FIGURE 1

TORPEDO MACULOPATHY

Watch for choroidal neovascularization associated with this generally benign condition.

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A 24-year-old man was referred by his optometrist because of a fundus lesion found in his right eye on routine examination. The patient was asymptomatic and had an otherwise normal examination. His medical and family history was negative.

Based on our clinical and imaging findings (Figures 1-3), we made a diagnosis of torpedo maculopathy (TM), a rare, asymptomatic congenital deformity of the retinal pigment epithelium (RPE) that is considered to be an incidental finding.[1] TM characteristically presents as a hypopigmented lesion temporal to the fovea with a narrow point directed toward the fovea, as seen in our case.

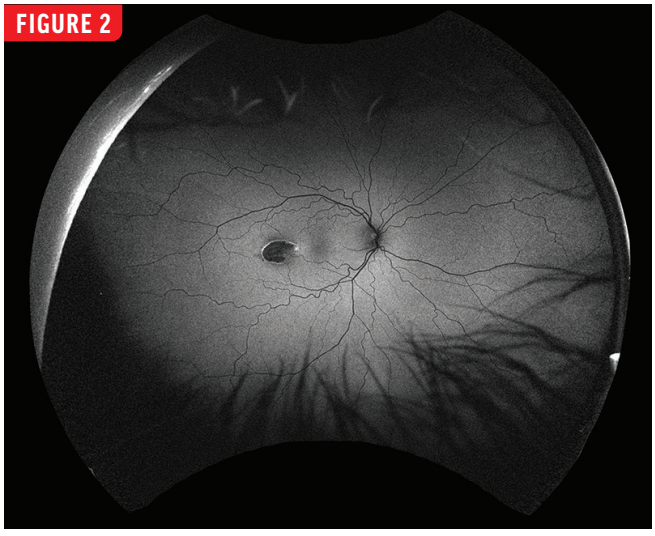
ABOUT TORPEDO MACULOPATHY

TM is typically unilateral but may rarely develop bilaterally. TM has a prevalence of two in 100,000 patients and is considered to be nonprogressive. Although most TM lesions are solitary in nature, satellite lesions can also exist in the same eye as the parent lesion. TM is underreported in the literature because of the asymptomatic nature of the condition. However, when found, it is important to differentiate TM from other pathologies that may require treatment. TM is classified into types based on subtle differences that can be detected on OCT[2]: Type 1 shows attenuation of the outer

(Continued on page 65)

(Continued from page 66)

FIGURE 2



retinal structures without outer retinal cavitation. Type 2 shows attenuation of the outer retinal structures with outer retinal cavitation, causing mild to moderate visual field defect.[3] A type 3 lesion is defined by excavated inner layers, retinal thinning, inner retinal hyperreflective spaces, and no subretinal cleft. Patient education must be performed when TM is suspected, as this condition, although benign, may progress into other pathologies, especially type 2 lesions.[2]

The etiology of TM is unclear but is thought to derive from the fetal temporal bulge at approximately 4 to 6 months of gestation. In type 2 TM, fluorescein angiography can be performed if choroidal neovascular membrane formation is suspected.

The differential diagnosis of TM includes other types of scars, both acquired and congenital, such as:

1) toxoplasmosis scars, which can be differentiated from TM based on the presence of full-thickness chorioretinal atrophy and non-uniform location, shape, and size, along with a known history of toxoplasmosis[2];

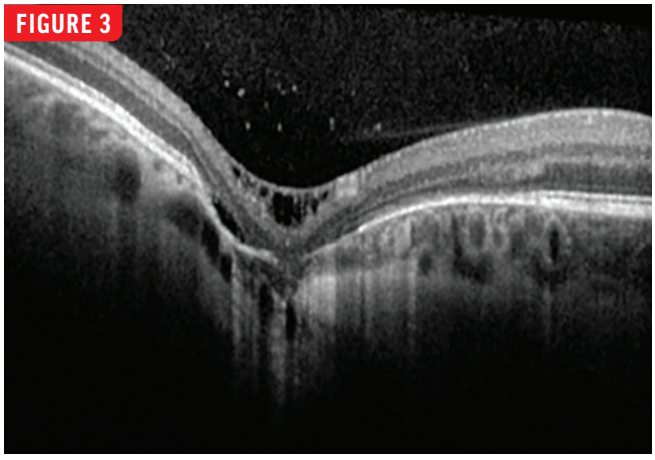
2) congenital hypertrophy of the RPE, which presents with pigmented lesions with well-defined borders typically found in the periphery of the retina and rarely near the macula, a differentiating factor from TM[2];

3) histoplasmosis, which typically presents with numerous scars, along with peripapillary scarring with choroidal neovascular membrane[2];

4) chorioretinal scars caused by blunt trauma to the orbit, which present with varying degrees of hyper- and hypopigmentation, are concentric to the optic nerve head, and are shaped in an elongated manner with a pattern in the choroidal rupture; and

5) amelanotic nevi, which are round and hypopigmented retinal lesions that typically lack the outer retinal attenuation and excavation seen in TM.[2]

FIGURE 3



KEEP AN EYE ON IT

TM should be monitored as part of a patient's annual eye examination, as there is risk of choroidal neovascularization growing through the scar. An at-home Amsler grid can also be used to supplement the in-office examination.[4] ■

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