ing most accurately described as a myopic peripapillary sinkhole.

Ronald L. Fellman, MD
Davinder S. Grover, MD, MPH

Author Affiliations: Glaucoma Associates of Texas, Dallas.
Correspondence: Dr Fellman, Glaucoma Associates of Texas, 10740 N Central Expressway, Ste 300, Dallas, TX 75231 (rfellman@glaucomaassociates.com).
Financial Disclosure: None reported.

1. Toranzo J, Cohen SY, Erginay A, Gaudric A. Peripapillary intrachoroidal cavi
2. Shimada N, Ohno-Matsui K, Nishimuta A, Tokoro T, Mochizuki M. Peripap-
4. Nakazawa M, Kurotaki J, Buike H. Longterm findings in peripapillary cres
5. Moryama M, Ohno-Matsui K, Hayashi K, et al. Topographic analyses of shape of eyes with pathologic myopia by high-resolution three-dimensional magn
6. Doshi A, Kreidl KO, Lombardi L, Sakamoto DK, Singh K. Nonprogressive glau-

Progression of an Acquired Vitelliform Lesion to a Full-Thickness Macular Hole Documented by Eye-Track ed Spectral-Domain Optical Coherence Tomography

Vitelliform lesions (VLs), classically seen in young patients in autosomal dominant Best disease, are also seen as acquired lesions in entities such as adult-onset foveomacular vitelliform dystrophy, cuticular drusen, and central serous chorioretinopathy. These lesions appear as round yellowish deposits of material exhibiting hyperautofluorescence with fundus autofluorescence imaging. Spectral-domain optical coherence to mography (SD-OCT) shows hyperreflective material in the subretinal space, often with focal thickening at the level of the retinal pigment epithelium. The natural course of these lesions is often a gradual reduction in lesion size with fragmentation and resorption of the vitelliform material and eventual photoreceptor disruption and atro-
phy with accompanying hypoautofluorescence. A few studies have shown the development of a macular hole (MH) in eyes with VLs due to Best disease or adult-onset foveomacular vitelliform dystrophy. Herein, we report the first case to our knowledge showing with eye-tracked SD-OCT the evolution of an acquired VL (AVL) to a full-thickness MH in the absence of vitreomacular traction or an epiretinal membrane.

Report of a Case. A 69-year-old man, with no significant ophthalmic or family history, reported progressive distortion in both eyes. Visual acuity was 20/50 OD and 20/40 OS. Slitlamp examination revealed mild cataracts. Funduscopic examination showed bilateral AVLs that were hyperautofluorescent on fundus autofluorescence imaging. A posterior vitreous detachment was noted. The SD-OCT findings confirmed the presence of vitelliform material in the subretinal space, without vitreomacular traction or an epiretinal membrane (Figure).

Over the following 11 months, the AVL in the right eye was noted to decrease in size, with new retinal pigment epithelial changes and progressive foveal atrophy noted on SD-OCT. Visual acuity was 20/60 at that time. Fifteen months following the initial visit, visual acuity was 20/80 OD and a full-thickness MH was noted (Figure).

Comment. First described in association with adult-onset foveomacular vitelliform dystrophy, AVLs have been the subject of several studies using multimodal imaging to define the location and morphology of these lesions. The material composing the VL has been shown to occupy the space between the external limiting membrane and retinal pigment epithelium and is believed to represent unphagocytized photoreceptor outer segments and pigmented cells laden with melanin and melanolipofuscin granules. The chronic separation of photoreceptors from the retinal pigment epithelium is the presumed mechanism for progressive outer retinal atrophy and the accompanying gradual decline in visual acuity. Formation of MHs has been reported in eyes with VLs and is thought to result from progressive retinal thinning rather than vitreofoveal traction related to posterior vitreous detachment formation, which accounts for the majority of MHs. With the enhanced resolution and eye-tracking ability of SD-OCT, the natural course of the AVL to foveal atrophy and ultimately to a full-thickness MH can be documented.

Naomi Goldberg, MD
K. Bailey Freund, MD

Author Affiliations: Vitreous Retina Macula Consultants of New York (Drs Goldberg and Freund) and Department of Ophthalmology, New York University Medical Center (Dr Freund), New York.

Correspondence: Dr Freund, Vitreous Retina Macula Consultants of New York, 460 Park Ave, Fifth Floor, New York, NY 10022 (kbfnyf@aol.com).

Financial Disclosure: None reported.

Funding/Support: This work was supported by the Macula Foundation, Inc, New York, New York.
Paraneoplastic Optic Neuropathy From Cutaneous Melanoma Detected by Positron Emission Tomographic and Computed Tomographic Scanning

Paraneoplastic optic neuropathy (PON) is a rare disorder reported most commonly with small cell lung carcinoma. Several reports have associated cutaneous melanoma with paraneoplastic retinopathy, but to our knowledge melanoma has not been previously associated with PON. We describe a unique case of PON associated with cutaneous melanoma metastatic to lymph nodes detected by whole-body positron emission tomographic (PET) and computed tomographic (CT) scanning in a patient with bilateral optic neuropathy, vitritis, and progressive vision loss.

Report of a Case. A 67-year-old woman had photopsias and scotomas in her right eye for 6 weeks. Her left eye was asymptomatic. Her ocular history was unremarkable and her medical history was notable only for removal of a cutaneous squamous cell carcinoma 2 years prior. Visual acuity was 20/30 OD and 20/20 OS, with a small afferent pupillary defect in the right eye. Color vision results, tested with Ishihara plates, were 3 of 13 correct with the right eye and 13 of 13 correct with the left eye. Slitlamp and dilated funduscopic examination findings were remarkable for vitritis (right > left) and venous sheathing in both eyes. Fluorescein angiography demonstrated perivascular and optic disc leakage (right > left) (Figure 1A). Visual field testing revealed diffuse constriction in the right eye (Figure 1C).

Findings were normal on the following: complete blood cell count, metabolic panel, erythrocyte sedimentation rate, chest radiography, and magnetic resonance imaging of the head and orbit. Serologies were negative for hepatitis, syphilis, Lyme disease, and toxoplasmosis, and polymerase chain reaction testing results with an anterior chamber biopsy specimen were negative for herpes simplex virus, cytomegalovirus, and varicella-zoster virus. Tuberculin purified protein derivative testing results were negative. Full-field electroretinogram findings were unremarkable. Despite initial improvement with oral corticosteroids, visual field loss continued.

Because of ongoing inflammation and worsening of visual fields in both eyes (Figure 1D), visual evoked response testing was performed; it demonstrated delayed latencies (125 milliseconds OD; 132 milliseconds OS). Findings on a paraneoplastic panel including collapsin response mediator protein 5 IgG were negative, but because of increased suspicion of a paraneoplastic syndrome, whole-body PET and CT scanning was performed. It demonstrated a 1.8 x 1.5-cm right inguinal lymph node (Figure 2). Excisional biopsy revealed melanoma, and repeated pathological analysis of the “squamous cell carcinoma” diagnosed previously revealed melanoma.

At her most recent visit, the patient’s visual acuity was 20/30 OD and 20/20 OS. She had inactive vitritis while...