

Spontaneous resolution of choroidal neovascular membrane in the fellow eye during hyperbaric oxygen treatment for retinal artery occlusion: a case report

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Keywords

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Abstract

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Retinal artery occlusion (RAO) is an ophthalmic emergency that causes sudden, painless vision loss due to retinal ischaemia. Hyperbaric oxygen therapy (HBOT), when initiated early, may help preserve photoreceptor function by increasing retinal oxygenation. A 69-year-old woman with a history of hypertension presented with sudden visual loss in the left eye and was diagnosed with RAO. HBOT was initiated within six hours of symptom onset (253 kPa [2.5 atmospheres absolute] for 90 minutes per session), and 20 sessions were planned. During the seventh session, the patient reported a marked improvement in visual acuity in the contralateral (right) eye, previously diagnosed with choroidal neovascular membrane (CNVM) and untreated with anti-vascular endothelial growth factor (VEGF) therapy. Optical coherence tomography demonstrated regression of the CNVM and complete resolution of subretinal fluid. To our knowledge, this is the first report suggesting that HBOT administered for unilateral RAO may also promote structural and functional improvement of CNVM in the contralateral eye.

Introduction

Retinal artery occlusion (RAO) is a vision-threatening ophthalmic emergency characterised by sudden, painless monocular vision loss due to the inability of the retinal tissue to meet its high oxygen demand.¹ The inner retinal layers are highly susceptible to ischaemia owing to their elevated metabolic activity. Therefore, early intervention is critical to preserve retinal function. Hyperbaric oxygen therapy (HBOT) enables the inhalation of 100% oxygen under high pressure, increasing oxygen diffusion to ischaemic retinal layers through the choroidal circulation and is considered a potential adjunctive treatment in RAO.^{2,3} However, systematic reviews evaluating HBOT in RAO have shown conflicting results; some studies suggest benefit when initiated within the first nine hours,⁴ whereas others report no significant improvement in final visual outcomes.⁵ Additionally, preliminary evidence indicates that HBOT may have therapeutic effects in other ocular pathologies such as choroidal neovascular membranes (CNVM).⁶ In this context, structural and functional improvement of CNVM in the contralateral eye during HBOT for unilateral RAO

represents, to the best of our knowledge, an unreported clinical observation.

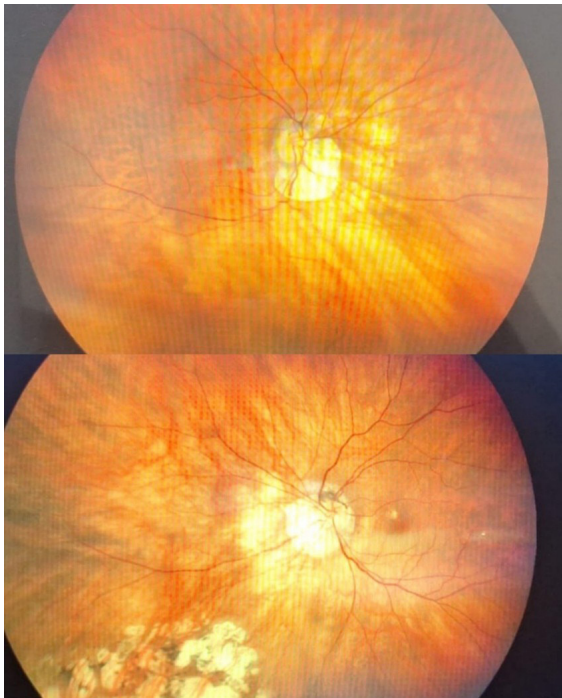
Case report

Written informed consent was obtained from the patient for publication of this case report and the accompanying images.

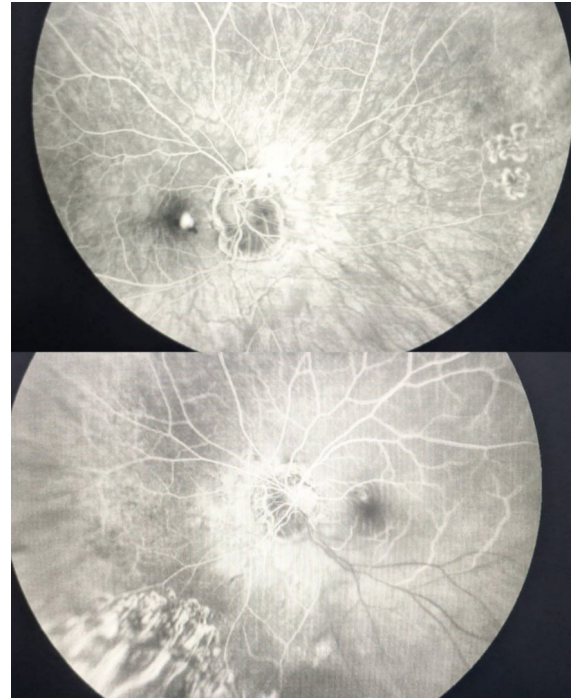
A 69-year-old woman with a history of hypertension presented with sudden, painless visual loss in the left eye, beginning six hours prior. Best-corrected visual acuity (BCVA) was counting fingers at five metres in the right eye and counting fingers at one metre in the left eye. Fundus examination of the left eye showed retinal whitening in the inferior hemiretina (Figure 1). Fundus fluorescein angiography demonstrated macular staining in the right eye and arterial filling defect in the inferior retina of the left eye, consistent with RAO (Figure 2). Both eyes exhibited peripapillary atrophy and high myopia > -5.00 D. Optical coherence tomography (OCT) of the right eye revealed marked hyperreflectivity of the inner retinal layers, loss of the foveal contour, and a subretinal hyperreflective

Figure 1

Pre-treatment fundus photograph of the left eye showing marked retinal whitening in the inferior hemiretina

**Figure 2**

Pre-treatment fluorescein angiography demonstrating an arterial filling defect in the inferior retina of the left eye



lesion (Figure 3). HBOT was initiated within six hours of symptom onset (253 kPa [2.5 atmospheres absolute], 90 minutes daily), with 20 sessions planned. During the seventh session, the patient reported subjective visual improvement in the right eye, previously diagnosed with CNVM but untreated with anti-vascular endothelial growth factor (anti-VEGF) injections. Follow-up OCT demonstrated significant regression of the CNVM and complete resolution of subretinal fluid (Figure 3). No anti-VEGF therapy or other interventions were administered during the treatment period. At final follow-up, BCVA was 2/10 in the right eye and counting fingers at five metres in the left eye.

Discussion

Although RAO is one of the most dramatic causes of sudden vision loss, there is still no standardised and effective treatment available. Interventions such as ocular massage, intraocular pressure-lowering agents, carbogen inhalation, and anticoagulant therapy have been used, but none have demonstrated a consistent improvement in visual prognosis.^{1,2} Therefore, HBOT, which aims to maintain the viability of hypoxic retinal tissue, stands out as one of the few rational treatment options with a physiological basis. The goal of HBOT is to increase systemic oxygen pressure and promote oxygen diffusion to ischaemic retinal layers through the choroidal circulation.³ However, studies to date have reported highly variable treatment outcomes, depending on factors such as time to therapy initiation, duration of ischaemia, and systemic health status of the patient.⁴⁻⁶ Choroidal neovascular membrane typically demonstrates

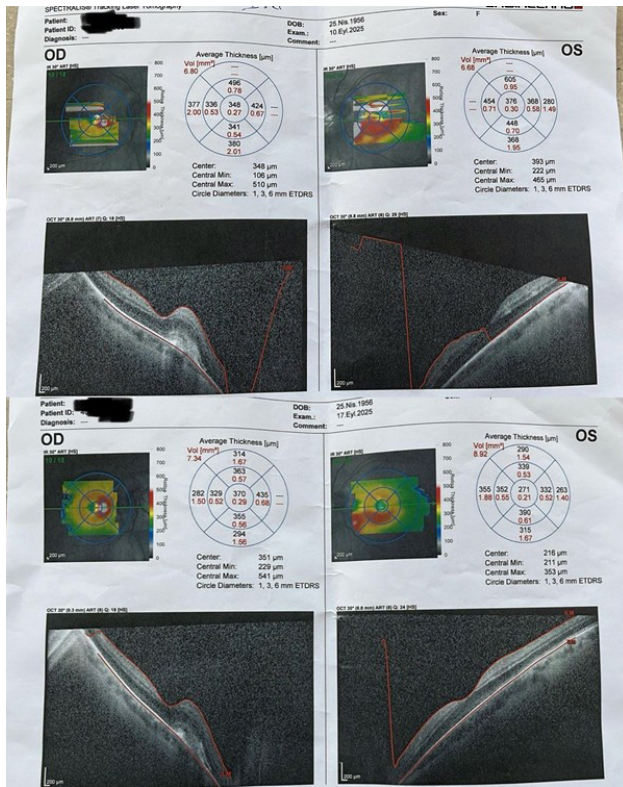
a progressive course if left untreated. The proliferation of abnormal choroidal vessels beneath the retina may lead to subretinal haemorrhage, exudation, and fibrotic scar formation, frequently resulting in permanent central vision loss. Spontaneous regression is uncommon, and the visual prognosis is generally poor without treatment.⁷

In the present case, while HBOT was initiated for unilateral RAO, an unexpected structural and functional improvement was observed in the contralateral eye with previously diagnosed CNVM. To our knowledge, there are no prior reports describing regression of CNVM in the fellow eye during HBOT administered for RAO. This finding suggests that HBOT may induce systemic or cross-ocular effects at the level of the retina and choroid.

The underlying pathophysiological mechanism remains unclear. HBOT increases the amount of oxygen dissolved in plasma, facilitating oxygen diffusion into hypoxic retinal tissues and potentially suppressing hypoxia-inducible factor and VEGF expression.⁸ Retinal hypoxia is known to upregulate VEGF, contributing to neovascularisation in both age-related macular degeneration and other ischaemic retinopathies.^{9,10} Increased systemic oxygenation may therefore indirectly downregulate VEGF-mediated angiogenesis, contributing to regression of CNVM. Experimental models have also demonstrated inhibitory effects of hyperoxia on choroidal neovascularisation, including downregulation of VEGF and inflammatory mediators.¹¹ Clinically, Malerbi et al. previously reported anatomical improvement in CNVM following HBOT,⁶ and

Figure 3

Optical coherence tomography(OCT) images; pre-treatment OCT shows hyperreflectivity of the inner retinal layers, loss of foveal contour, and a subretinal hyperreflective lesion. Post-treatment OCT demonstrates regression of the choroidal neovascular membrane (CNVM) and complete resolution of subretinal fluid



our observation is consistent with these findings, further suggesting that similar effects may occur in the fellow eye during systemic hyperoxia.

Spontaneous regression of CNVM cannot be completely ruled out; however, such regression is considered extremely rare (< 1%) in eyes not treated with anti-VEGF agents.¹² The temporal correlation between the initiation of HBOT and the observed CNVM regression, along with the absence of any additional therapy, supports the possibility that HBOT may have contributed to this outcome.

Conclusion

Structural and functional improvement of a contralateral CNVM may occur during HBOT administered for unilateral RAO. Further clinical and experimental research is needed to determine whether this represents a reproducible therapeutic effect or a coincidental finding.

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