

Postoperative Retinal Artery Occlusion Following Macular Hole Surgery: Risk Factors and OCTA Metrics

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ABSTRACT

Purpose: To describe the clinical features and potential mechanisms of retinal artery occlusion (RAO) after macular hole (MH) surgery.

Methods: A retrospective single-centre review was conducted of consecutive MH repairs performed between January 2015 and August 2025. All 583 eyes underwent 25-gauge pars plana vitrectomy with an inverted internal limiting membrane (ILM) flap and 20% SF6 endotamponade. RAO was observed in 5 patients. Collected variables included best-corrected visual acuity, intraocular pressure (IOP), RAO subtype and time to onset, and OCT and OCTA parameters.

Results: RAO subtypes were CRAO in three eyes and CLRAO in two. Onset occurred within 24 hours in two cases, on day 15 in one case, and on day 30 in two cases. MH closure was achieved in four of five eyes. Central macular thickness decreased from $261.5 \pm 61.3 \mu\text{m}$ to $168.2 \pm 36.6 \mu\text{m}$ ($p < 0.001$). The foveal avascular zone was larger in RAO eyes than in the fellow eye ($589.32 \pm 154.1 \mu\text{m}^2$ vs $317.29 \pm 171.2 \mu\text{m}^2$, $p < 0.001$). Superficial and deep plexus vessel densities were lower in RAO eyes at one month, with no significant interocular differences (all $p > 0.05$).

Conclusions: RAO after MH surgery is rare but vision-threatening. The pathogenesis appears multifactorial and may involve periocular anaesthesia, procedure-related haemodynamics (infusion pressure and gas expansion), and patient vascular risk. Preoperative risk optimisation, careful titration of intraoperative parameters, minimally traumatic ILM peeling, and early postoperative monitoring with IOP and OCT/OCTA are prudent. Prospective multicentre studies are needed.

Keywords: macular hole; optical coherence tomography angiography; pars plana vitrectomy; retinal artery occlusion.

INTRODUCTION

A macular hole (MH) is a full-thickness defect in the retinal layer involving the anatomical fovea, thereby impairing central visual acuity. MH surgery, which combines pars plana vitrectomy (PPV) with gas endotamponade, achieves a high rate of anatomical success.¹ Kelly and Wendel were the first to demonstrate that MHs can be effectively treated with PPV.² MH surgery, based on the combination of PPV, closure of the hole with an internal limiting membrane (ILM) flap, and gas endotamponade, has evolved over the

years through significant technological advancements and now achieves anatomical closure rates of approximately 93–98%, making it one of the most successful procedures in vitreoretinal surgery.^{3–5}

Complications include cataract, elevation of intraocular pressure (IOP), abnormalities of the retinal pigment epithelium, visual field defects, late reopening (recurrence) of a hole that was initially closed successfully, retinal detachment, and endophthalmitis.^{6–10} Nevertheless, although rare, vascular complications that compromise

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perfusion of the retina or optic nerve may occur after this surgery. One such complication, retinal artery occlusion (RAO), typically results in sudden and permanent vision loss. Only a small number of RAO cases have been reported in the literature following vitreoretinal surgery for MH.^{11, 12} In this study, we describe the clinical characteristics of six patients who developed RAO after MH surgery and explore plausible pathophysiological mechanisms. Our goal is to contribute preliminary, hypothesis-generating evidence by outlining associated risk factors and pragmatic preventive considerations for vitreoretinal surgeons.

METHODS

The study was approved by the Akdeniz University Hospital Ethics Committee (approval No. TBAEK-659) and conducted in accordance with the tenets of the Declaration of Helsinki. Owing to its retrospective design, written informed consent was not obtained from the patients.

Between January 2015 and August 2025, the medical records of patients followed in our retina surgery unit who underwent full-thickness MH surgery were retrospectively reviewed. Data from a total of 583 patients were screened, and RAO was identified in 5 cases during follow-up. Recorded variables included demographic characteristics, systemic risk factors, type and concentration of intraocular gas, target infusion pressure during surgery, RAO subtype (central retinal artery occlusion (CRAO) / cilioretinal artery occlusion (CLRAO), time to onset, best-corrected visual acuity (BCVA, logMAR), IOP, and optical coherence tomography (OCT) and OCT angiography (OCT-A) findings.

Surgical Technique

The surgery was performed using the inverted ILM flap technique as described by Michalewska et al.¹³ All procedures were carried out by a single experienced vitreoretinal surgeon (M.E.D.) using a 25-gauge vitrectomy system. Peribulbar anaesthesia (4–5 mL) was administered in all cases with a 1:1 (50%/50%) mixture of prilocaine and bupivacaine. When indicated, phacoemulsification with intraocular lens (IOL) implantation was performed during the same session. After core vitrectomy, posterior hyaloid adherence over the macula was assessed, and a posterior vitreous detachment was induced with triamcinolone assistance using either the vitrectomy cutter or a vitrectomy pick, as appropriate. Central and peripheral vitrectomy

were then completed. The epiretinal membrane (ERM) and ILM were peeled after staining with brilliant blue G when required. For the inverted ILM flap technique, a circular ILM peel was created to leave a hinged flap, which was inverted to cover the MH. Following fluid–air exchange, intraocular air was replaced with 20% sulphur hexafluoride (SF₆) gas as an endotamponade. Postoperatively, all patients were instructed to maintain a face-down (prone) position for at least 7 days.

OCT and OCTA measurements

All examinations were performed using a swept-source OCT system (DRI OCT Triton, Topcon, Japan) with an axial resolution of 8 µm, a digital sampling resolution of 2.6 µm, a 1050 nm light source, and a scan rate of 100,000 A-scans per second. OCTA datasets were processed in IMAGEnet 6 (version 1.24.1.15742) with the 3 × 3 mm SS-OCTA module. To minimise operator-related variability, a single experienced technician acquired all scans according to standardised imaging protocols. The instrument was routinely calibrated in line with the manufacturer's recommendations to ensure consistent measurements over time. In addition, representative OCT B-scan images are provided: a preoperative scan from a patient with a full-thickness MH and a postoperative scan from a patient who developed RAO after surgery (Fig. 1).

En face angiographic datasets were acquired to visualise the retinal and choroidal microvasculature, allowing clear delineation of the superficial and deep capillary plexuses (SCP and DCP). Quantitative parameters, including retinal thickness, foveal avascular zone (FAZ) area, and vessel density (VD), were obtained using the SS-OCT platform (DRI OCT Triton; Topcon, Tokyo, Japan). The central subfield was defined as a fovea-centred 1 mm circle, and the parafoveal region as the surrounding 3 mm annulus subdivided into superior, inferior, temporal, and nasal quadrants. Central macular thickness (CMT) was calculated automatically. VDs of the SCP, DCP were also generated automatically by the device. In contrast, the FAZ area was not provided by the automated software and was delineated manually using the “Area” tool, which calculates the surface of the outlined region; a representative post-RAO image illustrating the FAZ and its area measurement is shown in Fig. 2.

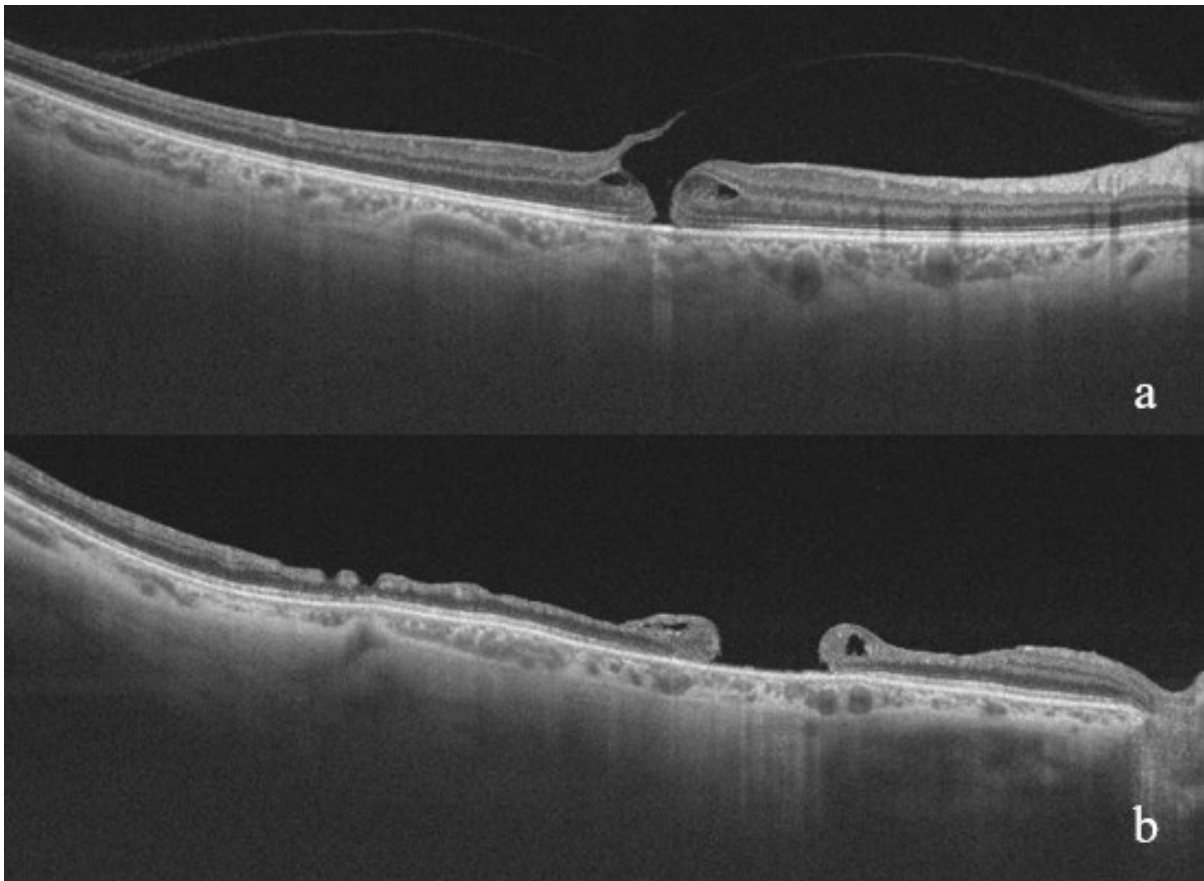


Figure 1. Spectral-domain OCT. (a) Preoperative image showing a full-thickness macular hole with coexisting vitreomacular traction (VMT). (b) At postoperative month 1 following retinal artery occlusion, note diffuse inner retinal atrophy/thinning and a persistent (non-closed) macular hole.

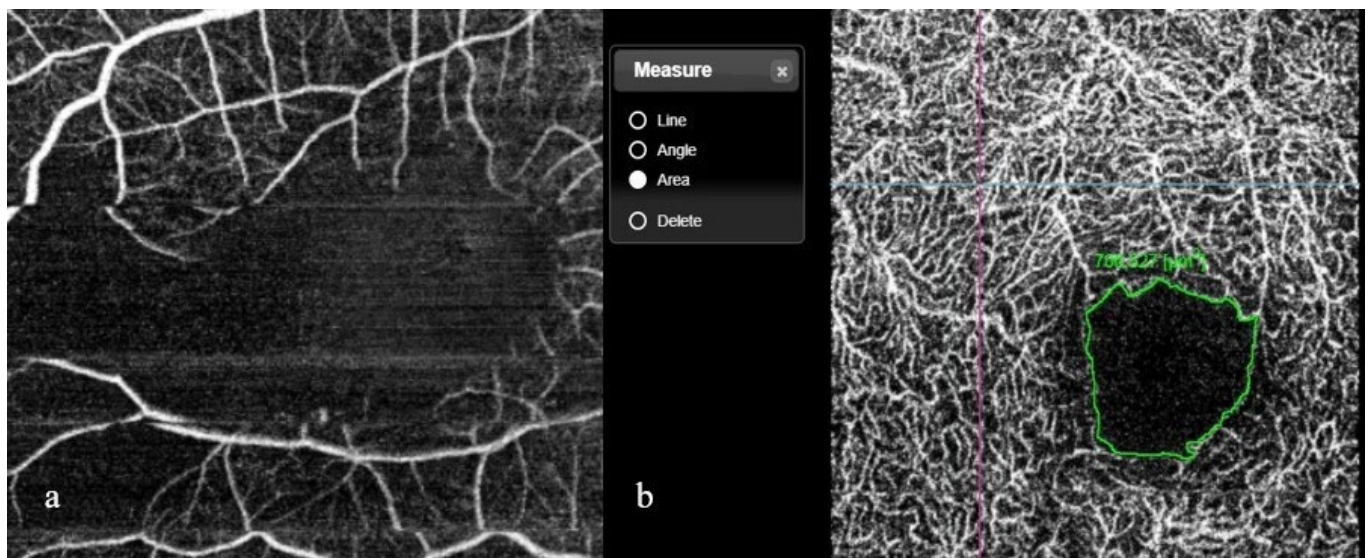


Figure 2. (a) Representative OCTA image demonstrating an enlarged foveal avascular zone (FAZ) after retinal artery occlusion. (b) Manual delineation and area measurement of the FAZ on OCTA (green contour).

Statistical Analysis

Analyses were performed using SPSS Statistics for Windows, version 16.0 (SPSS Inc., Chicago, IL). Normality was assessed with the Shapiro–Wilk test and visual inspection. Data are presented as mean \pm standard deviation (SD) for normally distributed variables and median (range) for non-normally distributed variables. Within-subject interocular and pre- to postoperative comparisons were analyzed using paired t-tests or Wilcoxon signed-rank tests, as appropriate (two-tailed). Categorical variables were compared with Fisher's exact test when applicable. Effect sizes and 95% confidence intervals were reported where relevant. Exact 95% binomial confidence intervals were calculated using the Clopper–Pearson method. Two-tailed p -values < 0.05 were considered statistically significant.

RESULTS

Among 583 MH repairs, RAO occurred in 5 eyes (0.86%; exact 95% CI, 0.28–1.99%). The mean age was 69.2 ± 7.3 years. Of the 5 patients, 3 (60%) were female and 2 (40%) were male. Comorbidities included diabetes mellitus (DM) in 1 patient (20%), hypertension (HT) in 2 (40%), combined HT, hyperlipidaemia, and DM in 1 (20%), and no known systemic disease in 1 (20%). One patient (20%) was an active smoker. No patient had a history of ocular trauma (Table 1).

In all cases, 25-gauge PPV was performed for full-thickness MH, 20% SF6 was used as the endotamponade, and peribulbar anaesthesia was administered as previously described. Coexisting pathologies included ERM in one eye and vitreomacular traction (VMT) in one eye (Fig. 1a). Three patients were pseudophakic; two were phakic and underwent combined phacoemulsification-PPV. The mean MH diameter was 511.3 ± 89.2 μm . The MH closed in four eyes, while one eye showed postoperative non-closure,

indicating surgical failure and yielding an anatomical success rate of 80.0% (4/5; exact 95% CI, 28.4–99.5%) (Fig. 1b) (Tables 1 and 2).

The distribution of RAO subtypes was as follows: CRAO occurred in 3 cases (60%), and CLRAO in 2 cases (40%). Occlusion was recognised within 24 hours postoperatively in 2 cases (40%), on postoperative day 15 in 1 case (20%), and on day 30 in 2 cases (40%). The mean preoperative BCVA was 0.7 ± 0.3 logMAR, declining to 1.2 ± 0.2 logMAR at the final visit (Tables 1 and 2).

The mean preoperative IOP was 15.4 ± 3.1 mmHg, increasing to 16.1 ± 2.1 mmHg at the final visit (Table 2). In one patient (20%), IOP measured 28 mmHg on postoperative day 1 and was controlled with medical therapy; in this case, CLRAO developed on day 15. In all other cases, postoperative IOP values remained within normal limits throughout follow-up.

All cases showed a significant reduction in CMT on OCT (preoperative 261.5 ± 61.3 μm to postoperative 168.2 ± 36.6 μm ; $p < 0.001$). As preoperative and early postoperative OCTA datasets were unavailable, analyses were limited to 1-month postoperative measurements and compared with the fellow (non-RAO) eyes of the same patients. The FAZ area was larger in RAO eyes (SCP FAZ 589.32 ± 154.1 μm^2 vs 317.29 ± 171.2 μm^2 ; $p < 0.001$). There was no significant difference across all parafoveal quadrants: mean VD in the SCP was $43.1 \pm 4.5\%$ in RAO eyes and $44.4 \pm 3.1\%$ in fellow eyes, and in the DCP $47.3 \pm 3.7\%$ and $48.1 \pm 4.2\%$, respectively (all $p > 0.05$) (Table 3).

All patients underwent acute management measures, including ocular massage, anterior chamber paracentesis, antiplatelet therapy, IOP-lowering medication, and, in selected cases, hyperbaric oxygen; however, visual improvement was generally limited.

Table 1. Baseline demographics, ocular characteristics, and OCT findings of patients with retinal artery occlusion after macular hole surgery (n = 5).

Age, years, mean ± SD (range)	69.2 ± 7.3 (66-75)
Sex (male/female), n (%)	2 (40) / 3 (60)
Laterality (left/right), n (%)	2 (40) / 3 (60)
Lens status	
<i>Phakic, n (%)</i>	2 (40)
<i>Pseudophakic, n (%)</i>	3 (60)
OCT findings	
<i>ERM, n (%)</i>	1 (20)
<i>VMT, n (%)</i>	1 (20)
Comorbidities	
<i>DM, n (%)</i>	2 (40)
<i>HT, n (%)</i>	3 (60)
<i>Hyperlipidemia, n (%)</i>	1 (20)
Smoking status n (%)	1 (20)
(CRAO/CLRAO)	3 (60) / 2 (40)
Anatomical success, n (%)	4 (80)

Data are presented as mean ± standard deviation (SD) with range, or as number (percentage) [n (%)]. Laterality indicates the affected eye. Anatomical success denotes macular hole closure at last follow-up.

Abbreviations: OCT, optical coherence tomography; ERM, epiretinal membrane; VMT, vitreomacular traction; DM, diabetes mellitus; HT, hypertension; CRAO, central retinal artery occlusion; CLRAO, cilioretinal artery occlusion.

Table 2. Preoperative and 1-month postoperative visual and tomographic metrics in eyes with retinal artery occlusion (n = 5).

	Preoperative	Postoperative (first month)	<i>p</i>
BCVA, logMAR	0.7 ± 0.3	1.2 ± 0.2	0.014
IOP (mmHG)	15.4 ± 3.1	16.1 ± 2.1	0.675
MH size (µm)	511.3 ± 89.2	-	
CMT (µm)	261.5 ± 61.3	168.2 ± 36.6	< 0.001

Values are mean ± standard deviation (SD). Normality was assessed with the Shapiro–Wilk test; paired comparisons used the paired t-test or the Wilcoxon signed-rank test, as appropriate (two-tailed). Statistical significance was set at *p* < 0.05. Macular hole size was measured preoperatively only; the postoperative value is not applicable.

Abbreviations: BCVA, best-corrected visual acuity; logMAR, logarithm of the minimum angle of resolution; IOP, intraocular pressure; CMT, central macular thickness; MH, macular hole; mmHg, millimeters of mercury; µm, micrometers.

Table 3. One-month OCTA metrics in RAO eyes versus fellow (non-RAO) eyes ($n = 5$).			
	RAO eye	Control (non-RAO) eye	<i>p</i>
SCP FAZ (μm^2)	589,32 \pm 154,1	317,29 \pm 171,2	<0.001
SCP VD- fc (%)	35.3 \pm 10.3	36.1 \pm 8.0	0.532
SCP VD- temp (%)	43.4 \pm 5.3	44.2 \pm 5.1	0.316
SCP VD- sup (%)	46.1 \pm 6.6	47.5 \pm 2.7	0.068
SCP VD- nas (%)	46.0 \pm 6.1	45.4 \pm 5.3	0.917
SCP VD- inf (%)	45.1 \pm 5.3	46.8 \pm 4.3	0.486
SCP VD- mean (%)	45.1 \pm 5.5	45.9 \pm 4.4	0.156
DCP FAZ (μm^2)	521.6 \pm 98.8	334.2 \pm 121.4	<0.001
DCP VD- fc (%)	31.4 \pm 4.7	32.3 \pm 10.4	0.532
DCP VD- temp (%)	48.8 \pm 4.1	48.4 \pm 3.6	0.415
DCP VD- sup (%)	51.9 \pm 3.5	50.8 \pm 4.8	0.499
DCP VD- nas (%)	50.4 \pm 6.7	51.3 \pm 4.5	0.546
DCP VD- inf (%)	48.1 \pm 7.2	49.9 \pm 6.3	0.447
DCP VD- mean (%)	49.8 \pm 3.1	50.1 \pm 2.8	0.432

Values are mean \pm standard deviation (SD). FAZ area is expressed in μm^2 ; vessel density (VD) in percent. "fc" denotes the foveal center (central 1-mm circle). "temp/sup/nas/inf" indicate temporal, superior, nasal, and inferior parafoveal quadrants; mean is the average across the four parafoveal quadrants. P-values are from paired t-tests or Wilcoxon signed-rank tests, as appropriate. RAO eyes showed significantly larger FAZ areas in both SCP and DCP (both $p < 0.001$), whereas interocular differences in SCP and DCP vessel densities at 1 month were not statistically significant (all $p > 0.05$).

Abbreviations: RAO = retinal artery occlusion; FAZ = foveal avascular zone; VD = vessel density; SCP = superficial capillary plexus; DCP = deep capillary plexus.

DISCUSSION

Following MH surgery, RAO may occur, although rarely, and can result in catastrophic outcomes. Apart from a few case reports, the literature on this topic remains limited. For example, Bilgin et al. reported CLRAO on postoperative day 15 in a 78-year-old man after MH surgery combined with phacoemulsification.¹¹ Similarly, Enaida et al. reported a case of CLRAO that developed on postoperative day 4 in a 68-year-old woman following MH surgery.¹²

Iatrogenic RAO associated with PPV may occur through several mechanisms. The most frequently discussed cause is RAO following retrobulbar, peribulbar, or sub-Tenon anaesthesia. Potential mechanisms include unintentional intra-arterial injection, mechanical injury to the arterial

wall, increased intraorbital pressure from retrobulbar haematoma exceeding retinal arterial perfusion pressure (orbital compartment effect), possible toxic effects of local anaesthetics and their preservatives, and the vasoconstrictive effects of epinephrine-containing mixtures.¹⁴⁻¹⁸ In our study, all patients received approximately 4–5 mL of peribulbar anaesthesia with a 1:1 combination of bupivacaine and prilocaine, and no complications were observed during the block. Nevertheless, the possibility that any of these anaesthesia-related mechanisms contributed to the development of RAO cannot be excluded.

Advanced age, HT, DM, and cigarette smoking are well-established vascular risk factors for RAO and share a common pathophysiological substrate with ischemic stroke and

myocardial infarction. In the presence of these factors, both the risk of RAO and the probability of concomitant cardiovascular events increase. Notably, in a substantial proportion of patients, previously unrecognized cardiovascular risk conditions are uncovered at presentation.¹⁹ Reviews of reported cases of RAO following ocular surgery under periocular regional anaesthesia indicate that, even when no overt block-related complication is identified, underlying systemic vascular risk factors may contribute to its occurrence.²⁰ The advanced age profile and comorbidity burden in our cohort may have predisposed patients to the development of RAO.

Another plausible mechanism is injury to the arterial wall during ILM peeling, which may act as a precipitating factor for this event RAO.²¹ No major intraoperative complications occurred in any patient. In one case, bleeding occurred during ILM peeling due to arterial wall injury; the infusion pressure was increased to 35 mmHg to achieve haemostasis and then returned to baseline. No other intraoperative complications were observed, and no additional issues arose during or immediately after the procedure. Nevertheless, this patient developed RAO by the end of postoperative day 1 (Fig. 3). Elevated infusion pressure, intraoperative and postoperative increases in IOP, and expansion of the endotamponade gas, all of which raise IOP, may facilitate the development of RAO.^{22, 23} In our study, one patient

had an IOP of 28 mmHg on postoperative day 1, which was controlled with medical therapy; in this case, CLRAO developed on day 15. In all other cases, postoperative IOP values remained within the normal range. To the best of our knowledge, none of the patients was exposed to high-altitude conditions during the postoperative period that could have promoted gas expansion.

As expected, following RAO, CMT decreased and the FAZ enlarged, both significantly compared with the fellow eye ($p < 0.001$). As acute-phase OCT-A data were unavailable, VDs in the SCP and DCP were compared at the 1-month visit; mean values were lower in RAO eyes, but the differences did not reach statistical significance ($p > 0.05$). Although reduced SCP and DCP VD is frequently reported in the acute phase, observational data and animal models suggest that recanalisation and collateral capillary remodelling may partially restore VD over time.²⁴⁻²⁶ This may explain the absence of a significant interocular difference at 1 month.

This study provides an original, data-driven contribution to the literature on the rare occurrence of RAO following MH surgery. Key methodological strengths include systematic screening of a broad surgical cohort, adherence to a standardised protocol under a single surgeon, and meticulous documentation of detailed perioperative records. The principal limitations are the small sample size, inherent to the rarity of this complication, and the absence of preoperative and early postoperative OCT-A datasets, which precluded full characterisation of the temporal dynamics of vascular change.

In conclusion, this study highlights the association between MH surgery and RAO but does not establish causality. In addition to the possibility of arterial wall injury during ILM peeling, haemodynamic factors related to PPV, including transient elevations in infusion pressure, expansion of endotamponade gas, and postoperative increases in IOP, together with patient-related vascular risk profiles, may contribute to RAO. Pragmatic safeguards include preoperative risk stratification, optimisation of HT, DM, and lipid control, and support for smoking cessation. In patients with risk factors, intraoperatively, careful titration of infusion pressure and gas concentration, minimally traumatic ILM peeling, and avoidance of mixtures containing vasoconstrictors should be considered. Postoperatively,



Figure 3. Colour fundus photograph. The white arrow indicates the area of retinal artery occlusion, while the black arrows show retinal haemorrhages secondary to microtrauma during ILM peeling.

close surveillance with IOP monitoring, OCT, and OCTA, particularly during the first 24 to 48 hours and throughout the first month, is advisable for early detection of vascular events. Acute RAO may be precipitated by direct arterial wall injury or anaesthesia-related orbital compartment syndrome, whereas in late-onset cases it is prudent to consider facilitating factors rather than a single inciting event. Confirmation in prospective, preferably multicentre, studies is needed to refine prophylactic measures and strategies for early diagnosis and intervention.

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