Incomplete CRAO: Rare Diagnosis and Potential Glaucoma Mimicker

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Abstract

Many different ocular conditions can mimic glaucoma in causing retinal ganglion cell death and visual field loss. One subgroup of said ocular conditions includes retinal vascular occlusive events. There is a particularly under-appreciated and rare condition within this subgroup, the partial or incomplete central retinal artery occlusion. Clinical presentation, prognosis, and systemic associations of partial/incomplete CRAO will therefore be examined using a case as an example. Optical coherence tomography methods to distinguish between different etiologies behind retinal ganglion cell death will also be discussed using this case. These methods will be of use to clinicians in cases where previous ocular history is unable to be acquired.

KEY WORDS
Incomplete/partial central retinal artery occlusion, disc arteriolar collaterals, glaucoma, optical coherence tomography, visual field

INTRODUCTION

Incomplete or *partial* central retinal artery occlusion (iCRAO) is discussed significantly less in the literature and optometric/ophthalmologic lectures than *complete*-CRAO.[1] There is very limited epidemiological data on iCRAO incidence/prevalence with only a handful of case reports published.[1-5] Clinicians therefore might not be aware of this condition's presentation and natural history. Systemic hypertension, giant cell arteritis, cigarette smoking, and ipsilateral carotid stenosis are risk factors for iCRAO.[1,2]

The CRA suffers less extensive obstruction in iCRAO compared to *complete*-CRAO.[2,4,6] Retinal regions in iCRAO thus suffer less hypoxia than in *complete*-CRAO, where severe oxygen deprivation leads to widespread/diffuse inner-retinal infarction.[6-8] Patients with iCRAO typically present with RAPD and unilateral acuity loss ranging from 20/40 to 20/400, though entering acuity was counting fingers in one report.[1,2,5,6] Reduced acuity and RAPD result from a combination of some localized inner-retinal neuronal death and surviving neurons in other areas entering a conservatory *hypometabolic* state until restoration of oxygen levels. This hypometabolic, reduced functioning state persists until spontaneous resolution of the partial occlusion occurs and/or arterial collateral connections open up with the choroidal circulation.[6,7] Unlike in *complete*-CRAO cases, significant acuity restoration often occurs in eyes over the coming days or weeks in iCRAO cases.[1,2] Ultimately, however hypoxia induced atrophy of the inner-retinal areas hit hardest by the acute oxygen depletion leaves areas of retinal nerve fiber (RNFL) defects and corresponding visual field (VF) loss following iCRAO.[2]

Glaucoma, like iCRAO, features *localized* RGC death, RNFL defects, and corresponding VF loss.[8] These initially structural defective areas are often located in the superior-temporal and inferior-temporal areas of both the macula and disc due to the presence of glaucoma *vulnerability* zones.[9] However, these characteristic areas of RGC cell axonal loss are not exclusive to glaucoma. Localized RNFL loss can also be seen in retinal vein occlusion (RVO), ischemic optic neuropathy, optic neuritis, iCRAO, branch CRAO and

cases of isolated CWS, to name a few.[10-13] With OCT use now common, it is important to be aware that not all cases of RNFL loss are glaucomatous (even if the loss is superior-temporal or inferior-temporal and there is a functional field loss correlation).[12] When the patient presenting with RNFL loss has ocular hypertension, it is easy to decide to start IOP lowering therapy . When such a patient presents with *normal* IOP, the entire clinical picture must be weighed. For example, more nasally located macular RGC loss, presence of disc collaterals and/or pallor, and recollection of sudden vision loss point away from glaucoma as the likely cause of the loss.[13-16]

CASE REPORT

A 58-year-old white female was referred to a teaching institution for ongoing glaucoma management after she lost her job and medical insurance. The referring optometrist had initiated treatment for glaucoma two years earlier. Upon presentation after referral, the patient claimed compliance with latanoprost 0.005% once per evening in both eyes and brimonidine 0.2% twice daily in the left eye. Systemic medications included irbesartan/hydrochlorothiazide 300/25 mg once daily, aspirin 81 mg po once daily, and rosuvastatin 20 mg po once daily. She admitted to smoking one pack of cigarettes per day for thirty years. Blood pressure measured 135/90 mmHg. Best-corrected acuity measured 20/20 OD and 20/25 OS with a refraction of +1.25 OD, OS. Pupil assessment showed 1+ RAPD OS. Goldmann IOP measured 12 mmHg OU. Gonioscopy revealed open angles with trace pigment. Central corneal thickness measured 545 mm OU. C/D ratios measured 0.5 OU. Mild temporal rim pallor and disc arteriolar collaterals were noted OS only (Figure 1 A, B). Hypertensive arteriolar narrowing was noted OU.

Healthy RNFL and macular RGC OCT analysis correlated with a clean 24-2C Humphrey visual field OD. A dense inferior paracentral VF defect correlated with superior temporal RNFL loss and abrupt, severe superior-nasal RGC loss OS (Figures 2A & 3A). An inferior-temporal RNFL defect correlated with the superior-nasal step on VF OS (Figure 2A, 3B). OCT-Angiography (OCT-A) showed flow deficits in the inter-arterial superficial and deep capillary bed regions of the superior nasal macula and inferior-temporal arcades OS, correlating with the above RNFL and field defects (Figure 3 C-E)

The patient was advised her left eye showed more evidence of a prior vascular occlusion than glaucoma. She recalled an event of abrupt, painless vision loss OS three years ago when asked probingly. She saw a retinal specialist within 4 days of onset. She did not remember being told about elevated eye pressures at any previous exams. Latanoprost, expensive for her, was discontinued in both eyes. The patient agreed to follow-up in 6 weeks for an IOP check. Previous exam records were requested.

Previous exam notes and sequential fundus photos obtained from the retina specialist demonstrated an iCRAO OS three years ago with subsequent CWS resolution and disc arteriolar collateral formation over ten weeks (Figure 1D-G). Aided VA at iCRAO presentation had measured 20/200 OS (no improvement with pinhole), improving to 20/50 OS at 3 weeks and 20/30 OS at ten weeks. Carotid duplex ultrasonography, echocardiogram as well as CBC with differential, ESR, CRP, p-ANCA, c-ANCA, ANA, lipid panel, comprehensive metabolic panel, GFR, lipase, amylase, blood sugar and A1C had been ordered. 50% stenosis of the left proximal internal carotid artery (ICA) with multiple small, heterogenous plaques was found. Echocardiography was unremarkable. There was no ESR, CRP or platelet elevation suggestive of RAO secondary to giant cell arteritis. ANA and ANCA screening for collagen vascular disease associated vasculitis was negative. Hematocrit, hemoglobin as well as total WBC and sub-populations were also normal, ruling out anemia and thrombocytopenia. Blood sugar/A1C were normal. The patient was referred to a cardiologist, who initiated rosuvastatin and low-dose aspirin. Serum amylase, lipase as well as creatinine and GFR were normal confirming normal pancreas and renal health, respectively. More history later received from the referring optometrist indicated pre-treatment IOP max of 17 mmHg OU. IOP lowering therapy OS>OD had been initiated two years ago due to glaucoma diagnosis based on RNFL/RGC thinning and field loss OS.

After discontinuing latanoprost, the patient returned to the teaching institution six weeks later. IOPs measured 17 mmHg OD and 15 mmHg OS. Given the extensive RGC and RNFL damage present, the potentially helpful neuro-protective effects of brimonidine 0.2% were discussed. However, the patient preferred stopping brimonidine given its sizeable cost. She agreed to follow-up in five weeks to fully rule out any predilection towards ocular hypertension.

Five weeks later Goldmann IOP measured 17 mmHg OU, confirming along with her case history and records, that neither eye had an ocular hypertensive tendency. Over the past year, IOP has remained in the mid-teens, measuring 16 mmHg OU via GAT at last follow-up. Visual field, RNFL and minimum rim width (MRW) have

also remained stable from baseline (Figures 2 & 4). Ten mid-peripheral dot-blot hemorrhages OS were noted at the last follow up (Figure 5). These were not visualized at earlier follow ups or at the time of the iCRAO. The patient denied any amaurosis fugax or ocular/orbital pain episodes. Anterior segment exam and gonioscopy did not reveal any inflammation or neovascularization. Blood pressure at last follow-up measured 160/94 mmHg. The patient claimed compliance with irbesartan/hydrochlorothiazide but admitted to not visiting her primary care physician (PCP) or cardiologist in the past six months due to lack of insurance. She was again counselled on smoking cessation, healthy diet and lifestyle choices and medication/medical visit compliance. She was counselled her hemorrhages may be associated with early ocular ischemic syndrome (OIS). Her PCP was called and agreed to requisition repeat blood work (including lipid panel and A1C) as well as repeat carotid imaging and see the patient for blood pressure measurement.

DISCUSSION

Sudden unilateral painless vision loss, RAPD as well as areas of CWS and middle-retinal whitening are consistent with the few published iCRAO cases.[1-7] The proposed pathophysiology of iCRAO helps explain its retinal signs. A partial CRA occlusion permits a small amount of residual flow through the artery lumen.[3,6,7] A retinal version of *misery perfusion* results, whereby the limited oxygen still flowing downstream of the occlusion is used by retinal neurons on a 'first-come, first-served basis'. Upstream neurons closer to the arteries and further from the veins extract the oxygen they need, leaving little for the more downstream neurons.[6,17] Hypoxia to bipolar cells and interneurons in the deep capillary plexus well downstream of the retinal arteries explains the mid-retinal whitening in inter-arterial areas known as hypoperfusion maculopathy in iCRAO cases (Figure 1 E).[6,7,17] The proximity to higher oxygen levels in the arteries explains the infarction-free zone immediately surrounding the retinal artery branches.[17] The blood flow voids on OCT-A in-fact appear most apparent in the capillary beds in watershed areas between different retinal artery branches (Figure 3 C,D). Moreover, the more superficially located CWS, also appear to be located in inter-arterial areas (Figure 1 D,E). These CWS represent cessation of axoplasmic mitochondrial flow in RGCs. This stagnation is due to ischemia in the axonal portions further away from the still functioning posterior ciliary circulation nourishing the nerve.[6,7] Finally in iCRAO, more peripheral retinal areas are thought to not infarct/whiten due to their cell layers being thinner with less neuronal density. These areas require relatively lower oxygen levels.[6]

Through hypometabolic and other protective processes, retinal neurons exposed to reduced oxygen levels during iCRA occlusion are able to survive for hours to even days. This survival-state persists until adequate flow through the CRA resumes and allows significant but incomplete restoration in acuity and field.[1-3,6,7] This patient's acuity in her affected eye improved from 20/200 at acute presentation to 20/50 and 20/30 at three and ten weeks, respectively. This remarkable improvement without therapeutic intervention is in line with other case reports.[1,2] One patient even improved from counting fingers at presentation to 20/50 four weeks later.[1] Spontaneous CRA recanalization is thought to occur at variable time intervals across patients, from minutes to hours to days.[6] Widely-accepted management after non-arteritic CRAO includes initiating anti-platelet medication to lower risk of thromboembolism to the CRA; further optimization of lipid/cholesterol and blood pressure levels; and cigarette smoking cessation to lower risk of plaque rupture.[3] Statin initiation reduces risk of plaque rupture and subsequent embolization, hence why this patient was started on rosuvastin. This addition was important given she was found to have small heterogenous (unstable) carotid plaques on ultrasound that could easily have micro-embolized, explaining her iCRAO event.[18] She was also started on low-dose aspirin to reduce risk of clot formation (and subsequent thromboembolism) from plaque rupture. She did not have a surgical-level of carotid stenosis estimated by doppler after the iCRAO event. However at most recent follow-up, she appears to have a possibility of *worsening* left carotid disease due to new mid-peripheral dot-blot hemorrhages OS (Figure 5). This may represent OIS onset and repeat carotid imaging, three years on, was thus suggested to the PCP. Carotid stenosis >90% leading to ocular hypoperfusion is often present in patients with OIS and requires endarterectomy or stenting. On top of this, her worsening blood pressure control is also worrying as turbulent, rapid blood flow due to hypertension predisposes towards plaque formation.[6]

This case demonstrates that arterial-arterial collateral formation might help restore flow in iCRAO cases.[6] Cilioretinal (or *Nettleship)* disc collaterals were seen to definitively form by ten weeks post-iCRAO (Figure 1B, E-G).[19] These connections between the deeper posterior ciliary artery circulation to the nerve and CRA branches downstream of the occlusion allow blood to bypass the blockage.[6,20] There is no data available on frequency of disc collateral formation in cases of iCRAO, given its rarity. However, disc arteriolar collaterals were found to form in roughly one-fourth of patients with non-arteritic *complete* CRAO.[19] Disc arteriolar collaterals are lighter and narrower compared

to venous disc collaterals moving blood from an occluded CRV to a choroidal vein following central or hemiretinal RVO.[20] Ocular hypertension and high-tension glaucoma are associated with increased risk for both RAO and RVO. It was thus important to ensure IOP did not rise to hypertensive levels after drop discontinuation.[21,22]

Differential diagnoses for this case include *complete*-CRAO and purtscher-like retinopathy (PLR). *Complete*-CRAO often presents unilaterally with sudden, painless vision loss therefore potentially fitting this patient's presentation. However, *complete*-CRAO typically yields more *diffuse* retinal whitening. This contrasts with the *patches* of middle-retinal whitening and CWS seen here (Figure 1D,E). Arteriolar segmented blood flow (box-caring) is also commonly seen acutely and chronically in *complete*-CRAO but was not present here.[19] Finally significant improvement in acuity is not typical of *complete*-CRAO.[1,2]

Purtscher-like retinopathy (PLR) presents similarly to iCRAO with numerous patches of inner-retinal whitening in the middle-retina (Purtscher-flecken) and superficial retina (CWS). It can present unilaterally but is more commonly bilateral.[23,24] Systemic associations include nephrotic syndrome, connective tissue disease, pancreatitis, anemia, thrombocytopenia, pre-eclampsia as well as retrobulbar and systemic subcutaneous injections. Blockage of the CRA by complement activation and/or air, fat, steroid, or oil embolism to the CRA and its downstream arterioles yields the PLR retinal appearance.[23-25] This case was labeled an iCRAO and not PLR as this patient's workup was negative for collagen vascular disease markers, pancreatitis, anemia, thrombocytopenia, or renal dysfunction. It was therefore not consistent with a clear, known PLR association. Moreover just like in this patient, carotid artery disease, hypertension, and hypercholesteremia were the only systemic findings after an extensive systemic workup in another published iCRAO case.[1] Disc arteriolar collateral vessels have not been associated with PLR either. Nonetheless, the similarity in retinal signs seen in PLR and iCRAO hints at a shared pathophysiology: embolic occlusion of the CRA and/or its branches.

It is vital for clinicians to distinguish between glaucomatous and non-glaucomatous RGC and VF loss. Glaucomatous loss is typically progressive, which can eventually be sight-threatening without appropriate IOP lowering therapy. CWS are a well-established non-glaucomatous cause of RNFL loss.[26] CWS can occur along the superior and inferior-temporal RNFL bundle regions, mimicking glaucomatous loss.[27] In this case, the author was fortunate to acquire previous photos and charts. However even without this, other clinical clues present would have helped in differentiating the cause of the RNFL and VF loss. Disc collaterals, mild disc pallor, and history of abrupt vision loss are more commonly seen in cases of vascular occlusion than glaucoma. Furthermore, patients with early-moderate glaucoma tend to most commonly present with macular RGC loss that is more pronounced in inferior-temporal, inferior, and/or superior-temporal sectors.[14] Informatively, this patient's RGC loss was most concentrated superior-nasal. Glaucomatous RGC also generally follows an arcuate pattern towards the disc. In contrast, RGC loss secondary to a vascular occlusion often is more focal, abrupt and severe. This is due to obliteration/loss of the local network of capillary beds, in addition to the RGC neurons themselves. As a result, eyes post-occlusion often have significantly thinner minimums macular RGC thicknesses OCT compared in glaucoma.[15] This patient had a minimum macular RGC of 32 mm OS on Cirrus, well lower than minimum thicknesses on average of 67 mm, 53 mm, and 46 mm seen in mild, moderate, and advanced stages of perimetric glaucoma, respectively.[14, 15]

Glaucoma is typically associated with enlarged cupping, which OCT can aid in confirming via its measured optic nerve head parameters including rim area, C/D ratio and cup volume. A rim area cut off of 0.96mm^2 on Cirrus corresponds to 90% specificity for perimetric glaucoma with increasing rim area above this cut off being inversely related to glaucoma risk.[28] This patient had a rim area of 1.17 mm^2 OS, thus inconsistent with glaucoma (Figure 3 B). Also inconsistent with glaucoma is the fact that vertical and average C/D ratio along with cup volume were not flagged. If this patient has glaucoma, progression on VF and OCT imaging would be expected over time. Lack of RNFL progression at very thin values, the RNFL 'floor', can give a false-sense of security. Her average RNFL values are already at the floor, even on the Spectralis, the OCT device with the lowest floor of roughly 50 mm for a roughly 3.5 mm diameter circle around the disc. (Figure 4 A,B).[29] More emphasis has thus been on monitoring VF and Spectralis MRW parameters, which still shows progression later into the course of optic neuropathies. MRW measures the perpendicular (shortest) distance between the end of Bruch's membrane approaching the disc and the internal limiting membrane to gauge neuro-retinal rim thickness.[30] Informatively, MRW has been stable thus far along with her VF (Figure 4 C,D). Continued monitoring will be needed to *fully* rule out glaucoma but it is sensible for the patient to use her limited healthcare budget elsewhere than glaucoma treatment for now.

CONCLUSION

In conclusion, iCRAO presents with sudden, painless vision loss, RAPD and circinate CWS around the nerve. Acuity often improves in the following weeks, however localized RNFL and VF loss often persists. iCRAO cases are often associated with hypertension, smoking, hyperlipidemia and carotid stenosis and plaques. In new patients presenting with chronic RNFL and VF defects, acquisition of previous records helps determine the etiology of the loss. Inquiring about a history of abrupt vision loss as well as looking for disc collaterals, rim pallor, and the pattern of macular RGC loss can also inform whether loss is or is not glaucomatous in nature. Finally, it is possible that the same eye suffers both vascular occlusion and glaucoma. Monitoring for progressive thinning and field loss representative of the later is therefore important. ●

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Figure 1: *A) Fundus photo of right eye upon referral to teaching clinic. B) Left eye upon referral with temporal rim pallor and cilioretinal collaterals (blue arrows). C,D) Right and left eye photos from retinal specialist at time of iCRAO OS. E: Magnified view of D showing circinate CWS pattern around disc and middle-retinal whitening known as hypoperfusion maculopathy (black asterisks). F) Left eye 3 weeks after iCRAO showing resolution of retinal whitening but not full disc collateral formation G) Left eye 10 weeks after iCRAO showing full collateral formation.*

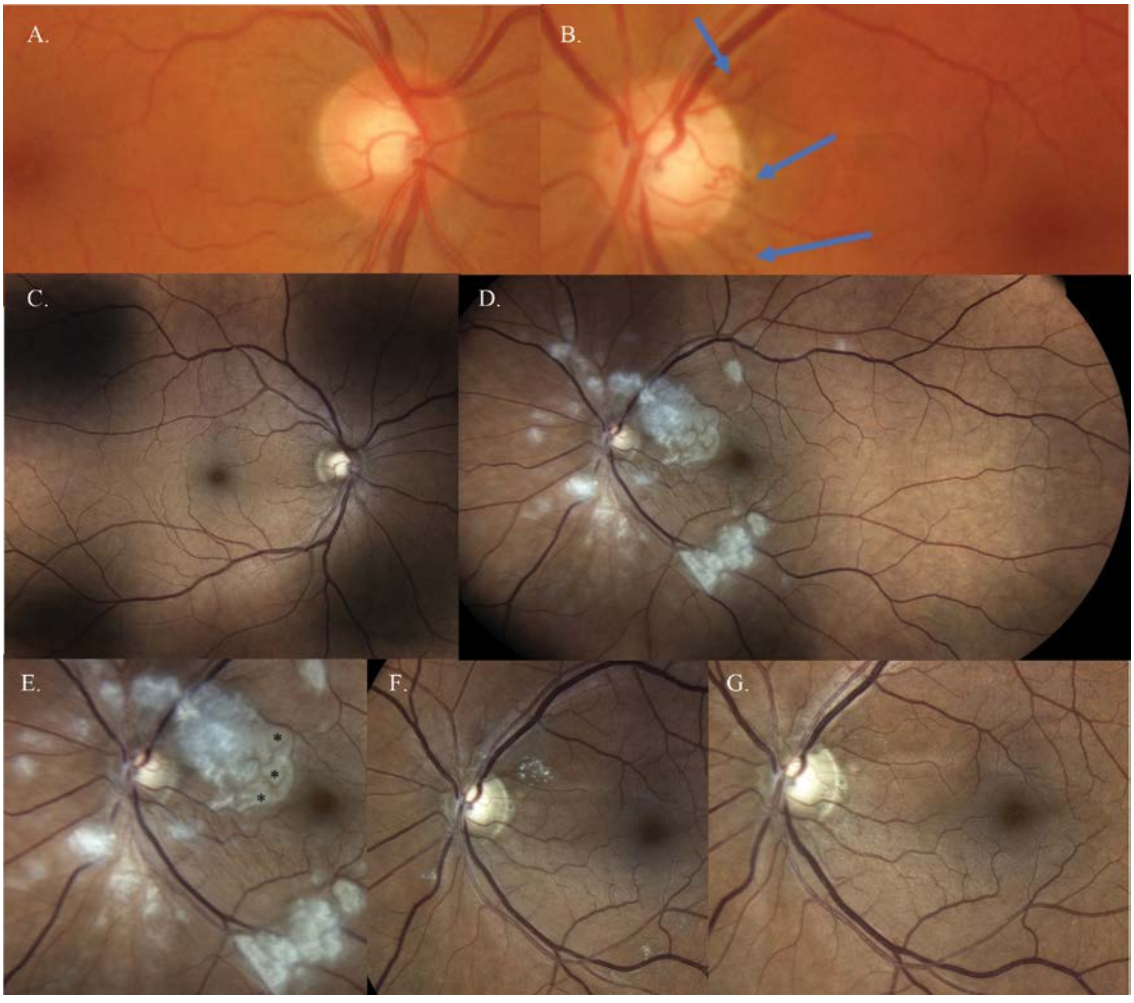


Figure 2: *Overview of reliable Humphrey fields OS at teaching clinic. A) Baseline August 2021 VF with superior nasal step and dense inferior paracentral loss, most obvious on pattern deviation. B) January 2022 follow-up VF, showing stability on pattern deviation plot, visual field index, and mean deviation. C) Latest follow-up VF, showing roughly stable pattern deviation and improved mean deviation and visual field index possibly due to practice.*

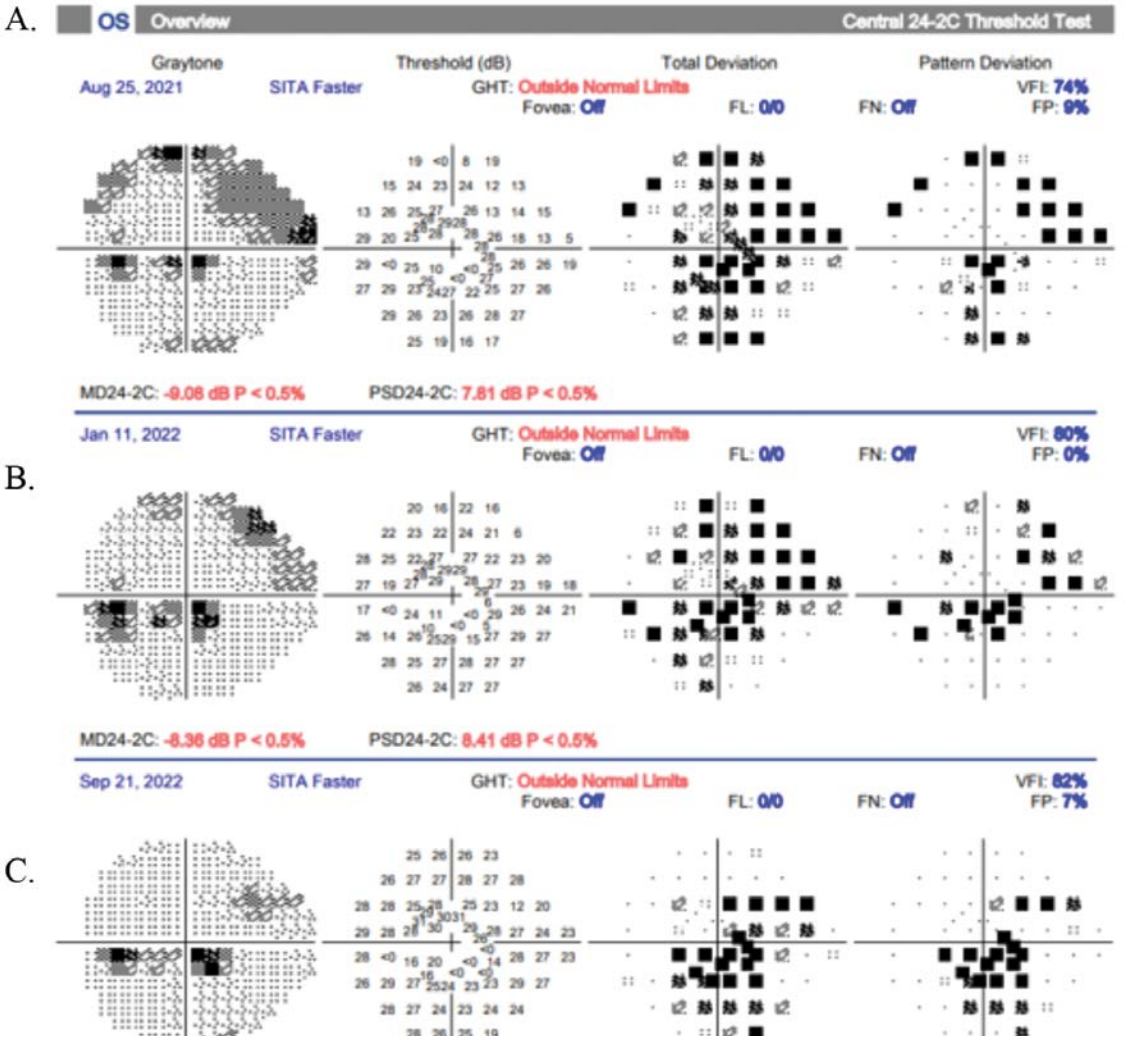


Figure 3: *OCT and OCT-A imaging at presentation to teaching clinic. A) Cirrus RGC analysis OU B) Cirrus RNFL and optic nerve head analysis OU. Note very thin (dark blue) superior-nasal region of OS thickness map, accounting for low minimum RGC thickness OS of 32 um. C) En-face analysis of superficial retinal circulation OS on OCT-A showing dark areas superior nasal macula (D shaped blue circle) and inferior temporal arcades (red circle). Dark areas represent lack of blood flow through missing capillary beds. D) En-face analysis of deep retinal circulation OS showing capillary bed loss in same inter-arterial areas. E) Lack of blood flow (red pixels) through atrophied inner-retinal area in the superior nasal macula (left portion of picture), again suggestive of prior vascular-occlusive event. The cross-sectional B scan slice used for E is marked by the horizontal turquoise colored line in C & D.*

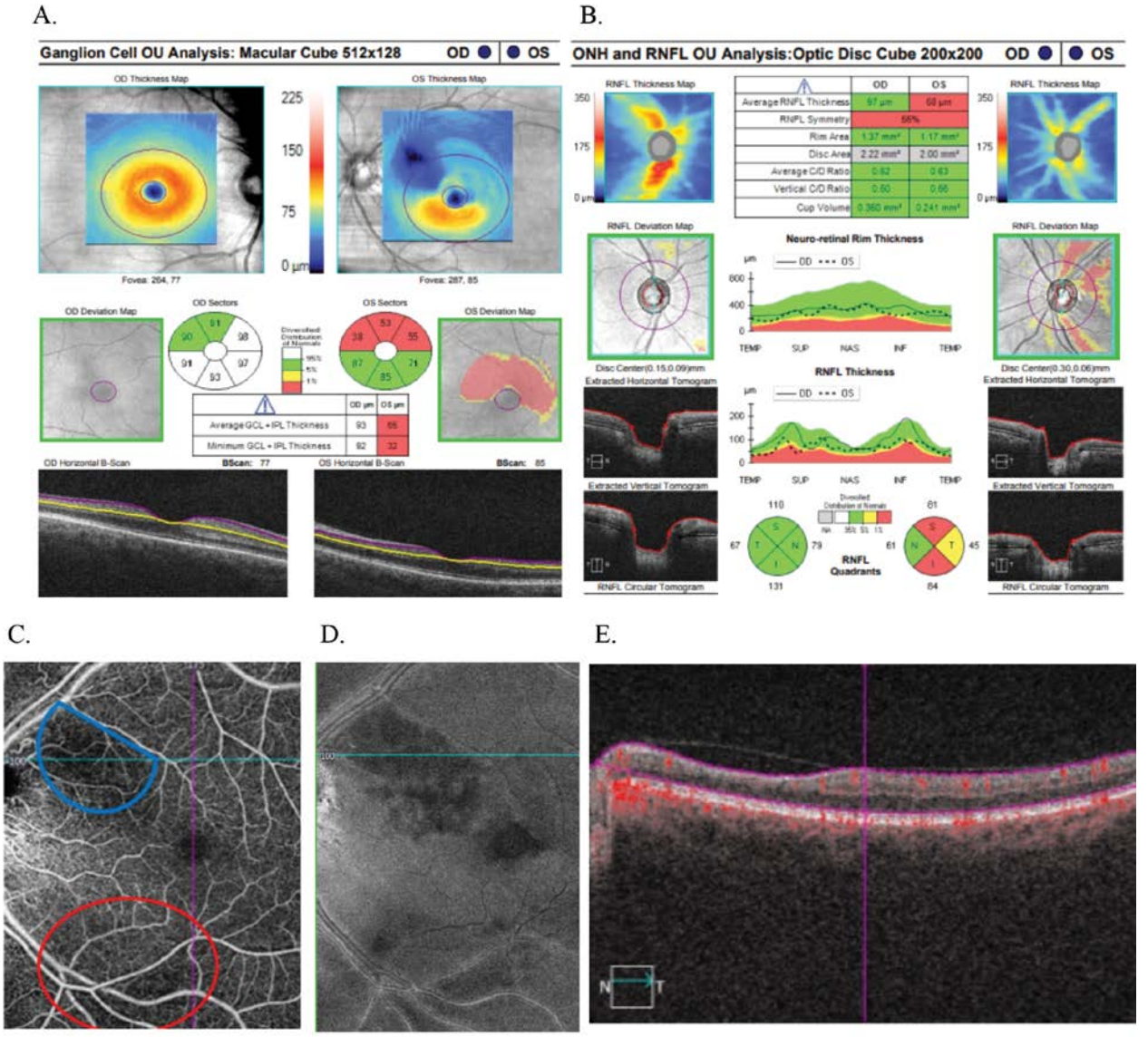


Figure 4: *A) Spectralis RNFL TSNIT plot OS at teaching clinic baseline (light grey) and latest follow-up (black), showing lack of progression. B) RNFL progression analysis showing average/global RNFL of 3.5 mm diameter circle roughly stable at Spectralis RNFL floor (~ 50 um). C) Minimum rim width progression analysis OS shows average neuro-retinal rim thickness is stable thus far over one year plus of follow-up. D) Spectralis Minimum rim width consists of 48 different measurements of the perpendicular distance (turquoise colored arrows) between bruch's membrane opening (red dot) and internal limiting membrane (red line) via 24 different radial nerve B scans. In bottom right TSNIT style plot, note that the latest follow up scan (dark black) shows roughly equivalent thickness to baseline (light grey) if some noise due to measurement variability is acknowledged.*

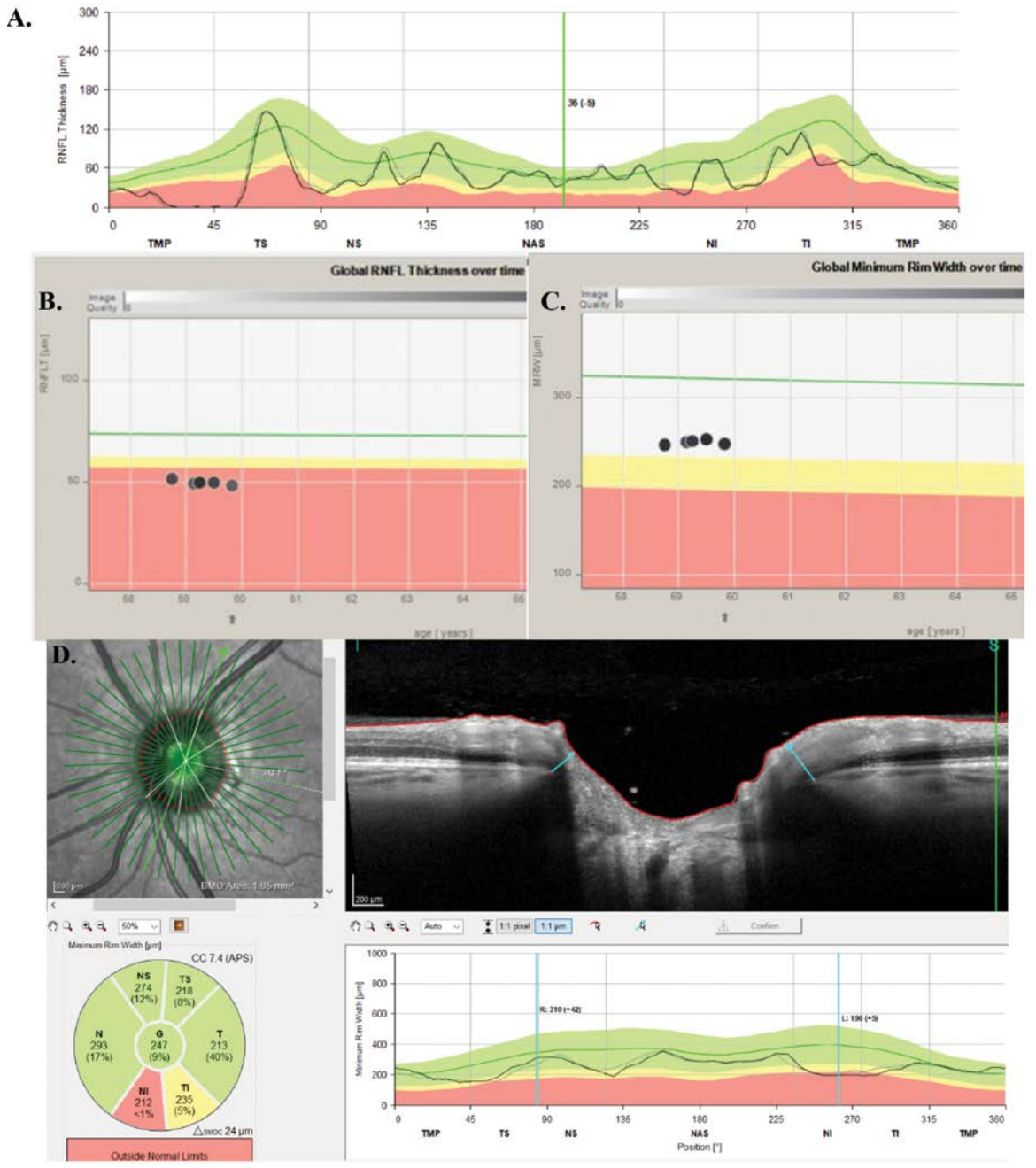
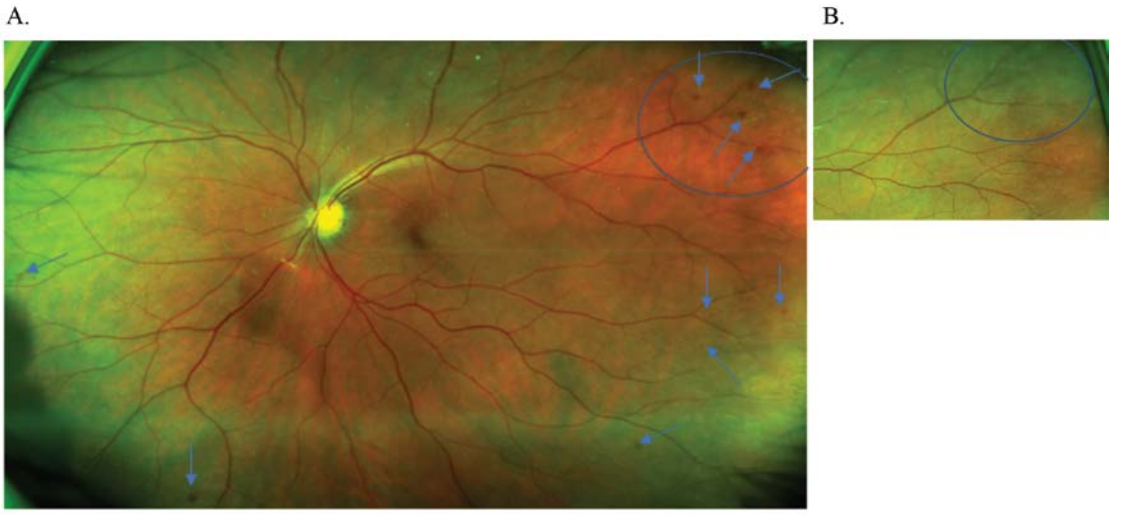


Figure 5: *A) Wide field retinal imaging OS at latest follow up. Blue arrows show ten mid-peripheral dot-blot hemorrhages that are new findings (not residual post-iCRAO event). B) Snippet of superior-temporal portion of wide-field imaging taken 5 months before A. Blue circle represents approximately the same retinal area in both A and B. Note hemorrhages are not present in B.*



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