SALUS UNIVERSITY
The Eye Institute

Pennsylvania College of Optometry

The Focal Point

January 2024 Edition

Ryan Walchuk

Scholars Class of 2025

Hometown: Saskatchewan, Canada
Undergrad: University of Saskatchewan
Major: Sports Medicine
Favorite Animal: Big frogs
Optometry Goal: Help people
Favorite instrument: Harmonica
Hobby: Competitive dog petting
Last Show I binged: Pediatric and Infant Vision Lec 10-15

Simran Kaur

Traditional Class of 2024

Hometown: Long Island, New York
Undergrad: New York Institute of Technology
Major: Biology **Minor:** Psychology
Favorite Animal: Dogs
Optometry Goal: Work/Life Balance
Favorite instrument: Violin
Hobby: Going to concerts
Last Show I binged: Loki

Behrad Garmsiri, OD, MS

Class of 2023, SUNY College of Optometry

Hometown: Mississauga, Ontario Canada
Undergrad: McMaster University
Major: Biological Sciences; Physiology
Favorite Diagnostic Instrument: Optical Coherence Tomography
Loves: coffee and pomegranates
Hobby: Real Time Strategy Video Games

Amar-OH-NO-sis Fugax

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Initial Emergency Service Visit:

Demographics

76 yo Black female; retired

Chief complaint: transient vision loss

History of present illness

Character/signs/symptoms: complete loss of vision
Location: OD
Severity: Severe
Nature of onset: Sudden
Duration: 1-2 minutes before full remission
Frequency: Second episode in last 2 months
Exacerbations/remissions: none
Relationship to activity or function: none
Accompanying signs/symptoms: none

Patient ocular history: None

Family ocular history: Non-contributory

Patient medical history hypertension, hypercholesterolemia, previous myocardial infarction, degenerative arthritis, asthma

Medications: chlorthalidone, clopidogrel, carvedilol, rosuvastatin, oxycodone, accutane sulfate inhaler, methylprednisolone

Patient allergy history: NKDA

Family medical history: Non-contributory

Review of systems

Constitutional/general health: (-) malaise
Ear/nose/throat: denies
Cardiovascular: denies
Pulmonary: cough
Endocrine: denies
Dermatological: denies
Gastrointestinal: denies
Genitourinary: denies
Musculoskeletal: numbness of left pointer finger for ~5 days (-) jaw pain
Neurologic: (-) headache
Psychiatric: denies
Immunologic: denies
Hematologic: denies

Mental status

Orientation: oriented to person, place, and time
Mood/Affect: normal

Clinical findings

BVA:	Distance	Near
OD:	20/20-1	.4/.4M
OS:	20/20-1	.4/.4M

Pupils: PERRL, (-) APD

EOMs: 95% Abduction deficit OU, 100% otherwise OU

Confrontation fields: FTFC OU

Hirschberg: Symmetric OU

Slit lamp:

lids/lashes/adnexa: few clapped glands superior OD, unremarkable OS
conjunctiva: bulbar inclusion cyst nasal and temporal OD, white and quiet OS
cornea: arcus 360 OU, oily tear film OU; normal endothelium and epithelium OU
anterior chamber: deep and quiet OU
iris: flat and intact OU; small, flat nevus 12:00 OD
lens: 1+ NS and trace cortical OU
vitreous: PVD OU

IOPs/method: 11/11 mmHg by iCare

Fundus OD and OS: optic nerve, C/D, maculae, posterior pole: ***See Image 1***

Blood pressure:

At 1:00pm: 185/77mmHg right arm, sitting by manual cuff
At 1:36pm: 180/75mmHg right arm, sitting by manual cuff

Initial Visit Images:

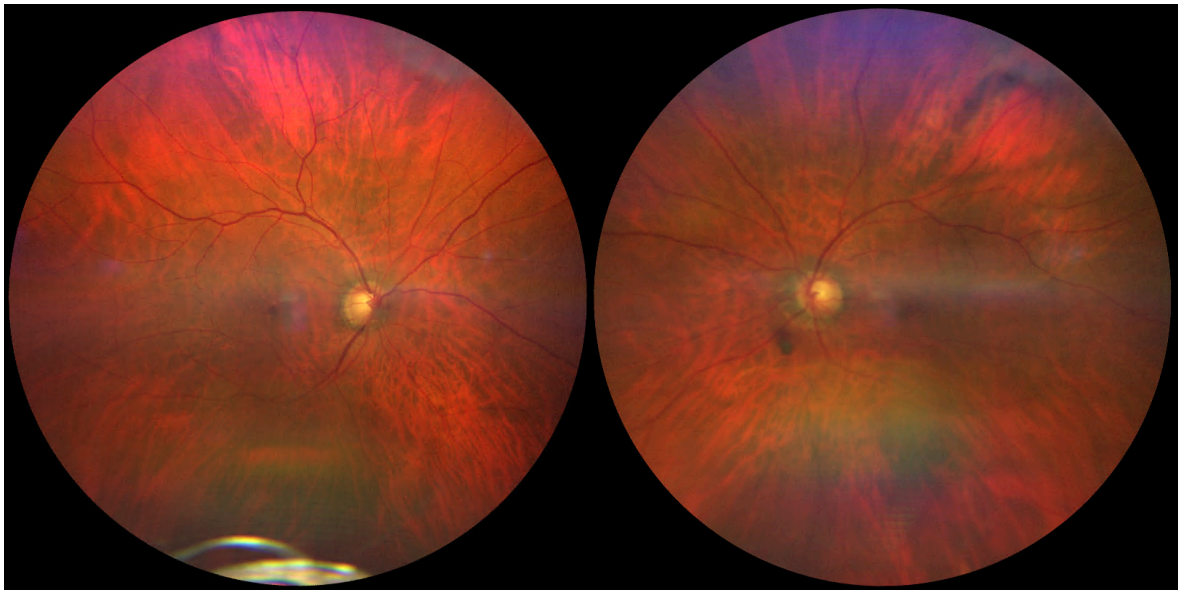

Image 1: Colored fundus photographs OD and OS, respectively. Of note: drusen inferior maculae and attenuated vessels OU; inferior artifact OD>OS and central artifact OS

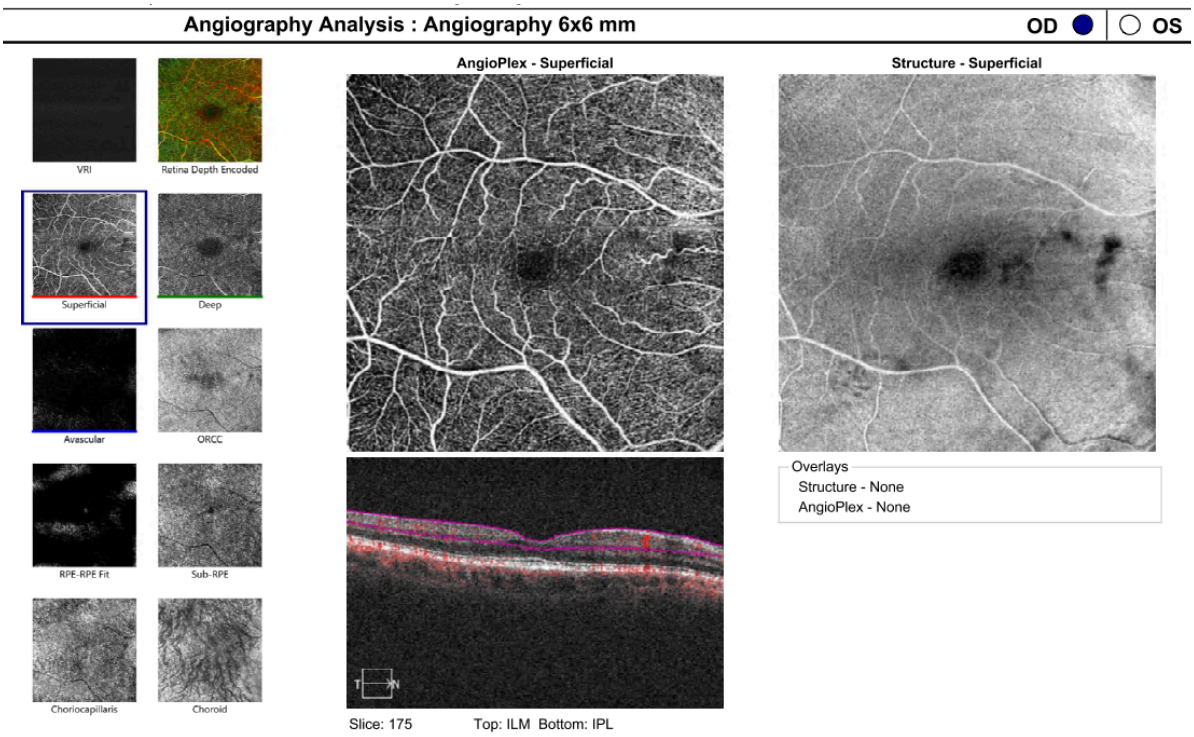

Image 2: OCT-Angiography OD demonstrating the superficial vascular layer on Cirrus OCT. Dark patches seen nasal to fovea on the "Structure" layer (right) are floaters that also cast shadows in the retinal cross section (bottom). OCT-A does not reveal issues with vascular flow OD. OCT-A OS (not shown here) is unremarkable.

Case Management Summary - Initial ER Visit

Assessment 1: Amaurosis Fugax OD. Diagnosis on history and symptoms alone as no retinal emboli were seen on undilated retinal examination, color fundus photos, or OCT-A scan. Patient has vascular risk factors of hypertension and hypercholesterolemia with elevated blood pressure in office today (180/75mmHg).
Plan 1: Patient was educated on exam findings and relationship of nature of symptoms to impending ischemic stroke. Emergent referral to hospital with Primary Stroke Center for stroke work-up and blood work to rule out GCA. RTC 1 month for follow up unless other changes noted.

Assessment 2: Grade 1 Hypertensive retinopathy OU with blood pressure in office 185/77mmHg at 1:00 pm & 180/75mmHg at 1:36 pm
Plan 2: See plan 1

Second Emergency Service Visit (Pertinent History only):
Chief complaint: worsening vision OD/ Transient vision loss
History of present illness

Character/signs/symptoms: central vision is dark/opaque/hazy OD; peripheral vision is not affected
Location: OD
Severity: severe
Nature of onset: ever since she left the ER hospital 4 days prior
Duration: persistent
Frequency: the intensity of the blur changes throughout the day
Exacerbations/remissions: none.
Relationship to activity or function: none
Accompanying signs/symptoms: (-) pain, scalp tenderness, muscle weakness, slurring of speech, or headaches

Pertinent Patient Medical History: patient reports going to ER hospital with dedicated stroke center directly after last visit and getting stroke work up and blood work . CT scan performed at that time of the head and neck and acute abnormalities were ruled out. Numbness of the left pointer finger was attributed to "trauma".
Pertinent Clinical findings:

BVA Distance
OD: 5/100 used eccentric viewing to see the letters
PH: unable
OS: 5/60 PH: 20/20

Pupils: PERRL, (+) APD OD
Red cap desaturation: 90% OD
Confrontation fields: central darkness (only examiner's eyebrow is clearly visible) with peripheral vision intact OD; FTFC OS
Slit lamp: unchanged from previous visit
IOPs/method: 13/15 mmHg by iCare
Fundus OD and OS: optic nerve, C/D, maculae, posterior pole: ***See Image 3***

Blood pressure: 152/60mmHg right arm, sitting by manual cuff

Second Visit Images:

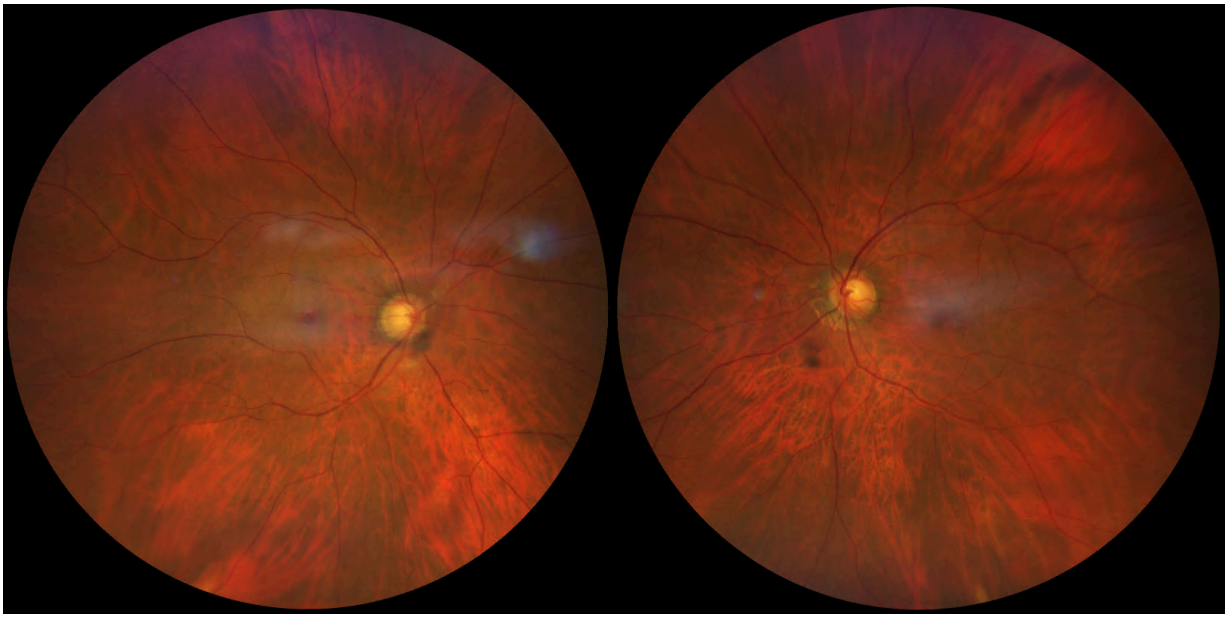

Image 3: Colored fundus photographs OD and OS, respectively at 4-day follow up visit. Of note: macular tissue whitening with cherry red spot consistent with a central retinal artery occlusion OD; unchanged presentation OS from initial visit. Note superior nasal artifact OD and central artifact OS. Difficult to see in this image, however visible on fundoscopy, is a cilioretinal blood vessel emerging from inferior optic disc OD.

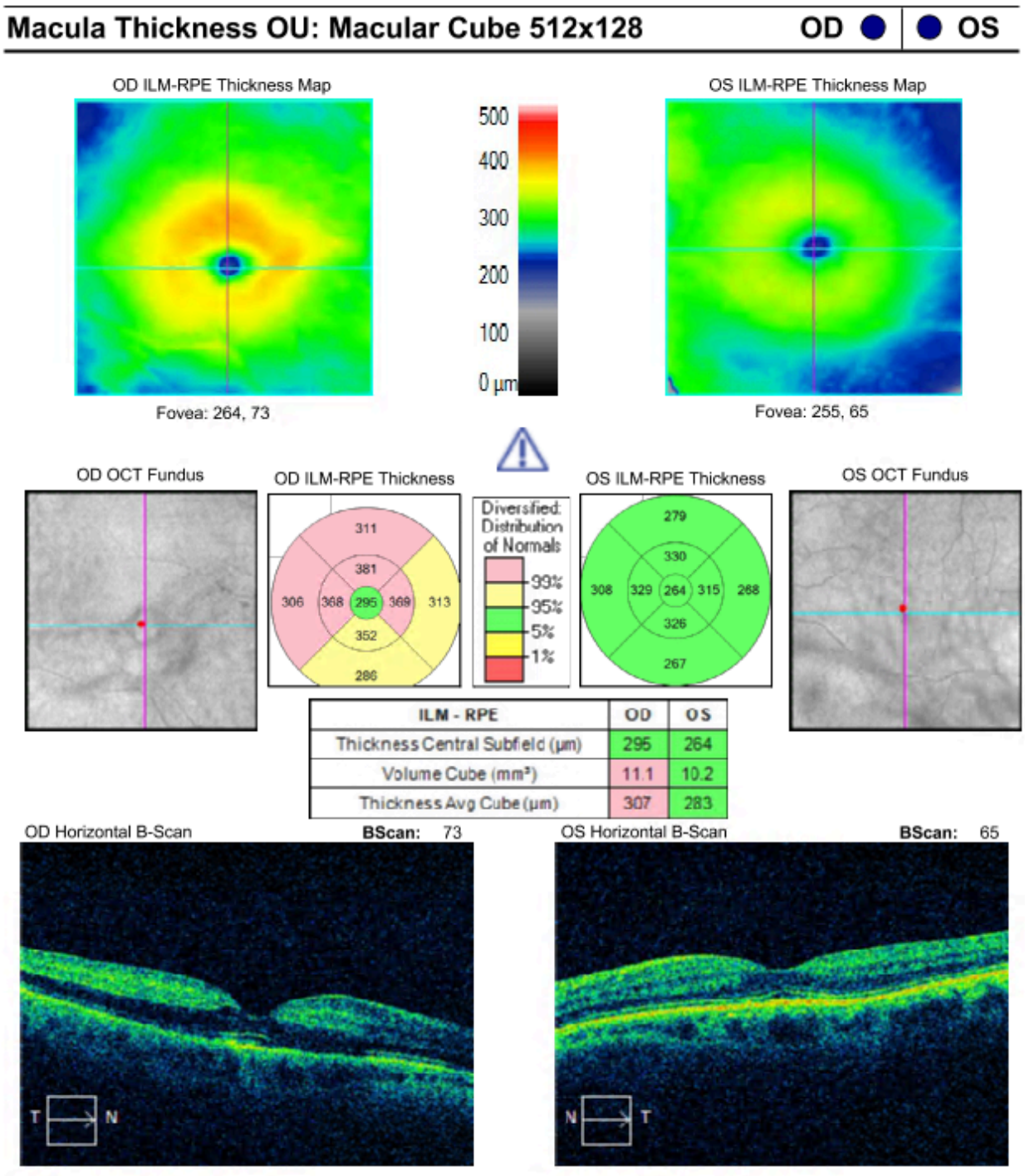

Image 4: 512x128 macular cube OCT OD and OS. Of note: increased tissue thickness and marked hyper-reflectivity of inner retinal layers and shadowing of deeper retinal layers which is consistent with acute tissue ischemia OD. Unremarkable OS.

Case Management Summary - Second ER Visit

Follow-up Visit

Assessment 1: Central Retinal Artery Occlusion (CRAO) OD
(+) Cherry red spot OD; (+) APD; macular thickening OD seen with OCT imaging; no other neurological symptoms

Plan 1: Patient was educated on examination findings and ischemic event to ocular tissue OD. Discussed the need for emergent intervention and work-up to prevent further complications. Urgent referral to emergency hospital with primary stroke center for a full stroke work up again; sent to different hospital from initial encounter.

Post visit co-management notes:

- Patient went to the emergency hospital and was admitted for 3 days. Full work up including CRP, ESR, platelets, EKG, transthoracic echocardiogram, and MRI of head, neck, and brain which were all unremarkable.
- Patient referred to a retinal specialist where CRAO OD was confirmed and recommended for monthly observation with no treatment available.
- Patient reports a small area of vision with eccentric viewing OD. This is likely due to luxury perfusion provided by the presence of cilioretinal artery OD.

Case Pearls

- Amaurosis Fugax (AF) is considered a sign of an impending stroke and a diagnosis can often be made on history and risk factors alone. Visualization of retinal emboli is not necessary for diagnosis. **An immediate referral to an emergency hospital with a dedicated stroke center for stroke work-up is warranted**. A correct referral can potentially prevent vision loss and mortality. Clinicians should be aware of local area hospitals with dedicated stroke centers.
- The recurrent nature of AF is indicative of a systemic condition where emboli are being "thrown around" throughout the body causing a transient ischemic incident. Common conditions include carotid artery disease, calcification of the valves of the heart, and deep vein thrombosis.
- The three main types of emboli are cholesterol, calcium, and platelet-fibrin. Both cholesterol and platelet-fibrin emboli typically arise from atheromas in the carotid arteries. Calcium emboli typically arise from cardiac valves. On fundoscopy, calcium emboli appear white, cholesterol emboli (Hollenhorst plaques) appear orange, and platelet-fibrin emboli appear dull white. Usually, CRAO are caused by larger, calcific embolus[1]. **Giant cell arteritis (GCA) in any patient over the age of 50 with AF, must be ruled out**. Clinicians should remember to ask about common symptoms such as: jaw claudication, headaches, scalp pain/tenderness, fever, weight loss, muscle aches, and general malaise as part of their review of symptoms. Important clinical manifestations of GCA to keep in mind include: elevated ESR, temporal artery abnormalities, arteritic anterior ischemic optic neuropathy, CRAO, along with ocular motility restriction and resulting diplopia.
- CRAO is an unfortunate complication of systemic disease that results in an occlusion of the central retinal artery - a branch of the ophthalmic artery - that often leads to permanent vision loss. **Vision is unlikely to return in the affected eye and treatment is directed towards preserving vision in the other eye by attending to systemic health.**

- Patients with a CRAO may still have some areas of vision in the affected eye **if they possess a cilioretinal artery**. The cilioretinal artery is another branch of the ophthalmic artery that is less likely to be occluded with systemic disease and can provide islands of vision (usually to the macular area) to patients who have had a CRAO, if one is present. This can manifest as eccentric fixation when testing visual acuities in-office. Eccentric viewing training is a tool that can be co-managed with low vision rehabilitation specialists.
- **CRAO can cause acute congestion and resultant tissue thickening** to occur in the macular area due to oxygen depletion that ultimately leads to break down of retinal cell homeostasis, leading to swelling of tissue[2]. Long-term sequelae include retinal atrophy of the affected tissue.
- Despite several suggested treatments for acute CRAO, there have been no conclusive evidence to support their use. Previous animal studies demonstrate that **the retina can tolerate ischemia for up to 100 minutes and suffers irreversible damage thereafter**[3].
- If identified early, treatment for CRAO with ocular massage, administration of vasodilators, or IOP lowering medications may be beneficial, especially if applied within **6 hours of initial artery occlusion**[4].

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1. Farris W, Waymack JR. Central Retinal Artery Occlusion. [Updated 2023 Sep 4]. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2023 Jan-. Available from: https://www.ncbi.nlm.nih.gov/books/NBK470354/
2. Furashova O, Matthé E. Retinal Changes in Different Grades of Retinal Artery Occlusion: An Optical Coherence Tomography Study. Invest Ophthalmol Vis Sci. 2017 Oct 1;58(12):5209-5216. doi: 10.1167/iovs.17-22411. PMID: 29049721.
3. Hayreh SS, Kolder HE, Weingeist TA. Central retinal artery occlusion and retinal tolerance time. Ophthalmology. 1980 Jan;87(1):75-8. doi: 10.1016/s0161-6420(80)35283-4. PMID: 6769079.
4. Cugati S, Varma DD, Chen CS, Lee AW. Treatment options for central retinal artery occlusion. Curr Treat Options Neurol. 2013 Feb;15(1):63-77. doi: 10.1007/s11940-012-0202-9. PMID: 23070637; PMCID: PMC3553407.