

SHORT COMMUNICATION

Intravitreal ranibizumab in a patient with choroidal neovascularization secondary to multiple evanescent white dot syndrome

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PURPOSE. *To report the effect of a single intravitreal injection of ranibizumab in a patient with choroidal neovascularization (CNV) secondary to multiple evanescent white dot syndrome (MEWDS).*

METHODS. *A 65-year-old woman with visual acuity (VA) 20/40, mild vitreous inflammation, optic disc edema, and white deep retinal round lesions in the right eye underwent fluorescein angiography, indocyanine green angiography, and optical coherence tomography. The diagnosis of MEWDS with peripapillary CNV was made and a single injection of ranibizumab (0.5 mg) was administered.*

RESULTS. *At the 6-month follow-up visit, the VA in the right eye was 20/20, the CNV completely regressed, and the MEWDS findings disappeared.*

CONCLUSIONS. *Intravitreal ranibizumab appears to be a safe and effective treatment option in cases of CNV secondary to MEWDS, resulting in fast resolution of the macular edema and regression of the CNV. On the other hand, it is unclear whether the administration of ranibizumab contributed to a prompt regression of MEWDS. (Eur J Ophthalmol 2007; 17: 996-9)*

KEY WORDS. *Choroidal neovascularization, High-speed ICG, Multiple evanescent white dot syndrome, White spots, Ranibizumab*

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INTRODUCTION

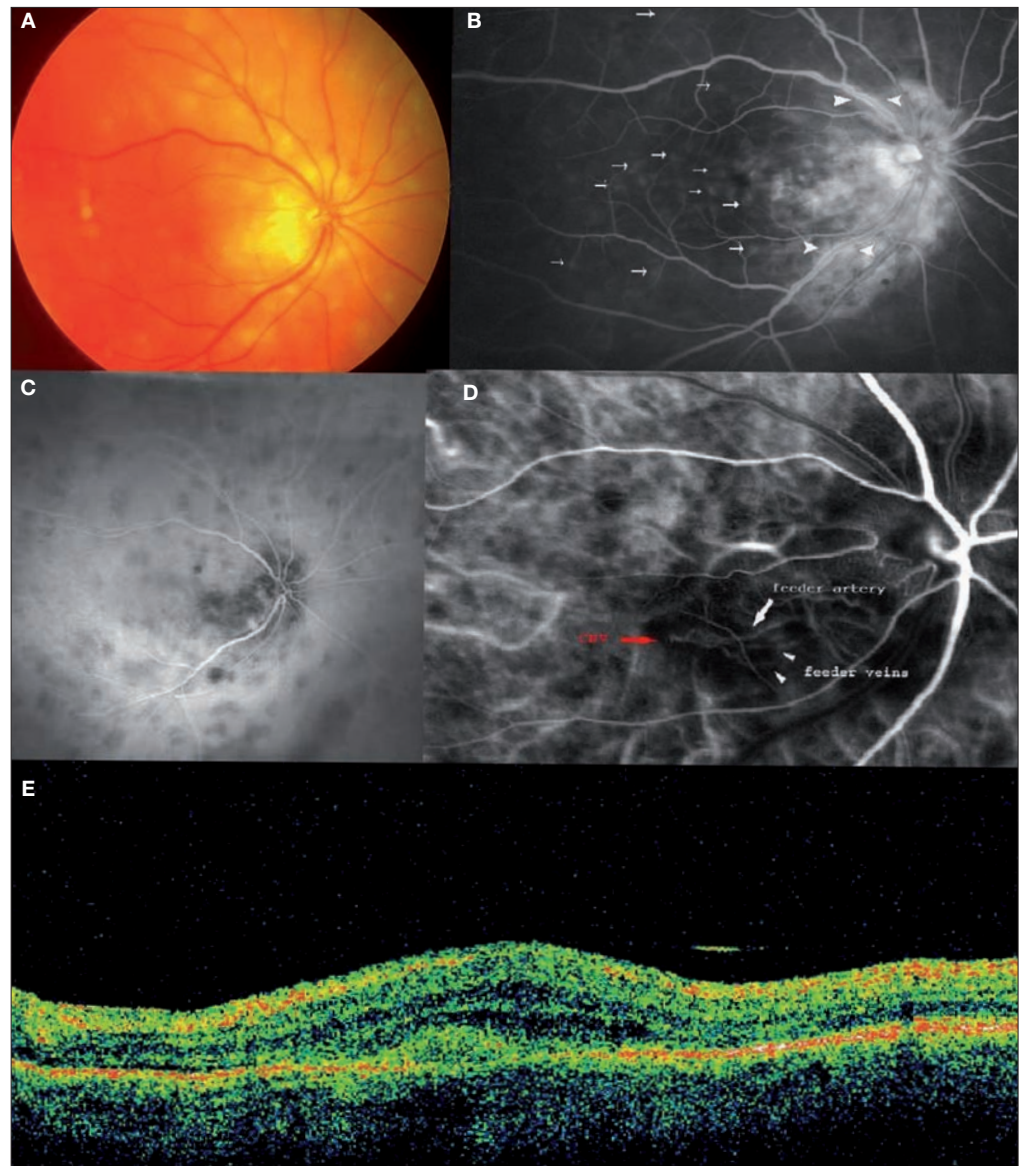
Multiple evanescent white dot syndrome (MEWDS), which was first described by Jampol et al in 1984 (1), is a benign inflammatory disorder with a strong female predilection. It primarily affects young adults and is typically unilateral. Patients present with various symptoms such as blurred vision, dyschromatopsia, photopsia, visual field defects located temporally or paracentrally, and sudden visual alterations in one eye (2). The etiology of MEWDS remains unknown. However, it is suspected to be the result of a viral infection in a genetically susceptible person (3-6). MEWDS is characterized by the development of small transient white dots at the level of the outer retina, the

retinal pigment epithelium, and the inner choroids which have characteristic appearance on fluorescein angiography (FA) and indocyanine green angiography (ICG). The prognosis of MEWDS is generally very good. However, the rare occurrence of choroidal neovascularization (CNV) (5, 7-9) may lead to permanent visual loss.

Case report

A 65-year-old woman presented to us with reports of blurred vision and metamorphopsia in the right eye for the past 10 days. Her visual acuity (VA) was 20/40 in the right eye and 20/20 in the left eye. Dilated fundus examination

Fig. 1 - Color (A), fluorescein angiography (B), indocyanine green angiography (ICG) (C, D), and optical coherence tomography (OCT) (E) images of the right eye at baseline. Note the white spots in the color photograph (A). The FA shows hyperfluorescent dots (white arrows, B) and diffuse hyperfluorescence, phlebitis (white arrowheads, B), and disc edema (B). The ICG reveals the late hypofluorescent spots and dots (C) and the choroidal neovascularization (CNV) (red arrow) with feeder vessels (white arrows, D). Cystoid macular edema as well as the CNV are shown on the OCT scan (E).



disclosed mild vitreous cells, edematous optic disc, and white dots scattered in the posterior pole of the right eye (Fig. 1A). The left eye was normal. Consequently, FA and ICG were performed utilizing the Heidelberg scanning laser ophthalmoscope (Heidelberg Engineering GmbH, Dossenheim, Germany). The FA showed hyperfluorescent dots, intraretinal leakage consistent with cystoid macular edema, phlebitis, optic disk edema with leakage, and diffuse hyperfluorescence peripapillary (Fig. 1B). The ICG showed scattered hypofluorescent dots and spots in a wreath-like manner in the late phases (Fig. 1C). Furthermore, the analysis of the high-speed angiograms revealed

the presence of a classic peripapillary CNV, located temporal to the optic disc (Fig. 1D), which would otherwise be undetectable on the FA due to the diffuse hyperfluorescence in the juxtapapillary area. Optical coherence tomography (OCT) showed cystoid macular edema (CME) and an area of hyperreflectivity corresponding to the CNV in the papillomacular area (Fig. 1E). The diagnosis of CNV secondary to MEWDS was made and we offered the patient intravitreal injection of ranibizumab as a treatment option. The patient signed an informed consent and a single injection of ranibizumab (0.5 mg) was administered.

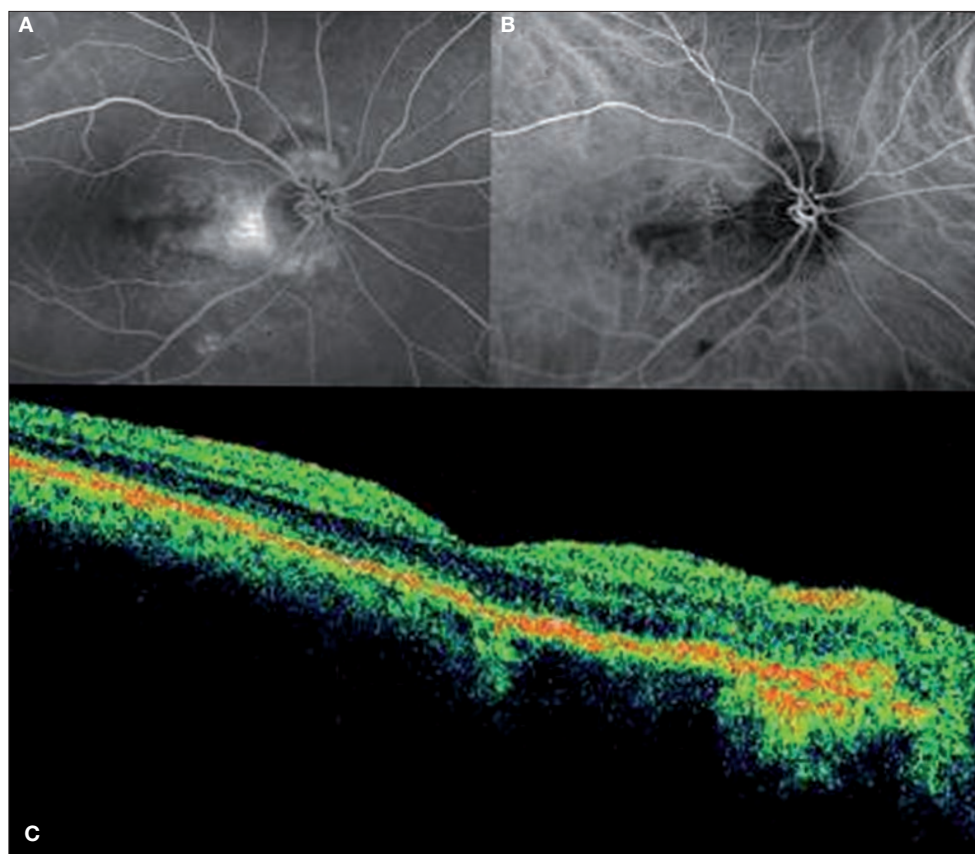


Fig. 2 - Fluorescein angiography (A), indocyanine green angiography (B), and optical coherence tomography (OCT) (C) images of the right eye at the 6-month follow-up visit. Note the resolution of the multiple evanescent white dot syndrome findings (spots and dots, phlebitis, disc staining) and the total regression of the choroidal neovascularization (A, B). The OCT scan (C) reveals resolution of the cystoid macular edema.

Fig. 3 - Color photograph at the 6-month visit showing scarring in the area of the choroidal neovascularization.

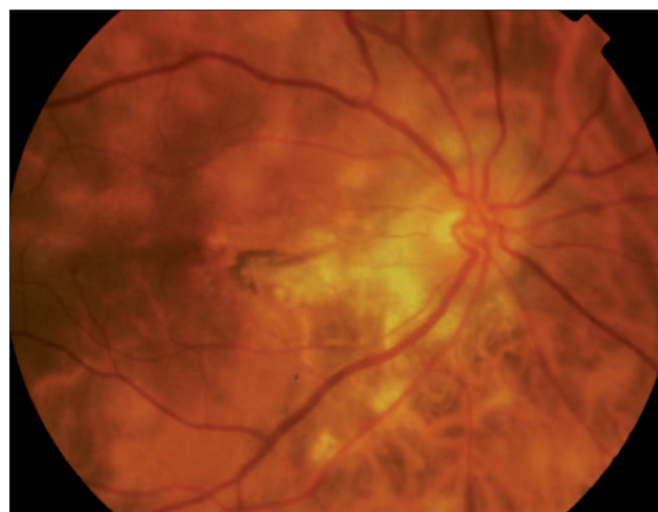


RESULTS

Follow-up examinations were performed 1 day, 1 week, and 1, 2, 3, and 6 months after the injection. The patient's VA at the 6-month follow-up was 20/20 and a total regression of the CNV was observed (Figs. 2A, 2B, and 3). Furthermore, the white dots and the vitreous inflammation had completely resolved at the 1-month visit as well as the phlebitis, the CME, and the disc edema, and this effect has been maintained (Fig. 2). No ocular adverse events have been observed.

DISCUSSION

MEWDS is a benign inflammatory disease which resolves spontaneously without need for treatment. The characteristic lesions of MEWDS are multiple small white dots located at the level of the outer retina, the retinal pigment epithelium, and the inner choroid (1). Mild vitritis is usually present and the lesions typically resolve within the first weeks of the disease. The optic disc may have an edema-



tous or hyperemic appearance. Furthermore, the fovea may have a granular appearance, which may persist after the resolution of the white dots. Despite the possible visual field loss, the photopsia, and the dyschromatopsia which may occur during the acute phase of the disease and persist, the prognosis is very good.

CNV is a rare event that may develop secondary to MEWDS. It has been reported in four previous cases. In the first case (9), a subfoveal CNV was described 4 months after initial presentation, and the second case (5) showed a peripapillary CNV which occurred 6 months after the initial visit. The third case described a CNV that developed 13 years after the initial onset of MEWDS (8) and the fourth case (7) concerned a CNV that occurred 4 weeks after the initial diagnosis. In all of the above cases the visual loss was permanent. In our patient, we assumed that the CNV was secondary to MEWDS and not secondary to age-related macular degeneration (AMD), a very common condition at age 65, as no clinical evidence of AMD was identified in either eye.

Ranibizumab is a fully humanized antibody fragment which has been recently approved for the treatment of CNV secondary to AMD (10). The ANCHOR study demonstrated the effectiveness of ranibizumab in predominantly classic CNV and its superiority versus verteporfin photodynamic therapy. The ANCHOR trial suggested multiple monthly injections of ranibizumab, in order to achieve regression of the CNV and maintain this effect. We experienced a dramatic anatomic and functional response after a single injection. This could be explained by the different nature of the CNV secondary to an inflammatory disorder, such as MEWDS and the CNV secondary to AMD. In AMD, the damage which happens over the years to the RPE cells and the photoreceptors may increase the difficulty, when it comes to the CNV, of regain-

ing the lost vision. This may not happen in inflammatory disorders as their course is not chronic and they do not cause such damage to the RPE cells and photoreceptors. This way, the results after the use of anti-VEGF agents, such as ranibizumab, can be that rapid and impressive. To our knowledge, this is the first report of regression of an inflammatory CNV secondary to MEWDS after a single intravitreal injection of ranibizumab as we could not find a reference to it in a computerized search using MEDLINE. However, we are unsure if ranibizumab contributed to the regression of the MEWDS findings.

This study has obvious limitations, as a single case is described and the follow-up is short. Larger controlled studies with longer follow-up are necessary in order to elucidate the potential role of ranibizumab in CNV secondary to inflammatory disorders such as MEWDS.

The authors have no financial or proprietary interest in any product mentioned in this article.

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