Salus at Drexel University
The Eye Institute

Pennsylvania College of Optometry

The Focal Point

August 2024 Edition

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Traditional Class of 2025

Hometown: Lumberton, North Carolina
Undergrad: University of North Carolina at Pembroke
Major: Biology
Favorite Animal: Cats
Optometry Goal: To graduate
Hobby: Coffee exploring
Last Show I binged: The Mole

Katie Harrison

Traditional Class of 2026

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Last Show I binged: The Cleaning Lady

Bhawan Minhas, OD, FAAO

Illinois College of Optometry 2013; Pennsylvania College of Optometry Residency in Primary Care and Ocular Disease 2014

Hometown: Calgary, Alberta Canada
Undergrad: University of Calgary
Major: Biological Sciences; Minor Primatology
The Most Expensive Thing I Own: my brain
Summer obsession: watermelon with lemon
Last Show I binged: Three Body Problem

The Story After Trauma: Optic Nerve Pallor and Enophthalmos

Some details of the case have been changed to protect patient confidentiality. Though the NBEO PAM-Style format is utilized for the case write-up, there is no affiliation with NBEO.

Demographics:

72 yo Black male new patient

Chief complaint: Ocular Injury OD with reduced vision since

History of present illness

Character/signs/symptoms: reduced vision OD
Location: OD
Severity: stable since initial injury
Nature of onset: after eye injury OD due to running into a table 8 years ago
Duration: 8 years
Frequency: constant
Exacerbations/remissions: foreign body sensation OU
Relationship to activity or function: none
Accompanying signs/symptoms: itchiness and redness

Patient ocular history: (+) eye injury OD w/ orbital floor fracture at that time - ran into table 8 yrs ago (+) enophthalmos OD since injury (-) eye surgery OU

Family ocular history: unremarkable

Patient medical history: (+) Diabetes, (+) Hypercholesterolemia (+)Hypertension (+)COPD (+)Depression (+)Stroke x 3 years; MRI obtained at that time w/ no sequelae per patient (+) arthritis

Medications taken by patient: Insulin, atorvastatin, amlodipine, carvedilol, Trelegy Ellipta, albuterol sulfate, Stiolto Respimat, duloxetine, clopidogrel, meloxicam

Patient allergy history: Penicillin

Family medical history: unremarkable

Review of systems

Constitutional/general health: denies
Ear/nose/throat: Cardiovascular: denies
Pulmonary: denies
Endocrine: denies
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:

	Distance	Near
OD:	20/150	0.4/3.2M
OS:	20/30-	0.4/0.5M

Pupils: PERRL (+) >1.8 log unit APD OD; Bright: 3/2.75mm and Dim: 3.75/3.5mm
Ductions: OD: 90% supraduction, 80% adduction ability; OS: Full with no restrictions
Forced Duction: positive test with limited ability for movement 360 degrees

Confrontation fields: OD: constriction inferior: OS: Full to finger counting and red cap; no defect respecting vertical midline noted OD or OS
Hirschberg: Asymmetric
Cover Test: 10pd right exo and 14pd right hyper
Worth 4 Dot: OD suppression distance and near
Exophthalmometry: 12mm OD 20 mm OS; Base 99mm
Mini Neurological Exam: no upper or lower extremity motor weakness found, normal gait, normal balance, normal heel-to-toe cerebellar function testing, and no dysdiadochokinesia or dysmetria noted on testing

Subjective refraction:	VA Distance	VA Near
OD: +1.25 -0.50 150 ADD +2.50	20/100+	0.4/3.2M
OS: +1.50 -0.75 100 ADD +2.50	20/25-	0.4/0.4M

Slit lamp:
lids/lashes/adnexa: clear lids and lashes OU; enophthalmos OD with right eye deviated temporally and superiorly in primary gaze
conjunctiva: 1+ bulbar conj injection OU; inferior concretions palp conj OU
cornea: 1+ diffuse SPK OD and 1+ diffuse SPK denser inferior OS
anterior chamber: deep and quiet OU, VH 4 T and N OU
Iris: flat and intact OU (-) NVI OU
lens: 1+ nuclear sclerosis, 1+ cortical cataract with central vacuoles OU
vitreous: clear and quiet OU (-) vitreous heme OU

TBUT: 2 seconds OD and 4 seconds OS
IOPs/method: 14/14 mmHg via Goldmann
Fundus OD: ***see image 1***
ONH: Distinct margins with 3+ diffuse pallor (-) NVD
C/D: 0.55/0.60
macula: macular flat and intact (-)CSME
posterior pole: unremarkable with normal course & caliber of vasculature
periphery: flat and intact 360 (-) breaks or RDs (-)NVE
Fundus OS: ***see image 1***
ONH: perfused, healthy, distinct
C/D: 0.40/0.40
macula: macular flat and intact (-)CSME
posterior pole: unremarkable with normal course & caliber of vasculature
periphery: flat and intact 360 (-) breaks or RDs (-)NVE
Blood pressure: 130/72 mmHg RAS manually

Case Images:

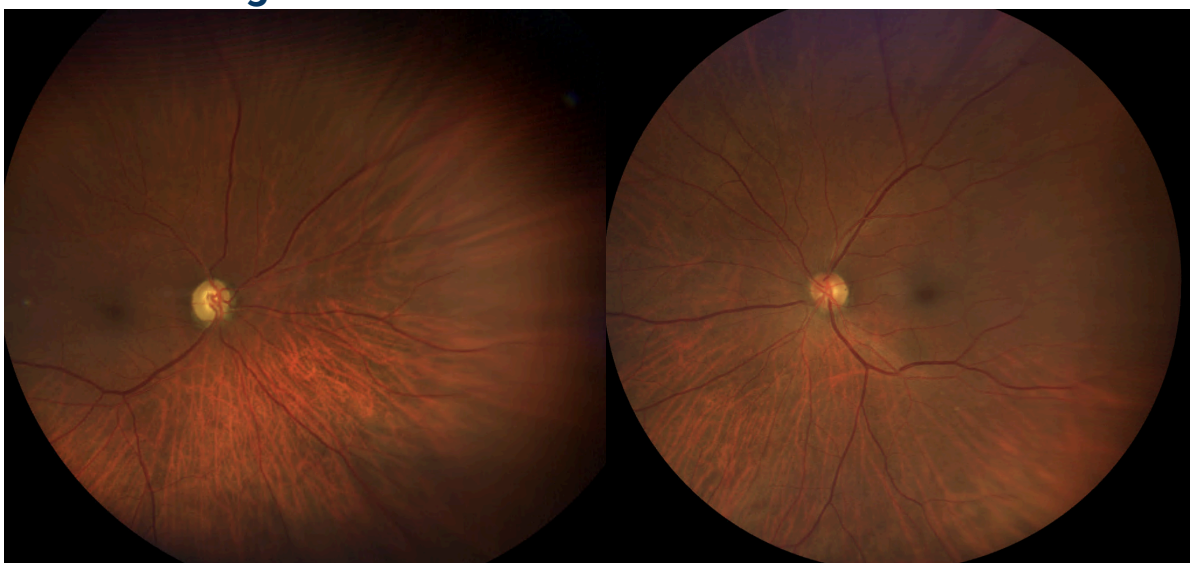

Image 1: Colored Clarus Photograph of the right and left eye, respectively. Note the pallor of the right optic nerve.

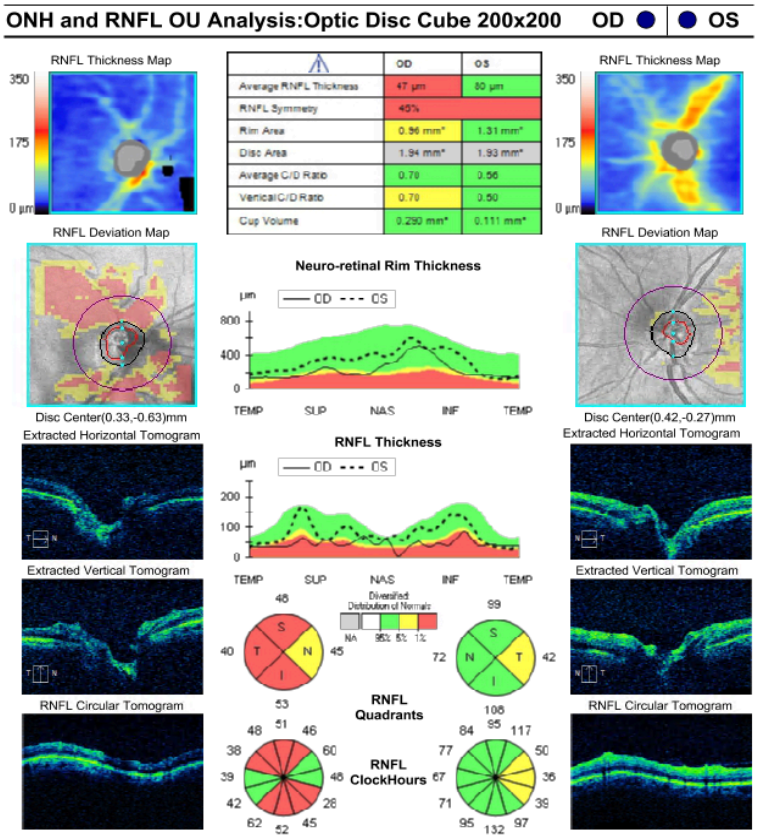

Image 2: Optical Coherence Tomography ONH and RNFL Analysis OU demonstrating significant asymmetry with RNFL thinning 360 OD matching history of trauma OD, afferent testing, and appearance of pallor OD on clinical evaluation.

Case Management Summary

A1: Enophthalmos due to trauma or surgery, right eye (H05.421), Chronic
Examination and history revealed right enophthalmos and optic neuropathy w/ history of trauma 8 years ago OD

- History of orbital fracture OD at time of trauma consistent with ductional deficit and enophthalmos OD noted today
- Exophthalmometry: 12 and 20mm/Base: 99mm; BCVA: 20/100 OD
- Neuro consult obtained with no need for additional work up

P1: Pt was ed on exam findings and sequelae of trauma OD with limited maximum visual potential given permanent optic nerve damage. Records requested from external ER facility treating the initial incident to determine the exact nature of orbital injury. Discussion re full time protective eyewear with polycarbonate lenses given essentially monocular status. Monitor 1 year.

A2: Injury of optic nerve, right eye, sequela (S04.011S), Chronic.
Examination revealed optic neuropathy OD s/p trauma 2015 OD

- Dilated exam revealed 3+ diffuse pallor consistent with visual acuity, APD OD, enophthalmos OD, and history of trauma
- Obtained fundus photos, ONH OCT and GCC OU for baseline

P2: See AP for H05.421. RTC next available for baseline HVF 24-2 given optic atrophy OD and history of stroke x 3years.

A3: Dry eye syndrome of bilateral lacrimal glands (H04.123), Symptomatic.
Examination revealed: Dry Eye Syndrome OU

- Pt symptomatic of: dryness
- BCVA OS: 20/25

P3: Pt was ed on exam findings. Ed on artificial tears, sample given (Refresh) QID - PRN and Gel QHS OU. Monitor 1 year.

A4: Combined forms of age-related cataract, bilateral (H25.813), Chronic.
Examination revealed 1+ Nuclear Sclerosis OU and 1+ Cortical Cataracts OU

- Pt asymptomatic
- BCVA: 20/100 OD (optic neuropathy), 20/25 OS (dry eye)

P4: Pt was ed on exam findings. Ed on signs/symptoms of cataracts including glare/halos and decreased contrast sensitivity. No surgery indicated at this time. Monitor 1 year, sooner if needed.

A5: Hypermetropia, bilateral (H52.03), Chronic and Presbyopia (H52.4), Chronic.
Refraction revealed: compound hyperopic astigmatism OU w/ presbyopia OU

- BCVA: 20/100 OD, 20/25 OS

P5: Updated bifocal Spec Rx released for full time wear with polycarbonate lenses for protection. Pt was ed on minor change from habitual Rx, no adaptation problems expected. Return to clinic 1 year comprehensive eye exam (CEE).

External Records Review:

1. 3 years ago: patient initially presented to ER facility with difficulty of balance and coordination x 5 days in addition to slurred speech noted by family member
    - Blood pressure range: 130-160s/80-90s mmHg
    - Bloodwork: CBC with differential and complete metabolic panel unremarkable
    - CT without contrast: subacute left occipitoparietal lobe infarct
    - CT angiography of head and neck: unremarkable
    - EKG: normal sinus rhythm with left axis deviation
    - Subsequent MRI: acute to subacute infarct of left parietal occipital lobe cortex and local mass effect
    - Management: not a TPA candidate given subacute nature of infarct
        - No neurosurgical intervention warranted
        - Medical management with Rx for Plavix and continued use of high intensity statin; maintain blood pressure and blood sugar control
    - Underwent SLP, OT, and PT for 6 months post incident
        - Worked on mild expressive aphasia post cerebral vascular accident with good recovery
2. CT of orbit indicated old right orbital floor fracture consistent with history of ocular trauma OD

Case Pearls[1]:

- Optic neuropathy is caused due to damage to the axons of the ganglion cells that form the optic nerve which commonly presents as optic nerve head pallor.
- As seen in our patient, trauma is one of many causes of optic neuropathy; however, other causes of optic neuropathy include ischemia, inflammation, infiltration, compression, or congenital causes.
    - Adequate case history is essential to differentiate between causes of optic nerve pallor along with thorough testing
    - Reduced best vision, reduced contrast sensitivity or color vision, visual field defect and afferent pupillary defect are all potential sequelae of optic nerve atrophy.
- External adnexal findings that can indicate a previous history of trauma including:
    - Enophthalmos
        - exophthalmometry should be performed to monitor for any changes
    - EOM dysfunction
        - secondary to compromised orbital integrity and possible EOM entrapment
    - Facial or adnexal scars
        - If adnexal scars are present, it is important to inquire about the cause by expanding upon patient history
- After initial injury to the optic nerve, optic atrophy typically develops after 4-6 weeks. Injury can occur to any portion of the optic nerve pathway (intraorbital, intracanalicular, or intracranial).
    - When suspecting traumatic optic neuropathy, OCT and visual field testing can prove to be a pertinent resource for baseline and continuous monitoring.

    - Patients with optic neuropathy have more fragile RNFL and neuro-retinal rim tissue and are at risk for developing glaucoma therefore should be monitored as such.
- The onset of vision loss is variable and the prognosis of optic neuropathy is dependent on the extent of the injury and the time before obtaining treatment.
    - Patients with traumatic optic neuropathy should be monitored closely, encouraged to wear polycarbonate lenses for protection, and educated to return for follow up sooner if there are any changes in vision.

Resource:

Hosseini Siyanaki MR, Azab MA, Lucke-Wold B. Traumatic Optic Neuropathy: Update on Management. *Encyclopedia*. 2023; 3(1):88-101. https://doi.org/10.3390/encyclopedia3010007