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Longitudinal retinal and choroidal microvascular changes in carotid artery stenosis: Comparative outcomes of surgical and medical management using optical coherence tomography angiography

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Abstract

Background & Objective: Carotid artery stenosis (CAS) is a major cause of chronic cerebral hypoperfusion and ischemic stroke. Because retinal circulation originates from the internal carotid artery, optical coherence tomography angiography (OCTA) may serve as a non-invasive surrogate marker of cerebral microvascular compromise. *Methods:* Forty-six patients with CAS (92 eyes) and 41 healthy controls (41 eyes) were enrolled. Ipsilateral stenotic eyes were classified as Group 1, contralateral fellow eyes as Group 2, and controls as Group 3. Stenotic eyes were further subdivided into surgically treated (Group 1A) and medically managed (Group 1B) subgroups. Layer-specific OCTA and structural OCT parameters were assessed at baseline and at 1, 3, and 6 months. The predefined primary outcome was deep capillary plexus (DCP) inner vessel and perfusion density. *Results:* At baseline, stenotic eyes demonstrated significantly reduced vessel and perfusion density across retinal and choroidal layers compared with controls (all $p < 0.05$). During follow-up, surgically treated eyes showed significant recovery in DCP inner metrics, whereas medically managed eyes exhibited smaller and less consistent changes. Several secondary choroidal parameters also improved gradually after revascularization. By month 6, multiple vascular indices in Group 1A approached values observed in contralateral non-stenotic eyes. Structural measures (RNFL and GCC thickness) showed modest, delayed improvement predominantly in the surgical subgroup.
Conclusions: CAS is associated with diffuse retinal and choroidal microvascular impairment. OCTA-derived parameters, particularly deep capillary plexus metrics, may represent accessible biomarkers of downstream hemodynamic compromise and microvascular recovery following intervention.

Keywords: Carotid artery stenosis, OCT angiography, cerebral hypoperfusion, treatment response, stroke risk.

INTRODUCTION

Carotid artery stenosis is a major cause of chronic cerebral hypoperfusion and remains one of the most important modifiable risk factors for ischemic stroke.[1,2] In clinical neurology, disease severity and treatment decisions are largely guided by the degree of luminal narrowing and neurovascular imaging findings. However, increasing evidence suggests that the biological impact of carotid disease cannot be fully explained by macroscopic stenosis severity alone, as downstream microvascular dysfunction may persist even in the absence of overt neurological events.[1-3]

Because the ophthalmic artery originates directly from the internal carotid artery, the retinal and choroidal circulations represent accessible microvascular territories that are directly exposed to carotid hemodynamic compromise.[3,4] In this sense, the eye may serve as a unique "window" into systemic and cerebrovascular

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microcirculatory health, providing end-organ information that complements conventional neuroimaging.[4,5] Identifying such microvascular biomarkers is increasingly relevant in cerebrovascular medicine, where early detection of hypoperfusion-related injury and longitudinal monitoring of treatment response remain clinical challenges. Given the substantial stroke burden in Asian populations, accessible markers of microvascular hypoperfusion may have particular clinical relevance.

Optical coherence tomography angiography has emerged as a non-invasive modality that enables depth-resolved visualization of retinal and choroidal microvasculature without dye injection.[6] Cross-sectional OCTA studies have reported that CAS is associated with enlargement of the foveal avascular zone and reductions in vessel density and perfusion density across both superficial and deep retinal capillary plexuses.[1,7-10] These observations support the concept that carotid hypoperfusion produces quantifiable microvascular alterations in the ocular circulation. Nevertheless, most available studies are limited by single time-point designs, heterogeneous patient cohorts, and a predominant focus on retinal circulation, leaving the longitudinal course of microvascular recovery after treatment insufficiently characterized. In clinical neurology, a major challenge in the management of carotid artery stenosis is the identification of patients with subclinical hemodynamic compromise who may remain neurologically asymptomatic yet harbor progressive microvascular dysfunction. Conventional imaging primarily evaluates macroscopic luminal narrowing and plaque characteristics; however, microcirculatory impairment may precede overt ischemic events. Identifying accessible biomarkers that reflect cerebral perfusion status remains an unmet need in cerebrovascular medicine.[9,11-13]

From a neurovascular standpoint, this knowledge gap is clinically important. Carotid revascularization procedures such as endarterectomy or stenting are performed to reduce stroke risk and restore cerebral perfusion, yet their downstream effects on microvascular beds remain less well defined.[2,4] In this context, OCTA may offer a practical adjunct approach for monitoring microvascular remodeling over time and for comparing recovery patterns between surgical and conservative management strategies. Given the sensitivity of the deep retinal plexus to chronic low-flow states, deep capillary plexus (DCP) metrics may represent particularly responsive indicators of treatment-related microvascular change.

In addition, vascular layers such as the choriocapillaris and choroid, which have limited autoregulatory reserve, may be especially vulnerable to systemic hypoperfusion but remain underexplored in longitudinal OCTA studies of carotid disease.[4,8,14,15] Therefore, choroidal circulation may provide clinically relevant secondary information regarding systemic microvascular adaptation beyond retinal findings alone. Structural retinal measures, including retinal nerve fiber layer (RNFL) and ganglion cell complex (GCC) thickness, may further offer complementary insight into neuroretinal integrity and its potential responsiveness to improved perfusion.[16-24] Together, integrated vascular and structural assessment may enhance understanding of neurovascular coupling and chronic ischemic stress in carotid stenosis.

Therefore, the present study aimed to longitudinally evaluate layer-specific retinal and choroidal microvascular changes using OCTA in patients with carotid artery stenosis and to compare surgically treated, medically managed, and healthy control eyes. By integrating vascular and structural parameters over a six-month follow-up period, we sought to clarify the temporal relationship between carotid revascularization, ocular microcirculation, and neuroretinal structural changes. In addition, this study explores the potential clinical role of OCTA as a complementary imaging tool in the multidisciplinary evaluation of CAS, providing microvascular insights that may extend beyond stenosis severity alone.

METHODS

This prospective, longitudinal case–control study was conducted between November 2023 and September 2024 at the Departments of Ophthalmology, Neurology, and Cardiovascular Surgery. Patients diagnosed with CAS and scheduled for either surgical revascularization or medical management were consecutively recruited prior to initiation of treatment. Neurological status and indication for intervention were determined by a multidisciplinary team including neurologists and cardiovascular surgeons in accordance with contemporary cerebrovascular guidelines. All participants underwent comprehensive ophthalmological evaluation and OCTA imaging at baseline and at 1, 3, and 6 months after treatment.

Follow-up intervals were predefined to capture early postoperative hemodynamic fluctuations (1 month), intermediate vascular adaptation (3 months), and longer-term microvascular remodeling (6 months).

Demographic data and systemic comorbidities, including hypertension, diabetes mellitus, and hyperlipidemia, were recorded for all participants.

The study protocol was approved by the Clinical Research Ethics Committee of Çanakkale Onsekiz Mart University (Decision date: 29 November 2023; Decision number: 2023/15-04; Project number: 2023-YONP-180). All procedures adhered to the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to enrollment.

Inclusion criteria were age >18 years, diagnosis of carotid artery stenosis confirmed by Doppler ultrasonography or computed tomography angiography, and sufficient image quality for quantitative OCTA analysis.

Exclusion criteria included any retinal vascular disease (diabetic or hypertensive retinopathy), glaucoma or optic neuropathy, amblyopia, neurodegenerative disorders, previous ocular trauma or surgery (except uncomplicated cataract extraction), refractive error exceeding ±3.00 diopters, and poor-quality OCTA scans (signal strength index <7/10). Images with motion artifacts or segmentation errors were excluded from analysis.

Participants were categorized into three groups: ipsilateral stenotic eyes (Group 1), contralateral non-stenotic fellow eyes (Group 2), and age- and sex-matched healthy control eyes (Group 3). Stenotic eyes were further subdivided into surgically treated (Group 1A) and medically managed (Group 1B) subgroups.

To minimize inter-eye dependency, only one eye per participant was included in the healthy control group. In patients with carotid artery stenosis, ipsilateral and contralateral eyes were analyzed separately within predefined groups, and results were interpreted with caution given shared systemic exposure.

Indications for carotid endarterectomy included ≥50% symptomatic or ≥70% asymptomatic internal carotid artery stenosis, in accordance with established clinical guidelines. Surgical procedures were performed under standardized conditions. Medically managed patients received antiplatelet therapy based on routine clinical indications, including aspirin, clopidogrel, or combination therapy.[25,26]

As treatment allocation was not randomized and antiplatelet regimens were not stratified by drug type, subgroup analyses based on specific medical therapies were not performed.

All OCTA examinations were performed using the Nidek RS-3000 Advance device (Nidek Co., Ltd., Japan) with a standardized 6 × 6 mm macular scan protocol. Vessel density (VD), perfusion density (PD), and foveal avascular zone (FAZ) area and perimeter were quantified for the superficial capillary plexus (SCP), DCP, choriocapillaris (CC), and choroid. Structural optical coherence tomography (OCT) parameters included retinal nerve fiber layer (RNFL) and ganglion cell complex (GCC) thickness. Layer segmentation and quantitative measurements were generated using the device's built-in software. Inner segments corresponded to the 1–3 mm annulus and outer segments to the 3–6 mm annulus of the ETDRS grid.

All scans were acquired by the same experienced examiner. Inter-observer reproducibility was assessed in a randomly selected subset of scans, yielding an intraclass correlation coefficient greater than 0.90 for all measured parameters.

Statistical analyses were performed using SPSS version 27.0 (IBM Corp., Chicago, IL, USA). Data distribution was assessed using the Shapiro–Wilk test. Normally distributed variables were expressed as mean ± standard deviation, whereas non-normally distributed variables were presented as median (minimum–maximum).

Baseline intergroup comparisons were performed using the Mann–Whitney U test. Longitudinal changes within groups were evaluated using repeated-measures ANOVA or the Friedman test, as appropriate.

Given the exploratory nature of this longitudinal study and the interrelated structure of OCTA parameters, no formal correction for multiple comparisons was applied. As the predefined primary endpoint was DCP inner vessel and perfusion density, secondary analyses were considered exploratory and interpreted cautiously.

RESULTS

A total of 46 patients with CAS (92 eyes) and 41 healthy control subjects (41 eyes) were included. Ipsilateral stenotic eyes were classified as Group 1, contralateral non-stenotic fellow eyes

as Group 2, and eyes of healthy participants as Group 3. Stenotic eyes were further subdivided into surgically treated (Group 1A) and medically managed (Group 1B) subgroups. Among the 46 patients with carotid artery stenosis, 8 underwent Group 1A and 38 received Group 1B. This distribution reflects real-world clinical decision-making guided by stenosis severity and symptom status rather than randomized assignment. Baseline demographic characteristics and systemic comorbidities are summarized in Table 1. There were no significant differences in age or sex distribution among the groups.

Baseline OCTA and structural differences

At baseline, ipsilateral stenotic eyes (Group 1) demonstrated significantly reduced VD and PD across retinal and choroidal vascular layers compared with healthy controls (Group 3) (all $p < 0.05$). Contralateral non-stenotic eyes (Group 2) generally showed intermediate values between stenotic and control eyes. (Tables 2-4, Supplementary (S) Tables 1-11)

Structural OCT analysis revealed significantly thinner RNFL and GCC measurements in stenotic eyes compared with controls at baseline (all $p < 0.05$). Detailed baseline values are provided in Tables S1–S11.

Table 1: Demographic characteristics of study participants

Group	Male, n (%)	Female, n (%)	Mean Age (Male)	Mean Age (Female)	Clinical Parameters	Male (n)	Female (n)
Group 1 (Ipsilateral stenotic eye)	27 (58.7%)	19 (41.3%)	66	71	Treatment - Aspirin	8	6
					Treatment - Clopidogrel	12	4
					Treatment - Aspirin + Clopidogrel	2	6
					Treatment - Carotid Endarterectomy	5	3
					Hypertension - Absent	16	0
					Hypertension -Present	11	19
					Diabetes Mellitus - Absent	23	10
					Diabetes Mellitus - Present	4	9
					Hyperlipidemia - Absent	17	6
					Hyperlipidemia - Present	10	13
					Coronary Artery Disease- Absent	13	13
					Coronary Artery Disease- Present	14	6
Group 2 (Contralateral Eye)							
Group 3 (Healthy Controls)	19 (46.3%)	22 (53.7%)	56	57			

n, number; Group 2 shares the same systemic characteristics as Group 1.

Primary outcome: Deep capillary plexus recovery

The predefined primary OCTA outcome was DCP inner vessel and perfusion density. At baseline, DCP inner VD and PD were significantly lower in stenotic eyes than in healthy controls. During follow-up, surgically treated eyes (Group 1A) demonstrated significant improvement in DCP inner metrics across baseline, 1-, 3-, and 6-month visits, whereas medically managed eyes (Group 1B) showed smaller and less consistent changes. Primary outcome data are presented in Tables 2-4. The magnitude of DCP recovery in surgically treated eyes paralleled the expected hemodynamic improvement following revascularization, supporting its potential relevance as an indirect marker of cerebral perfusion restoration.

Longitudinal OCTA changes

Significant temporal variation was observed in multiple secondary OCTA parameters among stenotic eyes. In Group 1A, VD and PD values across the SCP, choriocapillaris, and choroidal layers differed significantly over time (all $p < 0.05$). Similar trends were observed in Group 1B but reached statistical significance in fewer parameters.

FAZ area and perimeter measurements also showed significant variation during follow-up in stenotic eyes, whereas contralateral non-stenotic eyes and healthy controls demonstrated stable

Table 2: DCP vessel and perfusion density comparison (inner segments)

Parameter	Time Point	Group 1A	Group 1B	Group 2	Group 3	p-value (Overall)	Post hoc Summary
DCP Inner Segment Vessel Density	Baseline (0 month)	2.0 ± 0.4	2.2 ± 0.5	2.8 ± 0.6	3.1 ± 0.5	0.01	G3 > G2 > G1B > G1A
	1 Month	2.1 ± 0.4	2.3 ± 0.5	2.9 ± 0.6	3.2 ± 0.5	0.02	Mild early increase in G1A/B
	3 Months	1.9 ± 0.4	2.1 ± 0.5	2.8 ± 0.5	3.1 ± 0.4	0.008	G3 > G2 > G1A/B
	6 Months	2.6 ± 0.5	2.5 ± 0.5	2.9 ± 0.5	3.2 ± 0.4	0.04	Recovery trend, especially in G1A
DCP Inner Segment Perfusion Density	Baseline (0 month)	7.0 ± 1.0	7.3 ± 1.1	8.2 ± 1.0	8.9 ± 0.9	0.01	G3 > G2 > G1B > G1A
	1 Month	6.5 ± 1.0	6.8 ± 1.0	8.3 ± 0.9	8.9 ± 0.8	0.009	Early decline in G1A/B (p <0.05)
	3 Months	6.8 ± 0.9	7.0 ± 1.0	8.2 ± 0.9	8.8 ± 0.8	0.01	G3 > G2 > G1A/B
	6 Months	8.0 ± 1.0	7.8 ± 1.0	8.4 ± 0.9	8.9 ± 0.8	0.05	Recovery in G1A, nearly approaching G2

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD), n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes; Group 1B = medically managed stenotic eyes; Group 2 = non-stenotic fellow eyes; Group 3 = healthy control eyes, DCP = deep capillary plexus.

Table 3: DCP vessel and perfusion density comparison (Outer segments)

Parameter	Time Point	Group 1A	Group 1B	Group 2	Group 3	p-value (Overall)	Post hoc Summary
DCP Outer Segment Vessel Density	Baseline (0 month)	2.0 ± 0.4	2.2 ± 0.4	2.8 ± 0.5	3.0 ± 0.5	0.01	G3 > G2 > G1B > G1A
	1 Month	1.8 ± 0.4	2.0 ± 0.4	2.9 ± 0.5	3.1 ± 0.5	0.008	Early postoperative drop in G1A/B
	3 Months	2.0 ± 0.4	2.1 ± 0.4	2.8 ± 0.5	3.0 ± 0.5	0.02	G3 > G2 > G1A/B
	6 Months	2.5 ± 0.5	2.4 ± 0.4	2.9 ± 0.5	3.1 ± 0.4	0.05	Significant recovery in G1A ($p < 0.05$)
DCP Outer Segment Perfusion Density	Baseline (0 month)	6.8 ± 0.9	7.0 ± 0.9	8.0 ± 0.8	8.7 ± 0.8	0.01	G3 > G2 > G1B > G1A
	1 Month	6.2 ± 0.9	6.5 ± 0.8	8.1 ± 0.8	8.8 ± 0.7	0.008	Early decline in G1A/B ($p < 0.05$)
	3 Months	6.6 ± 0.8	6.7 ± 0.8	8.0 ± 0.8	8.8 ± 0.7	0.01	G3 > G2 > G1A/B
	6 Months	7.8 ± 0.9	7.5 ± 0.8	8.2 ± 0.8	8.8 ± 0.7	0.05	Recovery in G1A, nearing G2 levels

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD), n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes; Group 1B = medically managed stenotic eyes; Group 2 = non-stenotic fellow eyes; Group 3 = healthy control eyes, DCP = deep capillary plexus.

FAZ metrics throughout the study period. At baseline, FAZ measurements showed modest differences between groups; however, more pronounced temporal variation was observed during longitudinal follow-up in stenotic eyes. OCTA results are detailed in Tables S1–S11.

Treatment subgroup comparison

When treatment strategies were compared, surgically treated eyes exhibited greater microvascular recovery than medically managed eyes, particularly in deep retinal and choroidal circulation. By month 6, several vascular indices in Group 1A approached values observed in contralateral non-stenotic eyes, while Group 1B remained persistently lower than both non-stenotic and healthy control groups. Intergroup comparisons and post hoc analyses are provided in Tables S1–S11.

Structural OCT follow-up

RNFL and GCC thickness measurements demonstrated significant temporal variation in stenotic eyes during follow-up, including a transient early decline in RNFL at 1 month followed by partial recovery over time (all $p < 0.05$). Surgically treated eyes showed more consistent improvement across time points, whereas medically managed eyes exhibited smaller and less consistent structural changes.

Table 4: Summary of OCTA and structural parameters across four study groups

Parameter	Group 1A	Group 1B	Group 2	Group 3	Statistical Trend	Summary Interpretation
DCP Inner VD	Early ↓ → 6M recovery	Mild ↑	Stable	Highest	$p < 0.01$	Deep inner capillary network shows significant revascularization
DCP Inner PD	1M ↓ → progressive ↑	Slight ↑	Stable	Highest	$p < 0.05$	Deep inner perfusion improved after revascularization
DCP Outer VD	1M ↓ → 6M ↑	Stable	Stable	Highest	$p < 0.05$	Outer deep plexus flow normalized with surgery
DCP Outer PD	1M ↓ → 6M ↑	Stable	Stable	Highest	$p < 0.05$	Reperfusion effect prominent at 6M post-surgery

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD). n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes, Group 1B = medically managed stenotic eyes, Group 2 = non-stenotic fellow eyes, Group 3 = healthy control eyes. FAZ = foveal avascular zone, SCP = superficial capillary plexus, DCP = deep capillary plexus, VD = vessel density, PD = perfusion density. ↓=decrease, → = then, ↑= increase.

Contralateral non-stenotic eyes and healthy control eyes maintained stable RNFL and GCC values throughout follow-up. Structural OCT findings are summarized in Table S8.

Overall summary

Across all study groups, ipsilateral stenotic eyes consistently exhibited lower retinal and choroidal vascular parameters compared with contralateral non-stenotic and healthy control eyes. Longitudinal analysis demonstrated significant microvascular recovery following treatment, with greater magnitude observed after surgical revascularization. Summary comparisons across groups are presented in Tables S9-S11.

DISCUSSION

From a cerebrovascular perspective, the present findings suggest that retinal microvascular indices may reflect downstream effects of carotid hypoperfusion and its subsequent reversal following revascularization. Given that the ophthalmic artery arises directly from the internal carotid artery, retinal circulation may represent a readily accessible microvascular territory sharing hemodynamic vulnerability with cerebral tissue. This prospective longitudinal study evaluated retinal and choroidal microvascular alterations in patients with CAS and compared temporal changes following surgical revascularization versus medical therapy using OCTA. The main findings can be summarized as follows: (1) ipsilateral stenotic eyes demonstrated significant baseline impairment across retinal and choroidal vascular layers compared with healthy controls, consistent with OCTA evidence of ocular hypoperfusion in CAS[11,12,27]; (2) the predefined primary outcome, DCP inner vessel and perfusion density, showed the most dynamic longitudinal recovery, particularly after surgical intervention; and (3) choriocapillaris, choroidal, and structural parameters exhibited more gradual and variable changes, providing clinically relevant secondary information on systemic microvascular adaptation.

CAS is a major contributor to chronic cerebral hypoperfusion and ischemic stroke risk. Because the ophthalmic artery originates directly from the internal carotid artery, ocular microcirculation

represents an accessible downstream vascular territory that may reflect systemic hemodynamic compromise. Cross-sectional OCTA studies have consistently demonstrated reduced vessel density, perfusion density, and FAZ alterations in CAS patients.[11,13,27] However, most prior work has relied on single time-point assessment, whereas longitudinal data capturing microvascular remodeling after treatment remain limited.[14,15] Our serial follow-up extends this evidence by demonstrating that microvascular recovery is gradual and differs substantially between surgical and conservative management.

A key contribution of this study is the identification of DCP inner segment metrics as sensitive indicators of ischemic burden and treatment-related recovery. The deep retinal circulation is thought to be particularly vulnerable to chronic low-flow exposure, and DCP parameters have been shown to respond dynamically after carotid intervention.[14,27] In our cohort, surgically treated eyes demonstrated significant improvement in DCP inner vessel and perfusion density over six months, whereas medically managed eyes showed smaller and less consistent changes. These findings align with recent swept-source OCTA studies reporting delayed but progressive deep plexus recovery following revascularization.[7,16] From a neurovascular standpoint, such microvascular responsiveness may provide complementary insight beyond luminal stenosis severity, supporting the potential value of OCTA for monitoring downstream perfusion effects of carotid treatment.

In contrast, SCP changes were more limited over time, suggesting that superficial impairment may represent a more chronic manifestation of cumulative hemodynamic stress rather than an immediately reversible ischemic process. Persistent superficial attenuation after carotid endarterectomy or stenting has been described previously.[4,11] The differential behavior between SCP and DCP reinforces the importance of layer-specific OCTA analysis, as deep plexus indices may better capture dynamic recovery patterns in hypoperfusion states.[12,18]

Beyond retinal circulation, the choriocapillaris and choroid provide clinically relevant secondary information. These vascular layers have limited autoregulatory reserve and may be particularly susceptible to systemic hypoperfusion.[17,28] Several studies have reported improvement in choroidal or choriocapillaris flow after carotid revascularization[17,28] whereas others have emphasized early postoperative fluctuations related to reperfusion stress and vascular adaptation.[16,29] Our longitudinal findings support a gradual remodeling process, with secondary choroidal recovery occurring alongside deep retinal improvement. Plaque-related factors such as residual stenosis and plaque characteristics may further contribute to variability in downstream microvascular response.[13,28]

Structural OCT measures offered additional context regarding neurovascular coupling. Baseline RNFL and GCC thinning in stenotic eyes is consistent with chronic ischemic susceptibility described in CAS cohorts.[11,13] Over follow-up, structural changes were modest and appeared later than vascular recovery, particularly in surgically treated eyes, paralleling reports of partial neuroretinal improvement after revascularization.[1,16] This temporal dissociation supports the concept that microvascular dysfunction may precede measurable neuroretinal remodeling. Associations between retinal structure and cerebral hemodynamic parameters further reinforce the relevance of ocular biomarkers in systemic hypoperfusion states.[13,23]

From a neurological perspective, these findings have multidisciplinary implications. CAS management is primarily guided by cerebrovascular imaging and neurological risk stratification; however, microvascular consequences may not be fully captured by stenosis severity alone. OCTA offers a rapid and repeatable method to quantify downstream microvascular alterations that may complement established vascular assessments. Recent translational work suggests that integrating retinal neurovascular changes with carotid metrics may enhance prediction of ischemic risk and treatment response.[5] In this context, longitudinal OCTA monitoring may provide supportive information after carotid endarterectomy or stenting, as well as during conservative management.

Several limitations should be acknowledged. The study was conducted at a single center with a modest sample size, and follow-up was limited to six months. OCTA measurements are influenced by image quality and device-specific algorithms despite standardized protocols. Systemic factors such as blood pressure variability and medication adherence were not continuously monitored. The absence of direct cerebral perfusion imaging (such as CT or MR perfusion) and standardized neurological outcome measures limits the ability to directly correlate retinal findings

with clinical cerebrovascular endpoints. Future studies integrating retinal OCTA with cerebral hemodynamic metrics and longitudinal stroke outcomes are warranted. Finally, given the exploratory longitudinal design, outcomes were interpreted cautiously within the context of multiple interrelated vascular parameters.

In conclusion, this longitudinal OCTA study demonstrates that carotid artery stenosis is associated with diffuse retinal and choroidal microvascular impairment and that surgical revascularization results in greater longitudinal recovery than medical therapy. The predefined primary outcome, DCP inner vessel and perfusion density, emerged as a particularly sensitive marker of ischemic burden and delayed remodeling, while choroidal parameters provided clinically relevant secondary insight into systemic microvascular adaptation. These findings suggest that OCTA-derived retinal microvascular parameters may serve as accessible surrogate markers of hemodynamic compromise and recovery in carotid artery stenosis. While not replacing established cerebrovascular imaging modalities, retinal OCTA may provide complementary information regarding microvascular adaptation beyond stenosis severity alone.

DISCLOSURE

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Conflicts of interest: None

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Table S1: FAZ area and perimeter comparison

Parameter	Time Point	Group 1A	Group 1B	Group 2	Group 3	p-value (Overall)	Post hoc Summary
FAZ Area (mm^2)	Baseline (0 month)	0.68 ± 0.11	0.70 ± 0.10	0.63 ± 0.09	0.59 ± 0.08	0.04	G1B > G3 ($p < 0.05$)
	1 Month	0.70 ± 0.10	0.71 ± 0.09	0.65 ± 0.08	0.60 ± 0.07	0.05	G1A/B > G3 (n.s.)
	3 Months	0.66 ± 0.09	0.68 ± 0.09	0.62 ± 0.08	0.59 ± 0.07	0.07	Trend G1B > G3
	6 Months	0.67 ± 0.10	0.69 ± 0.09	0.64 ± 0.09	0.59 ± 0.07	0.06	G1A/B ≈ G2 > G3 (n.s.)
FAZ Perimeter (mm)	Baseline (0 month)	4.88 ± 0.45	5.02 ± 0.48	4.60 ± 0.42	4.42 ± 0.40	0.03	G1B > G3 ($p < 0.05$)
	1 Month	5.10 ± 0.50	5.16 ± 0.46	4.64 ± 0.40	4.41 ± 0.38	0.02	G1A/B > G3 ($p < 0.05$)
	3 Months	4.82 ± 0.44	4.93 ± 0.45	4.58 ± 0.39	4.43 ± 0.36	0.06	Trend toward normalization in G1A
	6 Months	4.86 ± 0.43	4.96 ± 0.44	4.61 ± 0.41	4.44 ± 0.37	0.07	G1A ≈ G1B > G2≈G3 (n.s.)

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD), n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes; Group 1B = medically managed stenotic eyes; Group 2 = non-stenotic fellow eyes; Group 3 = healthy control eyes, FAZ = foveal avascular zone.

Table S2: SCP vessel and perfusion density comparison (inner segments)

Parameter	Time Point	Group 1A (Surgical Stenotic)	Group 1B (Medical Stenotic)	Group 2 (Non-Stenotic Fellow)	Group 3 (Healthy Control)	p-value (Overall)	Post hoc Summary
SCP Inner Segment Vessel Density	Baseline (0 month)	5.0 ± 0.8	5.2 ± 0.7	5.8 ± 0.7	6.3 ± 0.6	0.01	G3 > G2 > G1B > G1A
	1 Month	5.4 ± 0.7	5.5 ± 0.6	5.9 ± 0.6	6.2 ± 0.5	0.03	Mild increase in G1A/B
	3 Months	4.8 ± 0.6	5.0 ± 0.6	5.7 ± 0.7	6.3 ± 0.5	0.008	G3 > G2 > G1A/B
	6 Months	5.7 ± 0.7	5.6 ± 0.6	6.0 ± 0.6	6.4 ± 0.5	0.04	G1A recovery toward G2– G3 levels
SCP Inner Segment Perfusion Density	Baseline (0 month)	25.0 ± 3.5	26.0 ± 3.2	29.0 ± 2.8	31.2 ± 2.5	0.01	G3 > G2 > G1B > G1A
	1 Month	27.5 ± 3.4	27.2 ± 3.1	30.0 ± 2.7	31.1 ± 2.6	0.03	Mild increase in G1A/B
	3 Months	24.0 ± 3.0	25.5 ± 2.9	28.8 ± 2.6	31.0 ± 2.4	0.008	G3 > G2 > G1A/B
	6 Months	28.5 ± 3.3	27.8 ± 3.0	30.5 ± 2.8	31.3 ± 2.5	0.04	G1A recovery toward G2– G3 levels

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD), n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes; Group 1B = medically managed stenotic eyes; Group 2 = non-stenotic fellow eyes; Group 3 = healthy control eyes, SCP = superficial capillary plexus.

Table S3: SCP vessel and perfusion density comparison (outer segments)

Parameter	Time Point	Group 1A	Group 1B	Group 2	Group 3	p-value (Overall)	Post hoc Summary
SCP Outer Segment Vessel Density	Baseline (0 month)	6.0 ± 0.9	6.2 ± 0.8	6.8 ± 0.7	7.3 ± 0.6	0.02	G3 > G2 > G1B > G1A
	1 Month	5.6 ± 0.8	5.8 ± 0.7	6.9 ± 0.6	7.3 ± 0.5	0.01	Early decline in G1A/B ($p< 0.05$)
	3 Months	5.9 ± 0.8	6.0 ± 0.8	6.8 ± 0.7	7.2 ± 0.5	0.03	G3 > G2 >G1A/B
	6 Months	6.4 ± 0.8	6.3 ± 0.7	6.9 ± 0.6	7.3 ± 0.5	0.07	G1A recovery trend; nearly normalized
SCP Outer Segment Perfusion Density	Baseline (0 month)	33.0 ± 3.5	34.0 ± 3.2	37.0 ± 2.9	39.0 ± 2.7	0.01	G3 > G2 > G1B > G1A
	1 Month	30.5 ± 3.1	31.0 ± 3.0	36.8 ± 2.8	38.9 ± 2.6	0.009	Early decline in G1A/B ($p< 0.05$)
	3 Months	31.2 ± 3.2	32.5 ± 3.1	36.9 ± 2.7	38.8 ± 2.5	0.02	Persistent reduction in G1A/B
	6 Months	34.8 ± 3.3	34.0 ± 3.0	37.2 ± 2.8	39.1 ± 2.6	0.05	G1A shows recovery, nearing G2 values

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD), n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes; Group 1B = medically managed stenotic eyes; Group 2 = non-stenotic fellow eyes; Group 3 = healthy control eyes, SCP = superficial capillary plexus.

Table S4: Choriocapillaris vessel and perfusion density comparison (inner segments)

Parameter	Time Point	Group 1A (Surgical Stenotic)	Group 1B (Medical Stenotic)	Group 2 (Non-Stenotic Fellow)	Group 3 (Healthy Control)	p-value (Overall)	Post hoc Summary
CC Inner Segment Vessel Density	Baseline (0 month)	4.8 ± 0.7	5.0 ± 0.8	5.6 ± 0.7	5.9 ± 0.6	0.02	G3 > G2 > G1B > G1A
	1 Month	5.1 ± 0.7	5.2 ± 0.7	5.7 ± 0.7	6.0 ± 0.6	0.04	Mild early rise in G1A/B
	3 Months	5.3 ± 0.7	5.4 ± 0.7	5.8 ± 0.6	6.0 ± 0.6	0.06	Steady improvement trend
	6 Months	5.6 ± 0.8	5.3 ± 0.7	5.8 ± 0.6	6.0 ± 0.6	0.05	G1A recovery, nearing G2–G3 values ($p< 0.05$)
CC Inner Segment Perfusion Density	Baseline (0 month)	26.5 ± 3.0	27.0 ± 2.9	29.5 ± 2.7	30.8 ± 2.6	0.02	G3 > G2 > G1B > G1A
	1 Month	27.8 ± 2.8	27.5 ± 2.7	29.6 ± 2.6	30.7 ± 2.5	0.05	Mild early improvement in G1A/B
	3 Months	28.5 ± 2.9	27.8 ± 2.7	29.4 ± 2.6	30.9 ± 2.5	0.06	G1A gradual rise toward G2 levels
	6 Months	29.2 ± 3.0	28.0 ± 2.8	29.6 ± 2.7	30.8 ± 2.5	0.04	Significant improvement in G1A ($p <0.05$)

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD), n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes; Group 1B = medically managed stenotic eyes; Group 2 = non-stenotic fellow eyes; Group 3 = healthy control eyes, CC=Choriocapillaris.

Table S5: Choriocapillaris vessel and perfusion density comparison (outer segments)

Parameter	Time Point	Group 1A	Group 1B	Group 2	Group 3	p-value (Overall)	Post hoc Summary
CC Outer Segment Vessel Density	Baseline (0 month)	5.0 ± 0.7	5.2 ± 0.6	5.8 ± 0.6	6.1 ± 0.5	0.02	G3 > G2 > G1B > G1A
	1 Month	4.6 ± 0.6	4.8 ± 0.6	5.9 ± 0.6	6.1 ± 0.5	0.008	Early decline in G1A/B ($p < 0.05$)
	3 Months	4.9 ± 0.6	5.0 ± 0.6	5.8 ± 0.6	6.0 ± 0.5	0.03	G3 > G2 >G1A/B
	6 Months	5.5 ± 0.7	5.3 ± 0.6	5.8 ± 0.6	6.1 ± 0.5	0.05	Recovery trend in G1A, approaching G2 levels
CC Outer Segment Perfusion Density	Baseline (0 month)	25.8 ± 2.9	26.2 ± 2.8	28.6 ± 2.7	30.1 ± 2.5	0.02	G3 > G2 > G1B > G1A
	1 Month	24.2 ± 2.8	24.8 ± 2.7	28.5 ± 2.6	30.0 ± 2.4	0.009	Early post-operative decline in G1A/B
	3 Months	25.0 ± 2.7	25.3 ± 2.6	28.7 ± 2.6	30.1 ± 2.4	0.03	G3 > G2 > G1A/B
	6 Months	27.4 ± 2.8	26.9 ± 2.7	28.8 ± 2.6	30.2 ± 2.4	0.05	G1A recovery trend ($p < 0.05$)

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD), n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes; Group 1B = medically managed stenotic eyes; Group 2 = non-stenotic fellow eyes; Group 3 = healthy control eyes, CC=Choriocapillaris.

Table S6: Choroidal vessel and perfusion density comparison (inner segments)

Parameter	Time Point	Group 1A	Group 1B	Group 2	Group 3	p-value (Overall)	Post hoc Summary
Choroid Inner Segment Vessel Density	Baseline (0 month)	3.8 ± 0.6	3.9 ± 0.6	4.4 ± 0.6	4.7 ± 0.5	0.02	G3 > G2 > G1B > G1A
	1 Month	3.6 ± 0.6	3.8 ± 0.5	4.5 ± 0.6	4.8 ± 0.5	0.01	Early decline in G1A/B ($p < 0.05$)
	3 Months	3.9 ± 0.6	3.9 ± 0.5	4.5 ± 0.5	4.8 ± 0.5	0.03	G3 > G2 > G1A/B
	6 Months	4.4 ± 0.6	4.2 ± 0.5	4.5 ± 0.5	4.8 ± 0.4	0.05	Recovery in G1A, near normalization ($p < 0.05$)
Choroid Inner Segment Perfusion Density	Baseline (0 month)	26.0 ± 3.0	26.8 ± 2.9	28.9 ± 2.8	30.4 ± 2.6	0.02	G3 > G2 > G1B > G1A
	1 Month	25.0 ± 2.8	25.8 ± 2.7	29.0 ± 2.7	30.5 ± 2.6	0.01	Early reduction in G1A/B ($p < 0.05$)
	3 Months	26.7 ± 2.9	26.5 ± 2.8	29.0 ± 2.7	30.3 ± 2.5	0.04	Gradual improvement in G1A
	6 Months	28.5 ± 2.8	27.6 ± 2.7	29.1 ± 2.6	30.4 ± 2.5	0.05	Recovery trend in G1A ($p < 0.05$)

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD), n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes; Group 1B = medically managed stenotic eyes; Group 2 = non-stenotic fellow eyes; Group 3 = healthy control eyes.

Table S7: Choroidal vessel and perfusion density comparison (outer segments)

Parameter	Time Point	Group 1A	Group 1B	Group 2	Group 3	p-value (Overall)	Post hoc Summary
Choroid Outer Segment Vessel Density	Baseline (0 month)	4.2 ± 0.6	4.4 ± 0.6	4.8 ± 0.5	5.1 ± 0.5	0.02	G3 > G2 > G1B > G1A
	1 Month	3.9 ± 0.5	4.1 ± 0.5	4.9 ± 0.5	5.1 ± 0.5	0.009	Early reduction in G1A/B ($p < 0.05$)
	3 Months	4.3 ± 0.5	4.2 ± 0.5	4.9 ± 0.5	5.1 ± 0.4	0.03	Gradual recovery, G3 > G2 > G1A/B
	6 Months	4.7 ± 0.5	4.5 ± 0.5	4.9 ± 0.5	5.1 ± 0.4	0.05	G1A recovery toward G2–G3 ($p <0.05$)
Choroid Outer Segment Perfusion Density	Baseline (0 month)	25.5 ± 2.8	26.0 ± 2.8	28.3 ± 2.7	29.8 ± 2.6	0.02	G3 > G2 > G1B > G1A
	1 Month	24.0 ± 2.7	24.5 ± 2.6	28.4 ± 2.6	29.9 ± 2.5	0.009	Early decline in G1A/B ($p< 0.05$)
	3 Months	25.2 ± 2.6	25.4 ± 2.6	28.5 ± 2.5	29.8 ± 2.4	0.03	Gradual improvement trend
	6 Months	27.5 ± 2.7	26.8 ± 2.6	28.6 ± 2.6	29.9 ± 2.4	0.05	G1A recovery nearing G2–G3 levels

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD), n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes; Group 1B = medically managed stenotic eyes; Group 2 = non-stenotic fellow eyes; Group 3 = healthy control eyes.

Table S8: Comparison of RNFL and GCC thickness

Parameter	Time Point	Group 1A	Group 1B	Group 2	Group 3	p-value (Overall)	Post hoc Summary
RNFL	Baseline (0 month)	91.0 ± 8.0	92.5 ± 7.8	98.0 ± 7.5	100.2 ± 7.0	0.02	G3 > G2 > G1B > G1A
	1 Month	89.0 ± 7.5	91.2 ± 7.4	97.8 ± 7.2	100.0 ± 6.8	0.01	Early thinning in G1A/B ($p < 0.05$)
	3 Months	90.5 ± 7.7	91.0 ± 7.3	97.5 ± 7.0	100.1 ± 6.9	0.03	Stable or slight increase in G1A
	6 Months	94.0 ± 7.6	93.5 ± 7.2	98.1 ± 7.1	100.0 ± 6.8	0.05	G1A shows significant recovery ($p < 0.05$)
GCC	Baseline (0 month)	86.0 ± 6.5	87.2 ± 6.4	91.0 ± 6.0	93.5 ± 5.8	0.02	G3 > G2 > G1B > G1A
	1 Month	88.5 ± 6.3	88.0 ± 6.2	91.2 ± 6.0	93.6 ± 5.8	0.03	Mild early increase in G1A ($p < 0.05$)
	3 Months	87.0 ± 6.4	87.5 ± 6.2	91.1 ± 6.0	93.5 ± 5.7	0.04	Temporary dip in G1A/B; stable in G2, G3
	6 Months	89.8 ± 6.2	88.7 ± 6.1	91.3 ± 5.9	93.7 ± 5.7	0.05	Recovery in G1A; near G2 levels ($p < 0.05$)

Non-normally distributed parameters were expressed as median, minimum, and maximum values with interquartile range (IQR), normally distributed parameters were expressed as mean ± standard deviation (SD), ns = not significant; G = Group, Group 1A: surgically treated stenotic eyes; Group 1B: medically managed stenotic eyes; Group 2: non-stenotic fellow eyes; Group 3: healthy control eyes. RNFL = retinal nerve fiber layer; GCC = ganglion cell complex.

Table S9: Summary of OCTA and structural parameters across four study groups

Parameter	Group 1A	Group 1B	Group 2	Group 3	Statistical Trend	Summary Interpretation
FAZ Area	Mild early enlargement → stabilization	Stable	Stable	Smallest, constant	ns	Slight postoperative FAZ fluctuation limited to stenotic eyes
FAZ Perimeter	Transient increase → normalization	Similar trend	Stable	Smallest, steady	ns	Temporary perifoveal dilation after reperfusion
SCP Inner VD	Early drop → recovery (↑6M)	Stable mild ↑	Stable	Highest	$p < 0.05$	Reversible superficial capillary rarefaction post-surgery
SCP Inner PD	Mild 1M ↑ → 3M ↓ → 6M ↑	Gradual ↑	Stable	Highest	$p < 0.05$	Partial restoration of superficial flow after reperfusion
SCP Outer VD	1M ↓ → 6M ↑	1M ↓ → partial ↑	Stable	Highest	$p < 0.05$	Outer plexus remodeling responsive to improved perfusion
SCP Outer PD	1M ↓ → gradual ↑	Slight ↑	Stable	Highest	$p < 0.05$	Outer superficial flow partially reversible after surgery

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD). n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes, Group 1B = medically managed stenotic eyes, Group 2 = non-stenotic fellow eyes, Group 3 = healthy control eyes. FAZ = foveal avascular zone, SCP = superficial capillary plexus, DCP = deep capillary plexus, VD = vessel density, PD = perfusion density. ↓= decrease , → = then, ↑= increase.

Table S10: Summary of OCTA and structural parameters across four study groups

Parameter	Group 1A (Surgical Stenotic)	Group 1B (Medical Stenotic)	Group 2 (Non-Stenotic Fellow)	Group 3 (Healthy Control)	Statistical Trend	Summary Interpretation
CC Inner VD	Gradual ↑ (6M sig.)	Mild ↑	Stable	Highest	$p < 0.05$	Recovery of choriocapillaris microflow after revascularization
CC Inner PD	Mild early ↑ → stable	Stable ↑	Stable	Highest	$p < 0.05$	Sustained flow improvement at choriocapillaris level
CC Outer VD	Early ↓ → 6M recovery	Early ↓ → partial ↑	Stable	Highest	$p < 0.05$	Outer CC flow reduction reversible post-surgery
CC Outer PD	Early ↓ → 6M ↑	Early ↓ → partial ↑	Stable	Highest	$p < 0.05$	Gradual perfusion normalization after reperfusion

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD). n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes, Group 1B = medically managed stenotic eyes, Group 2 = non-stenotic fellow eyes, Group 3 = healthy control eyes. FAZ = foveal avascular zone, SCP = superficial capillary plexus, DCP = deep capillary plexus, VD = vessel density, PD = perfusion density. ↓=decrease , → = then, ↑= increase.

Table S11: Summary of OCTA and structural parameters across four study groups

Parameter	Group 1A	Group 1B	Group 2	Group 3	Statistical Trend	Summary Interpretation
Choroid Inner VD	Early ↓ → 6M ↑	Slight ↓ → stable	Stable	Highest	$p < 0.05$	Improved choroidal microvascular density post-revascularization
Choroid Inner PD	Early ↓ → gradual ↑	Stable	Stable	Highest	$p < 0.05$	Progressive improvement in inner choroidal flow
Choroid Outer VD	1M ↓ → 6M ↑	Mild ↓ → partial ↑	Stable	Highest	$p < 0.05$	Outer choroidal network shows
Choroid Outer PD	1M ↓ → 6M ↑	Mild ↓ → partial ↑	Stable	Highest	$p < 0.05$	Reestablished choroidal perfusion after carotid intervention
RNFL Thickness	Early thinning → 6M recovery	Stable	Stable	Highest	$p < 0.05$	Structural retinal recovery parallel to vascular improvement
GCC Thickness	Early ↑ → 3M ↓ → 6M ↑	Stable	Stable	Highest	$p < 0.05$	Ganglion complex reflects delayed neurovascular recovery

Non-normally distributed parameters are expressed as median (minimum–maximum) with interquartile range (IQR), while normally distributed parameters are expressed as mean ± standard deviation (SD). n.s. = not significant, G = Group, Group 1A = surgically treated stenotic eyes, Group 1B = medically managed stenotic eyes, Group 2 = non-stenotic fellow eyes, Group 3 = healthy control eyes, CC = Choriocapillaris, RNFL = retinal nerve fiber layer; GCC = ganglion cell complex, VD = vessel density, PD = perfusion density. ↓=decrease , → = then, ↑= increase.