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CLINICAL CASE

The Expanding Clinical Spectrum of Torpedo Maculopathy

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ABSTRACT

Purpose. Torpedo maculopathy is an idiopathic, congenital, oval-shaped region of chorioretinal hypopigmentation located temporal to the macula. Torpedo lesions are typically unilateral, occasionally harbor an intraretinal cleft, and may be associated with varying degrees of hyperpigmentation. Visual acuity is usually normal, but the lesion may produce a scotoma in the visual field. There are no known associated systemic or ocular abnormalities. Diagnosis is based upon recognition of its characteristic shape and location. Because of its nonprogressive and generally benign nature, no treatment is required.

Case Reports. Two cases of torpedo maculopathy associated with fundus excavation are presented. To the best of our knowledge, this is the first reported association of torpedo maculopathy with fundus excavation. In one case, the visual acuity remained unaffected and in the other case the visual acuity was reduced to 20/50. In both cases, optical coherence tomography clearly demonstrates the excavated nature of the torpedo lesions. In case 1, where the visual acuity was normal, the excavation is remote from the fovea but in case 2, where the visual acuity was 20/50, the excavation encroaches upon the fovea. In both cases, a scotoma corresponding to the excavated region could be demonstrated.

Conclusions. Torpedo maculopathy is a usually benign condition associated with normal visual acuity and normal visual fields. Our cases demonstrate that torpedo maculopathy may be associated with excavation of the fundus and a corresponding scotoma in the visual field. Visual acuity may be compromised should the excavation encroach upon the central fovea. Knowledge of this previously unreported clinical manifestation of torpedo maculopathy may aid in advancing the understanding of this condition and the care of patients with the disorder.

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Key Words: torpedo maculopathy, macular coloboma, macular dysplasia, optical coherence tomography, fundus autofluorescence

Torpedo maculopathy is an oval-shaped, congenital, chorioretinal lesion in the temporal macula.[1–3] The clinical features of this lesion include location temporal to the macula with its pointed edge facing the fovea. There are no known associated congenital, systemic, or ocular abnormalities. Its etiology is unknown. However, its uniform location in the temporal macula suggests it does not represent a defect in the closure of the fetal fissure.

The diagnosis of torpedo maculopathy is clinical and based upon recognizing the distinctive features of the condition (Table 1).[4] The essential features of torpedo maculopathy are a congenital, hypopigmented lesion located temporal to the fovea that is horizontally oval or spindle-shaped.[1] While visual acuity is usually normal, scotomas corresponding to the torpedo lesion are not uncommon.[1]

Herein, two cases of torpedo maculopathy associated with fundus excavation are presented. To our knowledge, this is the first reported association of torpedo maculopathy with fundus excavation. Proximity of fundus excavation to the fovea appears to be a risk factor for loss of visual acuity in torpedo maculopathy.

CASE 1

A 25-year-old South Asian female college student presents for a routine eye examination. Her last eye examination was 1 year ago with no ocular or visual complaints. The patient had no significant medical or ocular history and was not on any medication, and reported no known drug allergies. Patient had an unremarkable family ocular history but reported type 2 diabetes and hypertension on both sides of her immediate family.

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TABLE 1.
Clinical features of torpedo maculopathy

Constant features	Variable features
Congenital	Frayed tail temporal margin
Located temporal to the fovea	Intraretinal cleft
Horizontally oval shape	Fundus excavation
Hypopigmentation	Presence of hyperpigmentation
	Visual field defect

Patient's best-corrected visual acuities were 20/15 in both right and left eyes. Pupil and ocular motility testing were normal. Goldmann tonometry readings were 14 mm Hg OD and 13 mm Hg OS. Biomicroscopic examination was unremarkable OU. Ophthalmoscopy examination through dilated pupils revealed clear media in each eye. The fundus of each eye was normal with the exception of a torpedo-shaped, excavated lesion temporal to the fovea in the right eye (Fig. 1).

The macular region of each eye was examined using spectral-domain optical coherence tomography (OCT) (Fig. 2). An OCT scan through the lesion in the right eye revealed minimal disorganization of the retinal pigment epithelium (RPE) and outer retinal layers. Excavation of the temporal area of the torpedo lesion is seen with associated cystic degeneration of the inner layers of the retina in this region. Short wavelength fundus autofluorescence (SW-FAF) imaging of the lesion revealed a mild signal loss of the nasal portion the lesion near the fovea and more extensive loss temporally (Fig. 3). The central 10 degrees of visual field were examined using standard automated perimetry (Fig. 4). No defects were found in either eye. However, a dense scotoma within the deeply excavated temporal portion of the lesion was documented using microperimetry (Fig. 5).

A diagnosis of torpedo maculopathy OD was made, and the patient was educated regarding our findings. An annual examination was recommended for monitoring of her condition.

CASE 2

A 22-year-old African American female college student presented to our clinic for a routine eye examination. She reported that the vision of her right eye has always been worse than her left eye for unknown cause. She has a medical history of lupus erythematosus and anemia. Her medication list consists of hydroxychloroquine, prednisone, methotrexate, metronidazole, and oral contraceptives. She had an unremarkable family ocular and medical history.

Her best-corrected visual acuities were 20/50 and 20/20 in the right and left eyes, respectively. Pupil and ocular motility testing were normal. Goldmann tonometry readings were 12 mm Hg OD, 14 mm Hg OS. Biomicroscopic examination was normal OU. Ophthalmoscopy through dilated pupils revealed clear media in each eye. The fundus of each eye was normal with the exception of a torpedo-shaped, excavated lesion temporal to the fovea in the right eye (Fig. 6).

The macular region of each eye was examined using spectral-domain OCT (Fig. 7). There was no evidence of bull's eye maculopathy in either eye. An OCT scan through the lesion in the right eye revealed disorganization of the RPE and outer retinal layers. Retinal atrophy was present and greatest in those regions of the lesion that were most steeply excavated. SW-FAF imaging of the lesion revealed severe signal attenuation within the lesion and a thin ring of hyperflourescence at the lesion margins (Fig. 8). The central 10 degrees of visual field were examined using standard automated perimetry (Fig. 9). A scotoma was uncovered in the right visual field that corresponded to the paramacular lesion in this eye. The visual field of the left eye was within normal limits.

A diagnosis of torpedo maculopathy OD was made, and the patient was educated regarding our findings. She was informed that the long-standing history of reduced vision in the right eye was consistent with our findings, and annual observation was recommended.

DISCUSSION

Torpedo maculopathy is a congenital, typically unilateral, torpedo-shaped, hypopigmented lesion in the temporal macula and has been referred to as a "paramacular coloboma".[2] Numerous theories have

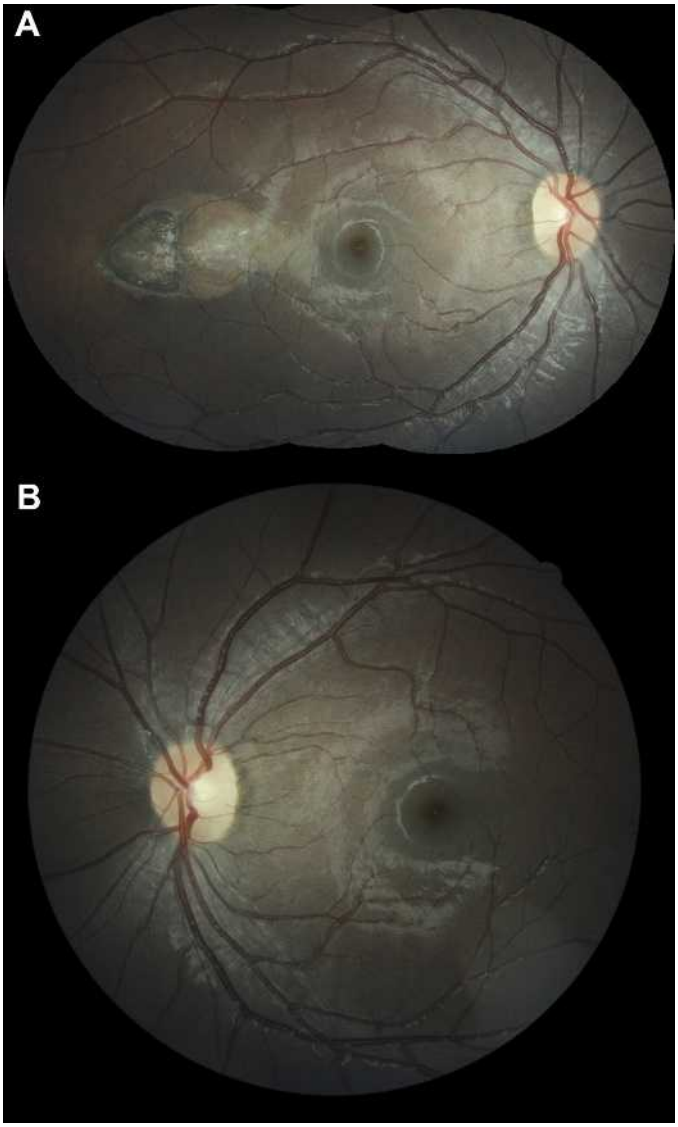

FIGURE 1.
Color photographs of case 1. A, Composite photograph of right eye with a torpedo lesion located temporal to the macula. Its nasal edge is hypopigmented, flat, and pointed towards the fovea. Temporally, it is excavated and contains a triangular region of hyperpigmentation. The retinal vessels appear normal as they cross over the surface of the lesion. B, Normal left eye.

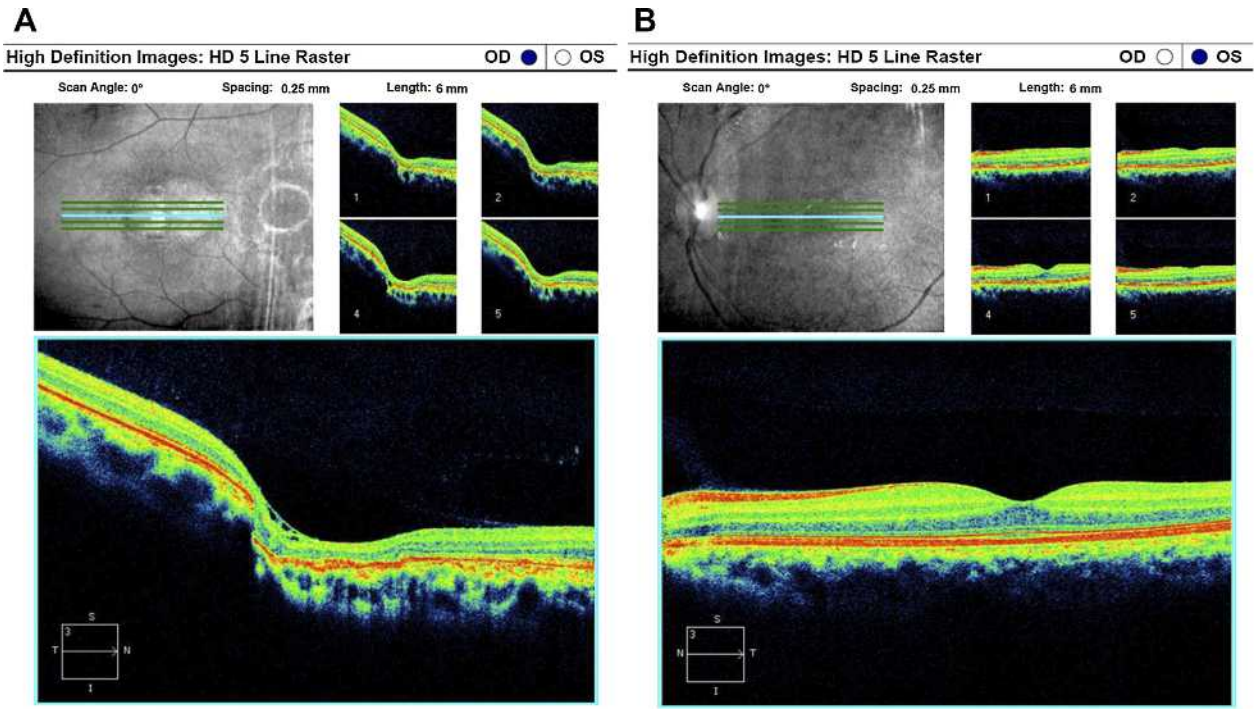

FIGURE 2.

Spectral domain optical coherence tomography (SD-OCT) of case 1. A, Raster scans through the torpedo lesion of the right eye reveals minimal disorganization of the retinal layers throughout most of the lesion. Retinal atrophy, degeneration, and excavation are most prominent temporally. B, Raster scans though a normal macula of the left eye.

been proposed regarding the pathogenesis of the lesion. Pian suggested it may a developmental defect in the nerve-fiber layer at the horizontal raphe.[2] Shields proposed that a defect in the development of the RPE within the fetal temporal bulge is responsible for the lesion.[5] Golchet et al suggested that the lesion may be related to malformation of the emissary canal of the long posterior ciliary artery and nerve.[1] It is possible that each of these mechanisms may be responsible for some torpedo lesions. Because the diagnosis of torpedo maculopathy is clinical, based primarily upon lesion appearance, we should keep in mind that a number of different and distinct disease conditions may produce similar-appearing fundus lesions. It is possible that torpedo-like lesions may occur as a consequence of a number of unrelated disease conditions. Similarly, it is conceivable that the appearance of torpedo lesions may vary depending upon any number of factors, including age, refractive error, and genotype. Review of the literature finds that reports of torpedo maculopathy lesions do, in fact, vary significantly in many of their clinical characteristics. A summary of fixed and variable characteristics of torpedo maculopathy is presented in Table 1.

Two essential features of torpedo lesions are their characteristic torpedo shape and their nonrandom location in the temporal macula. As the name implies, torpedo lesions have a characteristic oval shape which can resemble a torpedo. Typically, the nasal margin of the lesion is a sharply pointed ''head'' which is directed toward the fovea. Two different configurations of the temporal margin have been described—a rounded or frayed tail.[5] The temporal margin of the frayed tail variant is less sharply defined and may be composed of hypopigmentation and either linear or dotted hyperpigmentation. The rounded margin variant has a smooth, well-defined temporal margin that is composed of either linear, rounded, or no hyperpigmentation. In both configurations, the temporal margin of the torpedo lesion is typically positioned adjacent to the horizontal raphe in the temporal macula. Both of our cases had rounded tails.

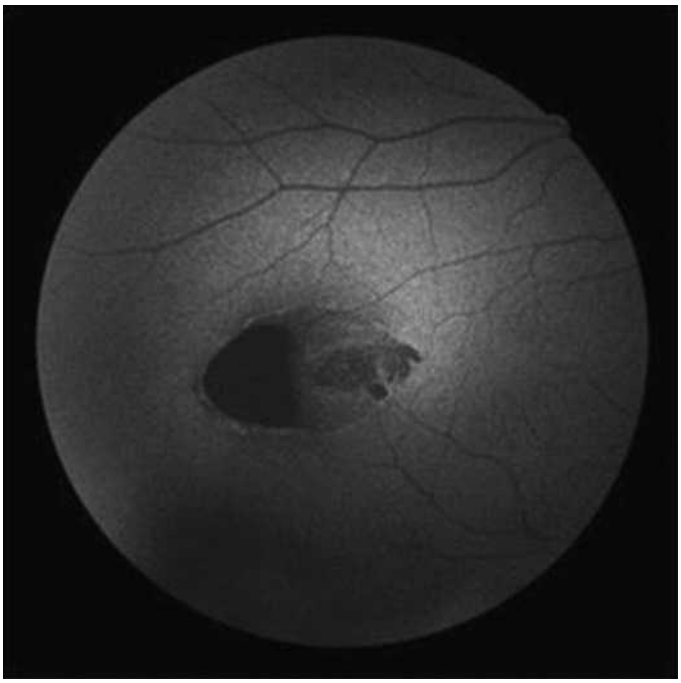

FIGURE 3.

Fundus autofluorescence of torpedo lesion in the right eye of case 1. A mild attenuation of signal intensity is seen in the nasal portion of the lesion and severe attenuation is present temporally. The loss of autofluorescence signal temporally falls within the triangular region of hyperpigmentation noted ophthalmoscopically.

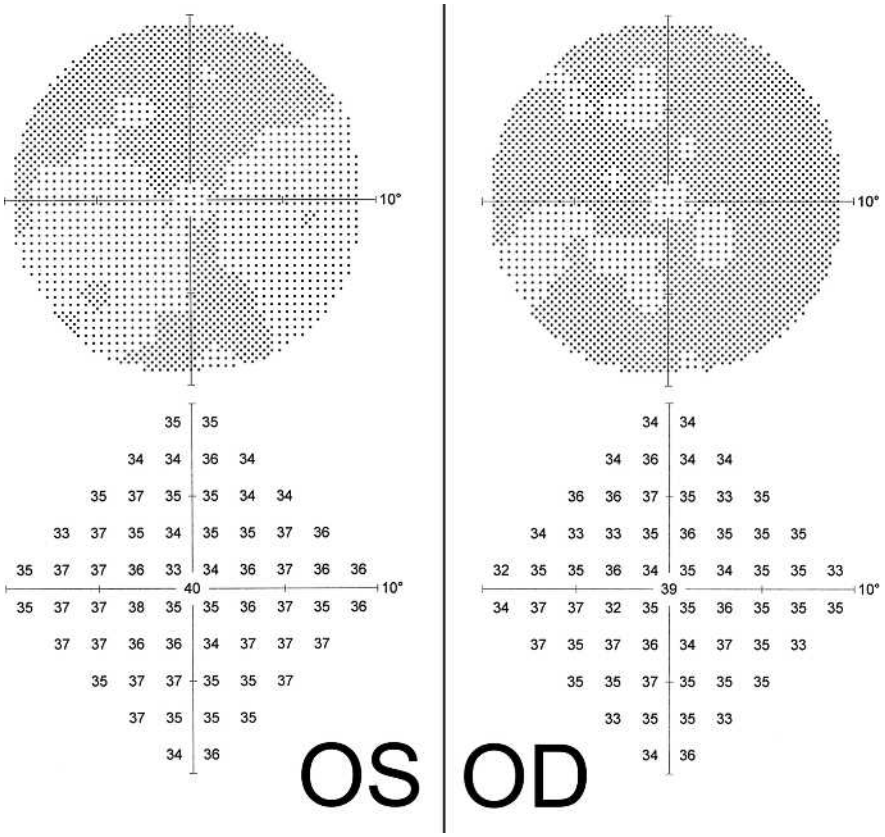

FIGURE 4.

Standard automated perimetry of case 1 using the Oculus 10-2 central visual field pattern. Grayscale (top) and threshold values (bottom) are displayed for each eye. No scotomas or defects were present in either eye.

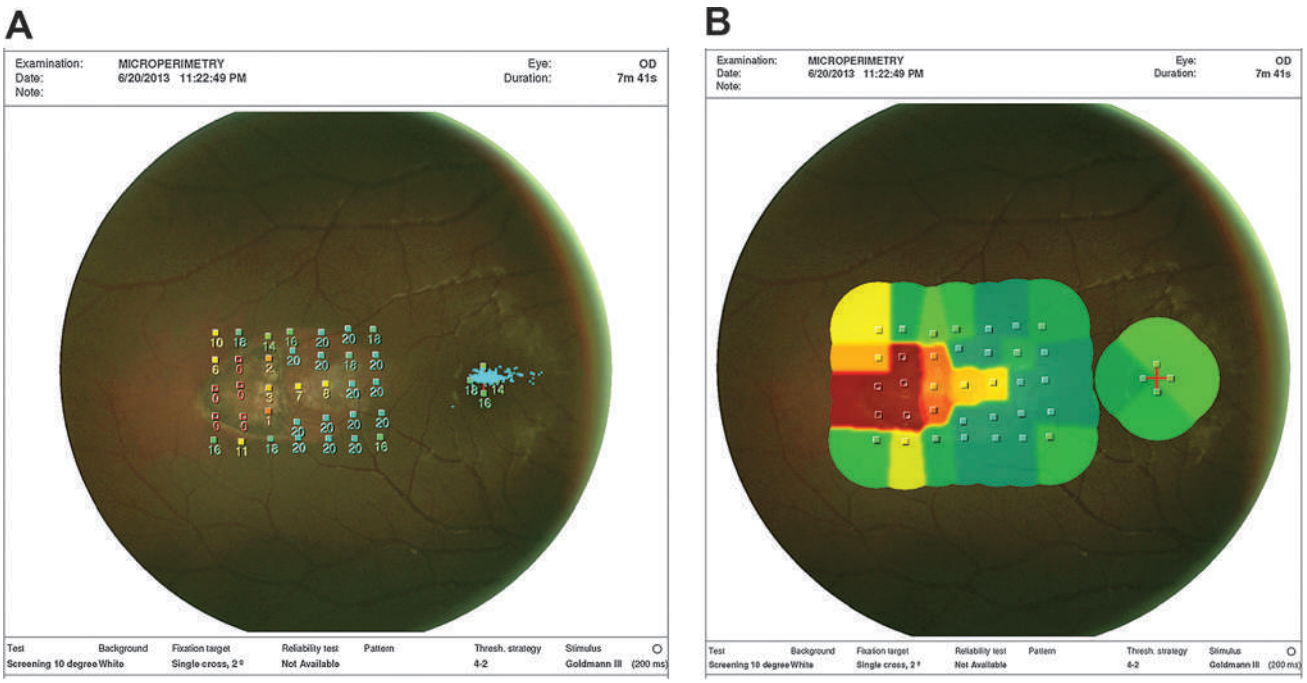

FIGURE 5.

Microperimetry of case 1 using the Nidek MP1 microperimeter. A test pattern was placed over the lesion to determine if there was any loss of retinal sensitivity associated with the torpedo lesion. The microperimeter numeric output map (A) and interpolated color map (B) reveal a dense scotoma corresponding to the floor of the excavation, with a region of moderate depression extending temporally within the torpedo lesion, reaching normal sensitivity levels at the nasal margin of the lesion.

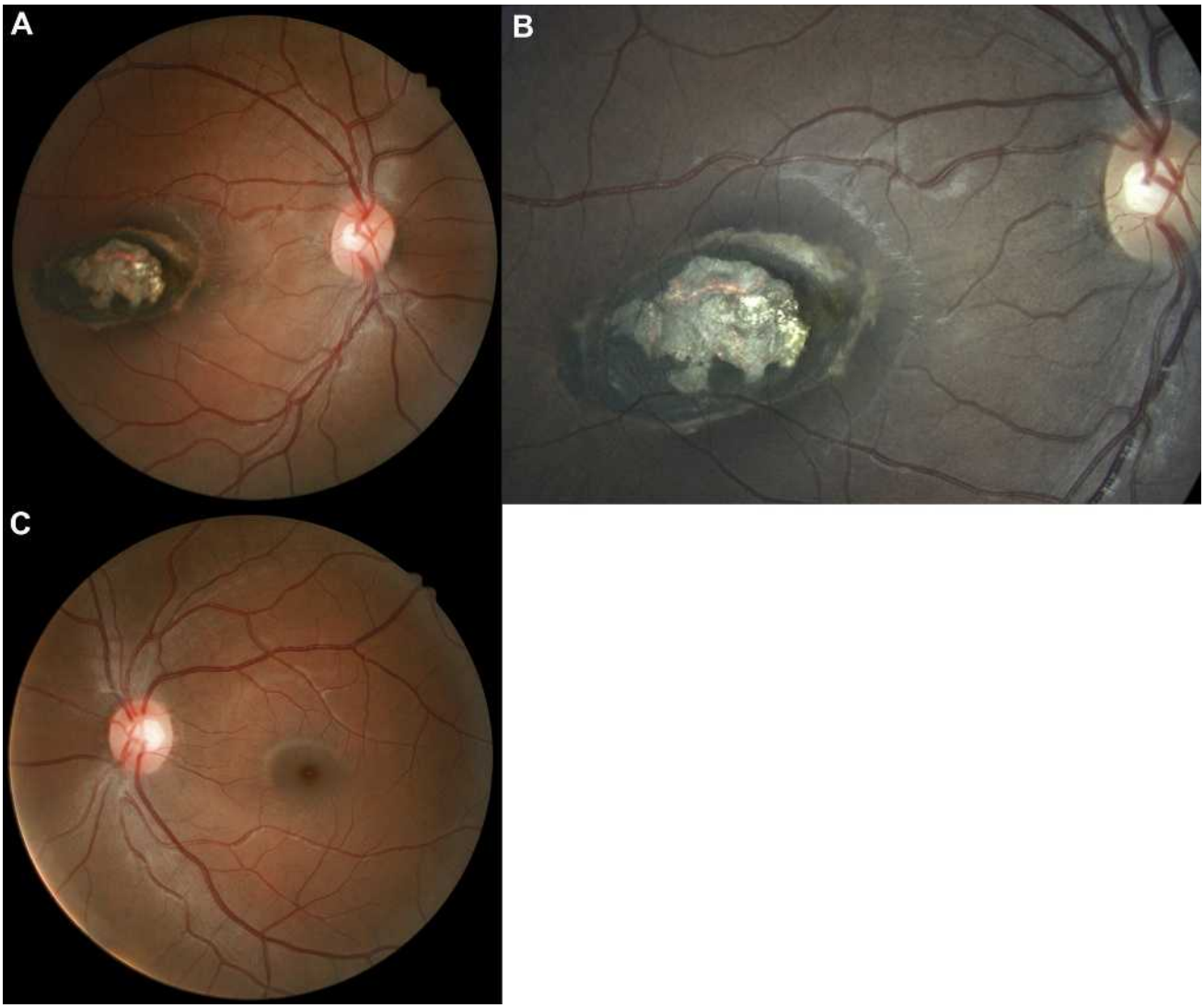

FIGURE 6.

Color photographs of case 2. A, Color photograph of the right eye with a torpedo lesion located temporal to the macula. There is a ring of dense hyperpigmentation surrounding a hypopigmented central area. It is possible to visualize few large choroidal vessels in the central region. The retinal vessels appear normal as they pass over the surface of the lesion. B, Magnified view of torpedo lesion. C, Normal left eye.

Torpedo lesions tend to not involve the central fovea, and the visual acuity is usually normal.[1,2,6,7] Lesions are usually about 1 to 2 disc diameters in horizontal length and 0.5 to 1.0 disc diameters in vertical height.[5,6] Although typically unilateral, bilateral presentations have been reported.[8,9] The nasal edge of the torpedo lesion in patient 1 gradually fades away about 1 disc diameter temporal to the center of the fovea, whereas in patient 2 the lesion encroaches upon the central fovea. A review of published reports finds that the nasal edge of torpedo lesions typically lie within 1 disc diameter of the foveal center. Golchet and colleagues reports that all of their 13 cases extended up to the fovea but did not involve the foveal center.[1] The lesions were located adjacent to or less than 1 disc diameter temporal to the fovea in the three cases reported by Pian.[2]

When visual acuity is affected in eyes with torpedo maculopathy, it is usually attributable to unrelated comorbidities, such as retinal vascular occlusions or glaucoma.[1] However, mild to moderate loss of visual acuity secondary to macular involvement of lesions resembling those that occur in torpedo maculopathy have been reported.[10,11] Patient 2 in this report had moderate (20/50) vision loss attributable to the presence of a torpedo lesion and patient 1 had normal (20/15) visual acuity in the affected eye. It has been suggested that torpedo lesions may evolve over time such that their morphology and impact on visual function may vary with age[12]; however, to date no longitudinal studies of torpedo maculopathy have been reported and there is currently no evidence that such changes do, in fact, occur. The longest reported follow-up of a patient with torpedo maculopathy is 2 years, and no changes were detected over that time interval.[1]

Published reports reveal that visual field loss is frequently associated with torpedo maculopathy. Standard automated perimetry will usually reveal a paracentral scotoma that corresponds to the location of the lesion.[1,13–15] However, visual field screening with Amsler grid[2,3,6,7] or other methods[2] may fail to detect the presence of a scotoma. Microperimetry makes possible precise correlation of retinal sensitivity to fundus characteristics and permits the detection of microscotoma within the central visual field that may not be detectable using standard perimetry methods.[16] In this case series,

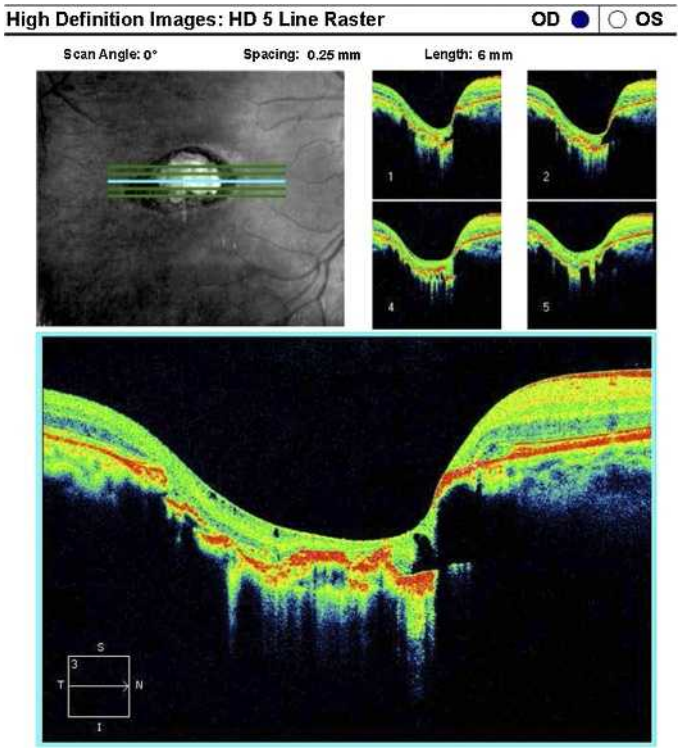

FIGURE 7.

Spectral-domain optical coherence tomography (SD-OCT) of torpedo lesion in the right eye of case 2. The lesion is deeply excavated with disorganization and degeneration of the retinal layers within the lesion. There is no clearly identifiable fovea.

patient 2 had a scotoma corresponding to her torpedo lesion that was readily detected using standard automated perimetry. However, we were unable to detect any visual field loss in patient 1 without the assistance of microperimetry. This may have been a result of the small size of the scotoma.

Optical coherence tomography studies of torpedo lesions find that some[1,13,17] but not all[6,12,14] lesions contain a shallow cleft, possibly created by the loss of photoreceptors or the RPE within the borders of the lesion. While this has been described as a serous detachment of the neurosensory retina,[13] Golchet et al states that the lack of overlying retinal abnormalities, such as intraretinal edema on OCT, and the absence of leakage of fluorescein on fluorescein angiography are evidence against accumulation of subretinal fluid in this space.[1] Another inconsistent OCT feature of torpedo lesions is thinning and disorganization of the inner and outer retinal layers,[6,14] which is minimal[14] or absent[12] in some patients. The RPE OCT signal has been reported as appearing thin[12] in some cases and hyperreflective[6,14] in others. Both of our cases demonstrated some degree of disorganization of the RPE and outer retinal layers on OCT that was greatest in the most deeply excavated region of the lesions.

Hyperpigmentation is usually minimal and when present is largely confined to the temporal edge of the lesion.[5] Geographic regions of hyperpigmentation within the central hypopigmented region of the torpedo lesion is not uncommon.[17] A less typical presentation is the occurrence of extensive hyperpigmentation of the lesion margins.[8] Such lesions, if also excavated, would fulfill Mann's criteria for a type 2 macular coloboma.[18] Therefore, a lesion such as that found in our patient 2 would appear to straddle the diagnostic criteria for both macular coloboma and torpedo maculopathy. This leads us to endorse the classification of torpedo maculopathy as a paramacular coloboma.[2]

Excavation has not previously been reported as a finding of torpedo lesions. However, both of our patients have lesions that would appear to be typical examples of torpedo maculopathy, but are distinctly excavated. This leads us to believe that the definition of torpedo maculopathy should be inclusive of both flat and excavated lesions that otherwise fulfill the usual diagnostic criteria of the condition. In the largest series reported to date, Golchet and colleagues found that each of their 13 cases of torpedo maculopathy consisted of a flat hypopigmented lesion.[1] Selection bias may have influenced the results of this retrospective record review if torpedo maculopathy was defined as a flat hypopigmented lesion a priori. Reports of torpedo-shaped hypopigmented but excavated lesions in the temporal macular area are frequently labeled as macular colobomas rather than torpedo maculopathy.[9,10] There is considerable overlap between the clinical features of the two conditions. Similarly, congenital toxoplasmosis scars are also frequently excavated[19] and can occasionally resemble torpedo lesions. The differential diagnosis of torpedo maculopathy is summarized in Table 2.

Autofluorescence imaging of the ocular fundus involves stimulation of naturally occurring fluorophores within the retina, the most significant being lipofuscin, and recording the light they emit.[20] The quantity of light emitted is related to the quantity of lipofuscin present within the RPE. FAF imaging may therefore provide valuable insights into the functional status of the retina. For example, nonfunctional RPE may result in the absence of autofluorescence.[21] FAF imaging is performed using short

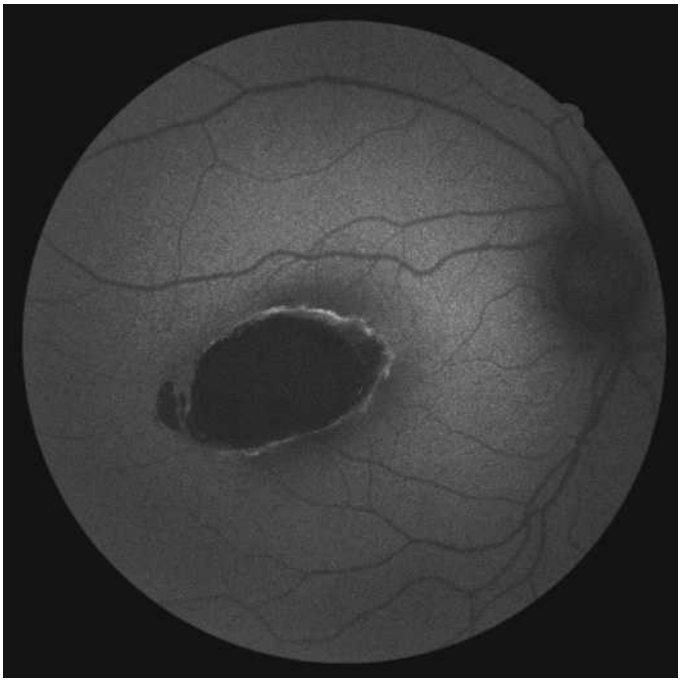

FIGURE 8.

Fundus autofluorescence of torpedo lesion in the right eye of case 2. There is severe loss of autofluorescence signal throughout the entire lesion. A narrow ring of hyperfluorescence extends around the margins of the lesion except temporally, where the boundary of the lesion appears less discrete.

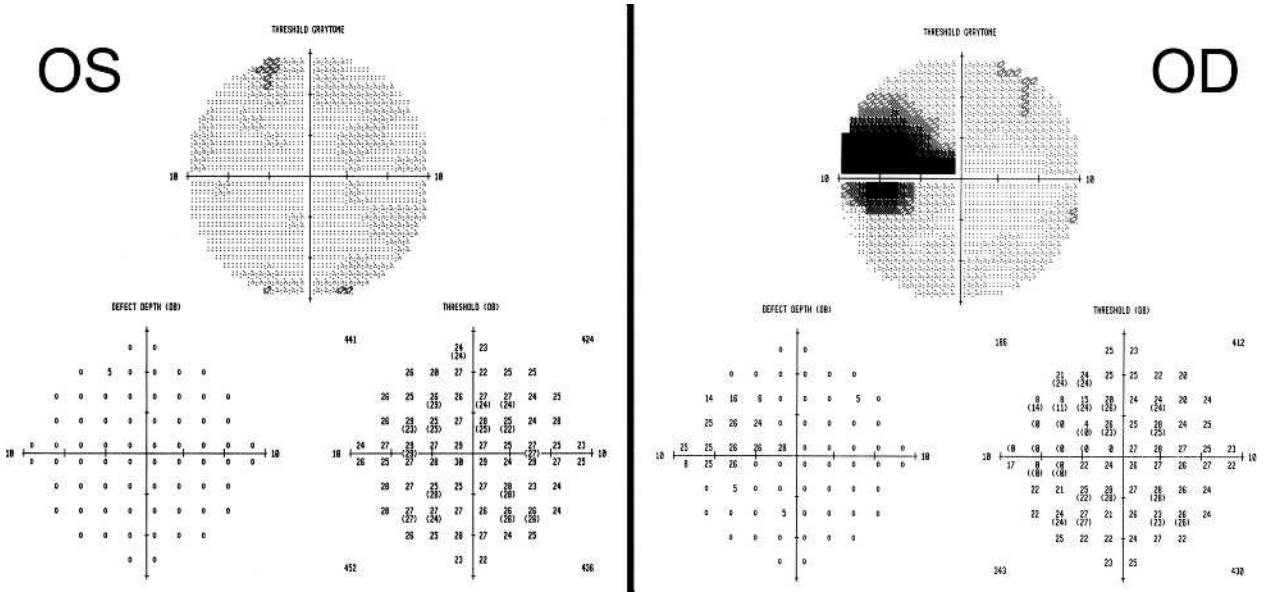

FIGURE 9.

The central 10-2 threshold test performed on the Humphrey automated perimeter. There is a dense scotoma nasal to fixation for the right eye, corresponding to the location of the torpedo lesion. The visual field is normal for the patient's left eye.

wavelength blue light to stimulate lipofuscin, but other wavelengths of light may be used to target other fluorphores.[20] Patient 1 in the current report reveals mild FAF signal attenuation in the nasal half of the lesion with signal absence in the more heavily pigmented and steeply excavated temporal portion of the lesion. In contrast, FAF imaging of patient 2 reveals absence of signal throughout most of the lesion (except for a small region near its temporal margin), and a zone of increased FAF intensity surrounding the nasal, superior, and inferior margins of the lesion. This would suggest that there is severe loss of RPE function within the torpedo lesion of patient 2 and severe loss of RPE function only within the steeply excavated temporal portion of the torpedo lesion in patient 1. This is consistent with the observed loss of visual function that was found in patient 2 but not in patient 1.

TABLE 2.

Differential diagnosis of torpedo maculopathy

Congenital lesions of the retinal pigmented epithelium
• Congenital toxoplasmosis[19]
• Congenital hypertrophy of the retinal pigmented epithelium
• Gardner syndrome[23]
Focal retinal pigmentation from external agents
• Trauma
• Drug-induced toxic effects
Choroidal lesions
• Choroidal melanoma
• Choroidal nevus
• Metastatic carcinoma
Serous detachments of the sensory retina
• Central serous chorioretinopathy[24]
• Polypoidal choroidal vasculopathy[25]
Macular and paramacular excavations
• Colobomatous defects[26]
• Macular dysplasia[27]
• Posterior staphyloma[28]

Reports of FAF imaging in patients with torpedo maculopathy have been mixed. Barbazatto et al[15] reported that severe FAF signal attenuation occurred within the torpedo lesion of their patient. Golchet et al[1] reported the FAF findings of a single patient in their series of 13 cases. This patient also exhibited severe FAF signal attenuation within the torpedo lesion. Interestingly, Pilotto et al[12] reported essentially normal FAF findings in their patient. Because their patient was a 4-year-old child, they suggested that any age-related changes that may affect the lesion may not have yet manifested. Another contributing factor may be that the torpedo lesion in this child did not contain a cleft, but a cleft was present in the patients reported on by Barbazatto et al and Golchet et al.

The differential diagnosis of torpedo maculopathy should include congenital and acquired lesions of the RPE, choroidal lesions that may masquerade as hypopigmented lesions of the RPE, conditions that may produce a serous detachment of the neurosensory retina in those torpedo lesions containing a cleft, and those conditions associated with macular or paramacular excavation in those torpedo lesions that are excavated. The differential diagnosis of torpedo maculopathy is summarized in Table 2. Among congenital lesions of the RPE, congenital toxoplasmosis scars are perhaps the most challenging differential consideration because it presents as a nonspecific chorioretinal scar that can closely mimic a torpedo lesion. Serologic testing can establish whether prior exposure to *Toxoplasma gondii* has occurred. Because serologic testing was not conducted in either of our patients, we cannot definitively rule out toxoplasmosis as a cause for these lesions. However, in neither patient did the lesion resemble a typical toxoplasmosis scar. For example, the degree of retinal disorganization found on OCT in our patients was much less severe than occurs in comparable toxoplasmosis scars.[19] Another important differential consideration is plaquenil maculopathy in patient 2. This patient presented to our clinic specifically for a hydroxychloroquine toxicity screening examination. Based upon current recommendations,[22] OCT and FAF

are both effective for detecting early signs of retinal toxicity from hydroxychloroquine. Our patient did not manifest any signs of plaquenil maculopathy.

CONCLUSIONS

Torpedo maculopathy lesions may vary significantly in their clinical presentation (Table 1). The essential characteristics that define the condition are (1) congenital onset, (2) horizontal oval or spindle shape, (3) hypopigmentation, and (4) location temporal to the fovea. Beyond those four key characteristics, there is significant variability. This may reflect differences in underlying etiology, lesion evolution over time, or differences in phenotypic expression.

Most torpedo lesions are asymptomatic because the lesion remains outside the central fovea and the retina within the lesion remains at least partially functional. Other lesions are associated with dense scotomas and mild to moderate visual acuity loss. Our cases suggest that greater proximity of the lesion to the foveola, lesion excavation within the central fovea, and more extensive loss of RPE function within the lesion by FAF are factors associated with vision loss secondary to torpedo maculopathy.

While some authors continue to describe torpedo lesions as being "flat",[12] this is clearly not always the case. The presence of an intraretinal cleft or neurosensory retinal detachment may produce a shallow elevation of the lesion.[13] These cases provide the first documentation, to our knowledge, that some torpedo lesions may be excavated. Lesion excavation would be consistent with several proposed etiologies for torpedo maculopathy, including defect of the fetal temporal bulge,[5] and malformation of the emissary canal of the long posterior ciliary artery and nerve.[1]

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