://retinatoday.com/articles/2022-may-june/a-case-of-mewds-following-covid-19-infection. https://assets.bmctoday.net/retinatoday/pdfs/0522RT_Medical_Retina.pdf.
15. Soifer M, Nguyen NV, Leite R, Fernandes J, Kodati S. Recurrent multiple evanescent white dot syndrome (MEWDS) following first dose and booster of the mRNA-1273 COVID-19 vaccine: case report and review of literature. *Vaccines*. 2022;10:1776. https://doi.org/10.3390/vaccines10111776.
16. Essilfie J, Bacci T, Abdelhakim AH, et al. Are there two forms of multiple evanescent white dot syndrome? *Retina*. 2022;42(2):227–235. https://doi.org/10.1097/IAE.0000000000003288.
17. Serrar Y, Cahuzac A, Gascon P, et al. Comparison of primary and secondary forms of multiple evanescent white dot syndrome. *Retina*. 2022;42:2368–2378.
18. Ong AY, Birtel J, Agorogiannis E, Sharma SM, Issa PC. Topographic patterns of retinal lesions in multiple evanescent white dot syndrome. *Graefes Arch Clin Exp Ophthalmol*. 2023. https://doi.org/10.1007/s00417-023-06032-1.
19. Schmidt-Erfurth U, Laqua H, Schlötzer-Schrehard U, Viestenz A, Naumann GOH. Histopathological changes following photodynamic therapy in human eyes. *Arch Ophthalmol*. 2002;120(6):835–844.
20. Gal-Or O, Priel E, Rosenblatt I, Shulman S, Kramer M. Multimodal imaging in an unusual cluster of multiple evanescent white dot syndrome. *Journal of Ophthalmology*. 2017. https://doi.org/10.1155/2017/7535320.
21. Pereira F, Lima LH, de Azevedo AG B, Zett C, Farah ME, Belfort Jr R. Swept-source OCT in patients with multiple evanescent white dot syndrome. *Journal of Ophthalmic Inflammation and Infection*. 2018;8:16. https://doi.org/10.1186/s12348-018-0159-2.
22. Lages V, Mantovani A, Papadia M, Herbort CP. MEWDS is a true primary choriocapillaritis and basic mechanisms do not seem to differ from other choriocapillaritis entities. *J Curr Ophthalmol*. 2018;30(281):6.
23. Khochtali S, Dridi T, Abroug N, Ksiaa I, Lupidi M, Khairallah M. Swept- source optical coherence tomography angiography shows choriocapillaris flow reduction in multiple evanescent white dot syndrome. *Journal of Current Ophthalmology*. 2020. https://doi.org/10.4103/JOCO.JOCO_107_20.
24. Norooznezhad AH, Mohammadzadeh V, Kadivar S, Ghassemi F. Multiple evanescent white dot syndrome: a case report and experience with corticosteroid therapy. *Iran J Allergy, Asthma Immunol*. 2020;19(Supple.1):91–94. https://doi.org/10.18502/ijaai.v19i(s1.r1).2865.
25. Sheng Y, Sun W, Gu YS. Spectral-domain optical coherence tomography dynamic changes and steroid response in multiple evanescent white dot syndrome. *Int J Ophthalmol*. 2017;10(8):1331–1333.
26. Takahashi Y, Ataka S, Wada S, Kohno T, Nomura Y, Shiraki K. A case of multiple evanescent white dot syndrome treated by steroid pulse therapy. *Osaka City Med J*. 2006;52:83–86.