

Aaron Miller

Accelerated Scholars Class of 2025

Hometown: Coal Township, Pennsylvania

Undergrad: Loyola University Maryland

Major: Biology

Favorite Animal: Red Panda

Optometry Goal: To work with and help people

Favorite instrument: Piano

Hobby: Baking

Last Show I binged: Masterchef



Stephanie Holt

Class of 2004, Pennsylvania College of Optometry

Hometown: Asheville, North Carolina

Undergrad: United States Air Force Academy

Major: Behavioral Science, Human Factors Engineering

Last show I Binged: All the Light We Cannot See

On My Bucket list: Wine tasting and sight-seeing in Tuscany or Friuli-Venezia with family and friends

Current Annual Hobby: Men's & Women's March Madness Brackets; next up Stanley Cup Brackets!

Darn Torpedoes!

Demographics

48 yo Black male, new patient

Chief complaint: Annual diabetic eye exam (DM Type 2 dx 2021); blurry vision at near OU and reports he was told at a previous eye exam that he has a "scar behind his eye" of unknown etiology and he is unsure which eye

History of present illness

Character/signs/symptoms: blur at near

Location: OU

Severity: moderate

Nature of onset: worse since losing PALs approximately 1 year ago

Duration: Frequency: constant with near tasks

Exacerbations/remissions: None

Relationship to activity or function: reading and other near tasks

Accompanying signs/symptoms: denies diplopia, LOV, headaches, flashes, floaters

Patient ocular history: "scar behind his eye" and unsure which eye, (-)eye injuries/trauma, (-) glaucoma, (-)diabetic retinopathy

Family ocular history: (-) Glaucoma, (-)Blindness

Patient medical history: T2 Diabetes Mellitus, Asthma, Hypercholesterolemia, Hypertension

Medications taken by patient: Albuterol sulfate HFA inhaler, Atorvastatin, Drysol Dab-O-Matic 20% topical solution, Toujeo Max U-300 SoloStar insulin, Trelegy Ellipta inhaler, Valsartan, Xigduo XR

Patient allergy history: Allergic to shellfish

Family medical history

Father: Hypertension (-) glaucoma (-) blindness

Review of systems

Constitutional/general health: denies

Ear/nose/throat: Cardiovascular: denies

Pulmonary: denies

Endocrine: Asthma

Dermatological: denies

Gastrointestinal: denies

Genitourinary: denies

Musculoskeletal: denies

Neurologic: denies

Psychiatric: denies

Immunologic: denies

Hematologic: denies

Mental status

Orientation: oriented to person, place, and time

Mood/Affect: normal

Clinical findings

BVA:(sc)	<u>Distance</u>	<u>Near</u>
OD:	20/40 PH:20/20	0.4/0.6M
OS:	20/40+2 PH: 20/20	0.4/0.6M

Pupils: PERRL OU (-) APD

EOMs: restricted abduction 75% OU, 100% otherwise OU; (-)diplopia

Confrontation fields: FTFC OU

Hirschberg/CT: ortho @ Dist, 4XP @ Near

Subjective refraction:	<u>VA Distance</u>	<u>VA Near</u>
OD: -0.50 -0.75 x 120 +2.25	20/20	0.4/0.4M
OS: -0.50 -1.00 x 076 +2.25	20/20-2	0.4/0.4M

Slit lamp:

lids/lashes/adnexa: normal lashes and adnexa OU; trace inspissation superiorly OU

conjunctiva: T+N pinguecula OU, saponification in lateral canthus OU, palpebral conjunctiva pink and quiet OD and a retention cyst inferiorly OS

Cornea: normal endothelium, epithelium and stroma OU

anterior chamber: deep and quiet OU; VH 4 T/N OU

Iris: flat and intact, (-) NVI OU

lens: clear lens capsule, cortex, and nucleus OU

Vitreous: vitreal syneresis OU

IOPs/method: 11/10 mmHg OD/OS Goldmann Tonometry

Fundus OD:

C/D, ONH: 0.45/0.45, pink with good rim; temporal RPE crescent, myelination inferior nasal rim margin, (-) NVD

macula: flat oval lesion starting temporal to the fovea with distinct borders and central RPE hyperplasia with hypopigment surround, longer horizontally than vertically; (-) edema; (+) foveal reflex

posterior pole: see image 1

periphery: flat x 360 degrees, no RD, no holes, (-) NVE

Fundus OS:

C/D, ONH: 0.4/0.4, pink with good rim, temporal RPE crescent, myelination inf rim margin, (-) NVD

macula: flat, no hemorrhages, CWS, exudates, pigmentary changes, or no macular edema, (+) foveal reflex

posterior pole: see image 1

periphery: flat x 360 degrees, no RD, no holes; lattice degeneration inferiorly without holes, (-) NVE

Blood pressure: 132/98 mmHg right arm, sitting by manual cuff

reports that he had not taken his medication yet; denies headaches, shortness of breath, chest pain, or numbness in extremities

Case Images:

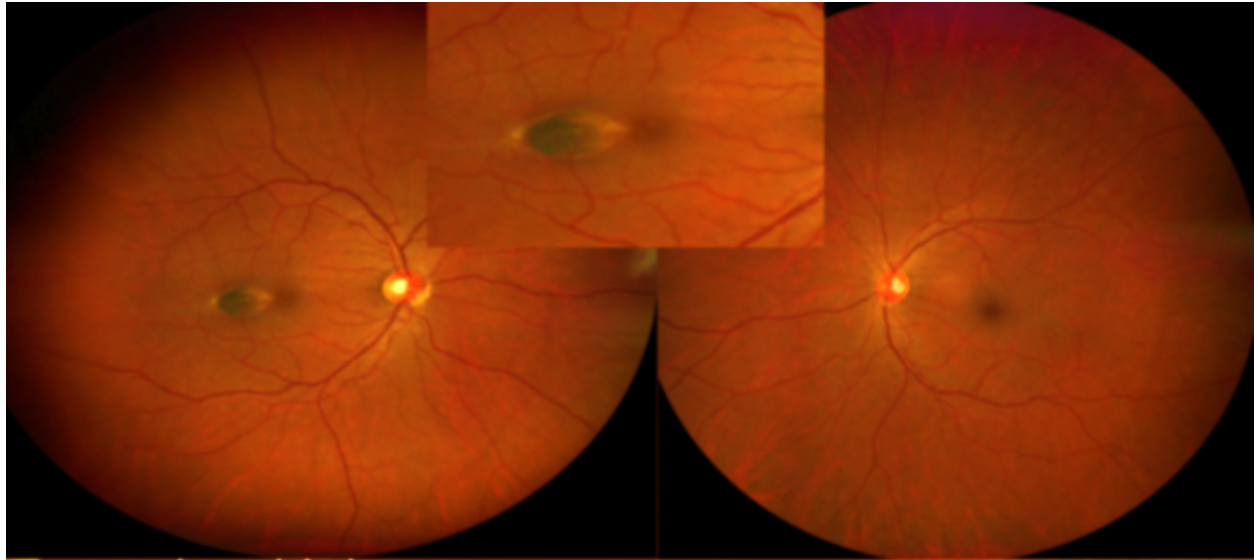


Image 1: Colored fundus photographs OD and OS, respectively, revealing a flat lesion temporal to the fovea OD consistent with torpedo maculopathy, slightly attenuated arterioles OU, and no diabetic retinopathy OU. (Note: Normal fundus behind enhanced image)

Case Management Summary - Initial comprehensive eye exam

Assessment 1: Type 2 Diabetes Mellitus without complications

- Dilated exam revealed no signs of diabetic retinopathy and no CSME/DME OU
- Patient reports good control with Insulin (Toujeo) and oral medication (Xigduo)
- Last A1c: 6.6%, Last FBS: 102 mg/dL

Plan 1: Patient educated on exam findings and importance of maintaining optimal control of blood glucose level. He was educated on the importance of yearly dilated eye exams. Letter sent to PCP. Patient to return for monitoring in 1 year.

Assessment 2: Dystrophy primarily involving the retinal pigment epithelium OD

- Dilation revealed flat oval lesion temporal to fovea resembling torpedo maculopathy
- Patient denied any history of ocular trauma OD
- Pt reports first learning about a "scar" behind one of his eyes about two years ago
- BCVA 20/20 OD, pt asymptomatic

Plan 2: Patient educated that retinal lesion appears consistent with torpedo maculopathy given the appearance and location and that this condition is congenital and typically remains stable. Patient was educated on the use of Amsler grid and home monitoring weekly and educated on the importance of returning urgently if any changes are noted. Patient referred to TEI Retina Service next available appointment for confirmation of diagnosis.

Assessment 3: Lattice degeneration OS without holes

- Patient is asymptomatic for floaters/flashes.

Plan 3: Patient educated on si/sx of retinal detachment including flashes/floaters/curtain over vision

and to return urgently if notice any of these symptoms. Monitor in 1 year and prn symptoms.

Assessment 4: Compound Myopic Astigmatism with Presbyopia OU

-BCVA: 20/20 OD/OS, 20/15 OU

Plan 4: Updated PAL Rx given. Pt ed on adaptation period to new Rx as he is a first-time PAL wearer. Monitor in 1 year and prn changes in vision.

Retinal exam at TEI 2 months later (pertinent history only)

Chief complaint: Macular lesion OD

History of present illness: denies any changes to vision including flashes/floaters/loss of vision OD

Patient Medical History: Patient reports no changes since comprehensive exam

Clinical findings:

BVA (sc): OD: 20/30, OS 20/50 (PH to 20/25)

Fundus OD: see Image 2 and 3

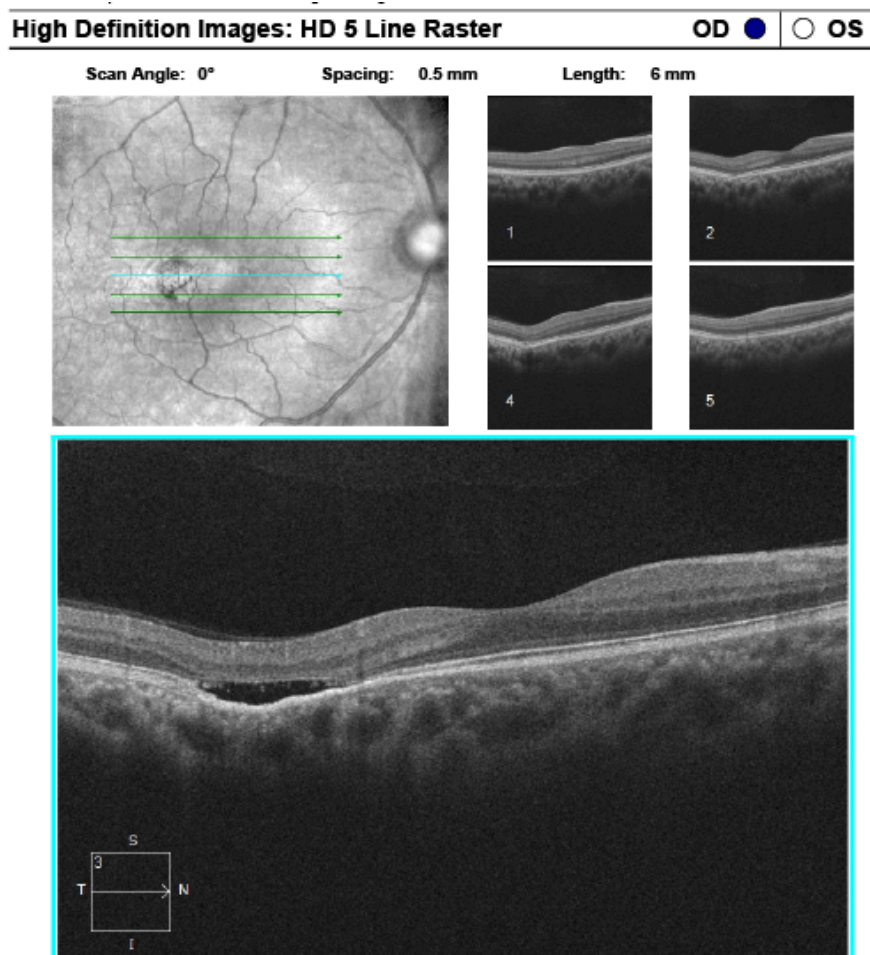


Image 2: High definition 5 line Raster OCT image (of the right eye) through the center of the macular lesion demonstrating outer retinal attenuation with concave retinal appearance and choroidal excavation consistent with Type 2 Torpedo Maculopathy

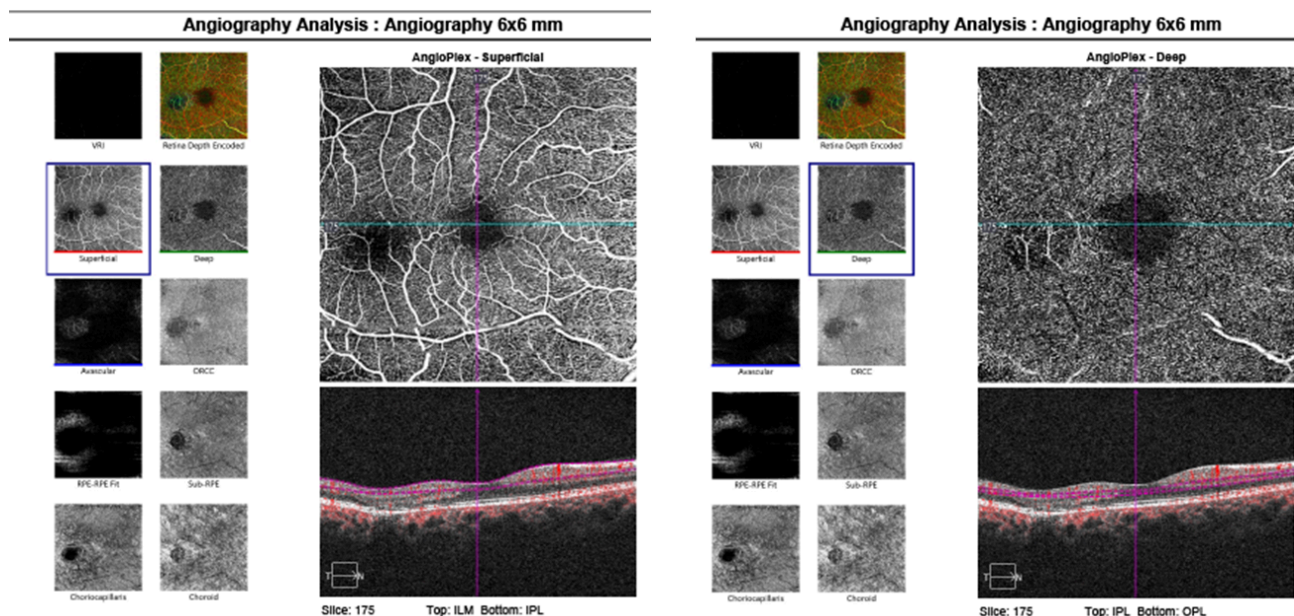


Image 3: Cirrus OCT-Angiography OD demonstrating hypofluorescence in the area of the lesion temporal to the fovea from the superficial view through the deep en-face cut. OCT-Angiography OS (not shown here) is unremarkable.

Case Management Summary - TEI Retina Service Exam

Assessment 1: Dystrophy primarily involving the retinal pigment epithelium, right eye

- Flat oval lesion, resembling torpedo maculopathy pattern
- Pt denies h/o ocular trauma.
- OCT indicates concave appearance, OCTA hyperfluorescent in area of lesion
- BCVA 20/20 OD, pt asymptomatic

Plan 1: Educated patient on findings. Additional testing indicated to rule out risk of vision loss. Patient to present to Philadelphia Retina Associates (PRA) at Nazareth hospital next available for IV fluorescein angiography.

*Patient was seen by Philadelphia Retina two months later. Due to stability of vision, fundus photos and OCT, IV fluorescein angiography was not performed. **Patient diagnosis of torpedo maculopathy of the right eye was confirmed with annual monitoring indicated. Patient was educated to continue home monitoring with Amsler grid and to return urgently if any changes are noted.***

Case Pearls

- Torpedo maculopathy is a rare, congenital condition that typically presents unilaterally as a hypopigmented, oval-shaped lesion in the temporal macula often with the tapered end of the oval pointing towards the fovea. The typical size of the lesion is normally 2 Disc Diameters by 1 Disc Diameter and patient vision is normally unaffected.
- Differential diagnoses include CHRPE, traumatic scar, amelanotic nevi, histoplasmosis

scar and toxoplasmosis scar. Thorough history, appearance, location, laterality, and OCT are important factors when differentiating these lesions.

- The exact etiology of torpedo maculopathy is unknown, but the most widely accepted explanation of etiology is that it occurs during a specific period in fetal development at the site of the “fetal bulge” that forms in the temporal-macular retina area.⁴ It is postulated that when this area retracts, a mild depression remains and a disruption in the RPE cells occurs within the depression resulting in the torpedo-shaped lesion. Classically, the lesion is noted to have hypopigmented tissue nasally and RPE hyperplasia temporally.⁴
- OCT can be used to differentiate and further classify the diagnoses of torpedo maculopathy showing the presence or absence of retinal and/or choroidal excavation. There are three types of presentations that can occur: *Type 1* includes lesions that show attenuation of the interdigitation zone and ellipsoid zone without outer retinal cavitation; *Type 2* shows loss of ellipsoid and interdigitation zones as well as thinning of the outer nuclear layer associated with outer retinal cavitation and inner choroidal excavation may be present or absent; *Type 3* lesions have been suggested as an additional category defined by excavated inner layers, retinal thinning, inner retinal hyper-reflective spaces, and no subretinal cleft.
- Torpedo maculopathy is typically non-progressive; however, there are rare case reports of documented progression of the lesion and few case reports of choroidal neovascular membrane (CNVM) associated with the lesion.
- Primary care management includes yearly monitoring for changes, home monitoring with Amsler grid and patient education on the rare possibility of changes in their condition.

References

1. Rohl A, Vance S. Hyperpigmented Torpedo Maculopathy with Pseudo-Lacuna: A 5-Year Follow-Up. *Case Rep Ophthalmol*. 2016 May 26;7(1):184-90. doi: 10.1159/000445497. PMID: 27462244; PMCID: PMC4943775.
2. Chitturi SP, Venkatesh R, Handa A, Mangla R, Chhablani J. A rare case of hyperpigmented torpedo maculopathy. *European Journal of Ophthalmology*. 2023;0(0). doi:10.1177/11206721231182736
3. Flores-Moreno, et, al. American Academy of Ophthalmology. (2023, August 31). *Torpedo Maculopathy*. EyeWiki. https://eyewiki.aao.org/Torpedo_Maculopathy
4. Shields CL, Guzman JM, Shapiro MJ, et al. Torpedo maculopathy at the site of the fetal “bulge”. *Arch Ophthalmol* 2010 Apr;128(4):499-501