may be considered part of the hamartomatous process.

Paul Hiscott, MD
Liverpool, England
R. Jean Campbell, MD
Dennis M. Robertson, MD
Rochester, Minn
Bertil Damato, MD
Liverpool

This study was supported in part by a grant from Research to Prevent Blindness, Inc, New York, NY, and in part by Mayo Foundation, Rochester, Minn.

The authors have no relevant financial interest in this article.

Corresponding author: R. Jean Campbell, MD, Department of Ophthalmology, Mayo Clinic, 200 First St SW, Rochester, MN 55905.


Atypical Manifestation of Multiple Evanescent White Dot Syndrome With Large Peripapillary Lesion

Multiple evanescent white dot syndrome (MEWDS) is on the spectrum of diseases that includes acute zonal occult outer retinopathy, acute idiopathic blind spot enlargement syndrome, punctate inner choroidopathy, multifocal choroiditis and panuveitis, and acute macular neuroretinopathy.1 We present an atypical case of MEWDS in which a strikingly large white peripapillary outer retinal lesion evolved into a lesion with a more classical MEWDS appearance.

Figure 1. Fundus photograph obtained at the patient’s initial examination demonstrated a peripapillary chorioretinal scar in the right eye (A) and a peripapillary lesion 7 disc areas in size in the outer retina or retinal pigment epithelium layer in the left eye (B). Digital fluorescein angiography in the left eye demonstrated patchy peripapillary choroidal filling and disc hyperfluorescence at 13 seconds (C) and staining of the disc and peripapillary vessels at 15 minutes (D).

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terior pole was otherwise normal in appearance. Fluorescein angiography demonstrated early patchy peripapillary choroidal filling in the left eye and minimal peripapillary hyperfluorescence in the middle of the phase (Figure 1C), as well as late staining of the disc and peripapillary vessels (Figure 1D). The patient underwent intensive workup at another hospital that included routine blood and chemistry examinations, as well as testing for antinuclear antibodies, antineutrophil cytoplasmic autoantibodies, rapid plasma reagin, human immunodeficiency virus, human leukocyte antigen-B27, and erythrocyte sedimentation rate. Results of these tests and that of head magnetic resonance imaging were all unremarkable.

The patient returned 2 weeks later with similar complaints, though stating that the “spots had changed somewhat in appearance.” Visual acuity remained 20/20 OU. Findings in the right eye remained unchanged. However, there were dramatic changes in the left eye. There was substantial resolution of the peripapillary whitening and the development of moderate disc edema (Figure 2A). More interestingly, however, small white dots ranging in size from 100 to 400 µm had emerged in the outer retina or retinal pigment epithelium layer throughout the posterior pole and middle of the periphery (Figure 2B and C). The patient declined both electroretinography and visual field testing. She returned 3 weeks later still complaining of a central “fuzzy spot” in the left eye. Amsler grid testing revealed a temporal defect. Octopus (Interzeag AG, Schlieren, Switzerland) visual field testing revealed a somewhat enlarged blind spot measuring 10° in the left eye. Residual disc hyperemia and elevation were noted, with almost complete resolution of the white dots (Figure 3).

Comment. To our knowledge, there was 1 previous report of MEWDS that initially manifested as a transient circumpapillary retinal lesion that preceded the appearance of white dots. Results in our article and others in which indocyanine green angiography was used to demonstrate peripapillary hypofluorescence may suggest that this site is important in the early stages of the disease. Although the cause of MEWDS remains a mystery, Gass
et al\textsuperscript{1} proposed that in acute zonal occult outer retinopathy viral introduction in the retina may occur at the optic nerve margin and ora serrata because of the absence of surrounding neuroepithelium at these sites. The initial manifestation of transient discrete peripapillary outer retina or retinal pigment epithelium involvement centered around the optic nerve in our patient would be consistent with such a hypothesis. The funduscopic appearance subsequently evolved into more typical MEWDS findings, including deep retinal white dots surrounding the paramacular area with foveal sparing, blurring of the disc margin, and vascular involvement at angiography.\textsuperscript{3} The patient’s subjective complaints of photopsia and temporal scotoma and the blind spot enlargement at visual field testing are all consistent with the diagnosis of MEWDS.\textsuperscript{3} Because of their transient nature, these peripapillary findings may be more common in MEWDS than previously appreciated.

\textbf{Bilateral Congenital Trigemino-abducens Synkinesis}

To our knowledge, congenital bilateral trigemino-abducens synkinesis has not previously been reported. Herein we report a case in which ipsilateral jaw movement occurred.

\textbf{Report of a Case.} A 21-year-old man had ipsilateral jaw movement associated with abduction of either eye. The patient had been aware of this phenomenon since early childhood. He was a term infant with a normal vaginal delivery and had no dysmorphic features or developmental delay. There was neither history of orbital or ocular trauma, strabismus, nor amblyopia nor family history of a similar phenomenon. On examination, his visual acuity was 6/6 OU and his pupils were 3 mm equal and reactive to light and accommodation. Extraocular movements were full with no nystagmus or widening or narrowing of the palpebral fissures in any position of gaze. On abduction to the left or right, subtle ipsilateral jaw movement was noted (Figure). Findings from neurological examination were normal and electromyographic study demonstrated that abduction of the eyes produced ipsilateral masseter and pterygoid activity.

\textbf{Comments.} Freedman and Kushner\textsuperscript{1} documented that congenital ocular aberrant innervations are more common than previously described and exist in many varieties. Ocular “miswirings” typically involve the sixth nerve and the most recognized clinical entities are Duane syndrome and Marcus Gunn jaw-winking ptosis.

We believe we have now presented the first case of congenital bilateral trigemino-abducens synkinesis. Furthermore, ocular abduction producing ipsilateral masseter and pterygoid activity is contrary to pre-