

HYPERBARIC OXYGEN THERAPY IN ACUTE RETINAL ISCHEMIA (CRAO AND BRAO): A NARRATIVE REVIEW

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ABSTRACT

Background: Acute retinal ischemia, including central retinal artery occlusion (CRAO) and branch retinal artery occlusion (BRAO), is associated with sudden and often profound visual loss, with no universally accepted effective treatment. Hyperbaric oxygen therapy (HBOT) has been proposed as a potential rescue therapy by enhancing retinal oxygenation through choroidal diffusion, particularly in the early stages of ischemia; however, its clinical role remains controversial.

Objective: To provide a narrative review of the current clinical evidence regarding the use of HBOT in acute retinal ischemia caused by CRAO and BRAO, with a focus on treatment timing, therapeutic protocols, and visual outcomes.

Methods: A narrative review of English-language studies published between January 2016 and December 2025 was conducted using PubMed. Studies evaluating HBOT in CRAO or BRAO were included. Data regarding treatment timing, protocols, and visual outcomes were synthesized descriptively.

Results: Twenty-three studies met the inclusion criteria, comprising predominantly case reports, small case series, and retrospective cohort studies, with a limited number of prospective investigations. Baseline visual acuity was typically poor, frequently exceeding 2.0 logMAR. HBOT protocols were heterogeneous but most commonly involved 100% oxygen at pressures between 2.0 and 2.8 ATA, with session durations of 90-120 minutes and intensified treatment during the first 24-72 hours. Across studies, clinically meaningful visual improvement (≥ 0.3 logMAR) was reported in a substantial proportion of patients, particularly when HBOT was initiated early. Patients with BRAO generally demonstrated more favorable visual recovery compared with those with CRAO. Several studies also reported visual improvement despite delayed initiation of HBOT beyond traditional therapeutic time windows.

Conclusions: HBOT has been associated with visual improvement in selected patients with retinal artery occlusion, particularly when treatment is initiated early. Visual outcomes are more favorable in branch retinal artery occlusion than in central retinal artery occlusion, and HBOT is generally well tolerated. However, the available evidence remains limited and heterogeneous, highlighting the need for prospective, standardized studies.

Keywords: Hyperbaric oxygen therapy, retinal ischemia, central retinal artery occlusion, CRAO, branch retinal artery occlusion, BRAO.

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INTRODUCTION

Acute retinal ischemia represents a spectrum of ocular vascular events resulting from sudden interruption of arterial blood flow to the retina. This spectrum includes transient monocular vision loss, BRAO, and CRAO, which are increasingly regarded as ocular equivalents of cerebral ischemia [1,2]. In this context, CRAO is often described as an “ocular stroke,” requiring urgent evaluation and management analogous to acute ischemic stroke [3].

The incidence of CRAO is relatively low but clinically significant, with population-based studies estimating approximately 1-2 cases per 100,000 person-years, predominantly affecting older individuals with vascular risk factors [4,5]. BRAO appears to be more common, though precise epidemiological data are less consistent, and visual outcomes are generally more favorable due to the limited extent of retinal ischemia [6]. Importantly, retinal artery occlusion is not only an ophthalmic emergency but also a marker of systemic vascular disease, as patients demonstrate an increased short- and long-term risk of cerebrovascular and cardiovascular events [7].

The natural history of CRAO is associated with poor visual prognosis. In the absence of a cilioretinal artery, profound and permanent vision loss is common, and ischemic complications such as ocular neovascularization or neovascular glaucoma may develop within weeks to months [8,9]. Experimental and clinical studies suggest that retinal ganglion cells have a limited tolerance to ischemia, underscoring the importance of rapid intervention [10]. Despite this, effective acute treatment remains elusive.

To date, no universally accepted, evidence-based therapeutic strategy has been established for the management of CRAO or BRAO. Traditional acute interventions, including ocular massage, anterior chamber paracentesis, intraocular pressure-lowering agents, and normobaric oxygen, are widely used but lack robust evidence of consistent benefit [3,11]. While thrombolytic therapy has been explored, its use remains controversial due to narrow therapeutic windows, variable outcomes, and safety concerns [3,12]. Consequently, current guidelines emphasize urgent diagnostic evaluation, identification of embolic sources, and secondary prevention rather than definitive restoration of retinal perfusion [3,11].

HBOT has been proposed as a potential adjunctive treatment for acute retinal ischemia. By substantially increasing the partial pressure of oxygen in plasma, HBOT may enhance oxygen diffusion from the choroidal circulation to the ischemic inner retinal layers, thereby sustaining retinal metabolism until reperfusion occurs [13]. This physiological rationale is particularly compelling in CRAO, where early intervention could theoretically preserve retinal viability and improve functional outcomes. Nevertheless, the clinical application of HBOT is limited by availability, variability in treatment protocols, and uncertainty regarding optimal timing and patient selection.

Although the number of publications addressing HBOT in retinal artery occlusion has increased over the past decade, the available evidence remains heterogeneous. Studies vary widely in design, inclusion criteria, timing of treatment initiation, HBOT protocols,

and outcome reporting. Moreover, existing reviews often focus exclusively on CRAO or include mixed retinal vascular pathologies, limiting their clinical applicability. Consequently, there remains a need for an updated, structured synthesis of the clinical evidence on HBOT in acute retinal ischemia due to CRAO and BRAO, with particular attention to treatment timing, therapeutic protocols, and visual outcomes.

PATHOPHYSIOLOGY OF RETINAL ISCHEMIA

RETINAL VASCULAR SUPPLY: CENTRAL RETINAL ARTERY VERSUS CHOROIDAL CIRCULATION

The retina is characterized by a unique dual blood supply that plays a pivotal role in the pathophysiology of retinal ischemia. The inner retinal layers, including the retinal ganglion cells and inner nuclear layer, are primarily supplied by the central retinal artery (CRA), a branch of the ophthalmic artery. In contrast, the outer retina - most notably the photoreceptor layer - derives its oxygen and metabolic support predominantly from the choroidal circulation, which is supplied by the posterior ciliary arteries and is among the highest-flow vascular beds in the human body [1,6].

This anatomical arrangement has critical functional implications. Occlusion of the CRA leads to abrupt ischemia of the inner retinal layers, while the outer retina often remains relatively preserved due to continued oxygen diffusion from the choroid. However, because the CRA is an end artery with minimal collateralization, compensatory mechanisms are limited, rendering the inner retina particularly vulnerable to ischemic injury [6,2]. This disparity in vascular supply forms the physiological basis for both the clinical manifestations of retinal artery occlusion and the rationale for therapies aimed at enhancing oxygen delivery via the choroidal circulation.

RETINAL SURVIVAL TIME UNDER ISCHEMIC CONDITIONS

Experimental and clinical studies have demonstrated that the retina has a limited tolerance to ischemia. Hayreh et al. established that irreversible retinal damage occurs after a critical duration of arterial occlusion, with retinal ganglion cells showing irreversible injury after approximately 240 minutes of complete ischemia [10]. Shorter ischemic intervals may result in partial or reversible dysfunction, highlighting the time-dependent nature of retinal injury. Clinical observations support these experimental findings. Patients presenting early after symptom onset occasionally demonstrate visual recovery, whereas delayed presentation is typically associated with poor functional outcomes. Importantly, the ischemic tolerance of the retina may vary depending on residual perfusion, partial occlusion, or the presence of a cilioretinal artery, which can provide limited alternative blood supply to the macular region [1,11]. These factors contribute to the heterogeneity of visual outcomes observed in retinal artery occlusion and underscore the critical importance of time in therapeutic decision-making.

MECHANISMS OF NEURONAL INJURY IN RETINAL ISCHEMIA

Ischemic injury to the retina initiates a cascade of molecular and cellular events leading to neuronal dysfunction and cell death. Oxygen and glucose deprivation rapidly impair mitochondrial oxidative phosphorylation, resulting in ATP depletion and failure of ion homeostasis. Subsequent depolarization of neuronal membranes promotes excessive release of excitatory neurotransmitters, particularly glutamate, triggering excitotoxic injury through overstimulation of NMDA and AMPA receptors [14]. Concomitantly, intracellular calcium overload, oxidative stress, and activation of apoptotic signaling pathways contribute to progressive retinal ganglion cell loss. Inflammatory responses and microvascular endothelial dysfunction further exacerbate tissue injury during both the ischemic phase and subsequent reperfusion [14]. These mechanisms collectively explain why retinal ischemia often results in permanent visual deficits despite restoration of arterial patency and provide a biological rationale for neuroprotective or oxygen-enhancing interventions during the acute phase.

PATHOPHYSIOLOGICAL DIFFERENCES BETWEEN CRAO AND BRAO

Although CRAO and BRAO share common ischemic mechanisms, important pathophysiological differences exist. CRAO results in diffuse ischemia of the inner retina, typically involving the macula, and is therefore associated with profound and often irreversible visual loss. In contrast, BRAO affects a localized arterial branch, leading to sectoral ischemia that spares unaffected retinal regions and frequently preserves central vision [1,2]. The more limited extent of ischemia in BRAO confers a greater potential for functional recovery, particularly when the fovea is not involved. Additionally, residual perfusion from adjacent arterial territories may partially mitigate ischemic injury in BRAO, further contributing to its generally more favorable prognosis. These distinctions are clinically relevant when interpreting treatment responses and visual outcomes, as therapeutic interventions - including HBOT - may yield different benefits depending on the extent and distribution of retinal ischemia.

PHYSIOLOGICAL BASIS OF HYPERBARIC OXYGEN THERAPY IN RETINAL ISCHEMIA

MECHANISM OF ACTION OF HYPERBARIC OXYGEN THERAPY

HBOT involves the inhalation of near-100% oxygen under increased ambient pressure, typically between 2.0 and 2.8 atmospheres absolute (ATA) for retinal artery occlusion. Under these conditions, arterial oxygen tension rises to supraphysiological levels, resulting in a marked increase in the amount of oxygen dissolved directly in plasma. Unlike normobaric oxygen delivery, which relies predominantly on hemoglobin-bound oxygen, HBOT enables significant oxygen transport independent of erythrocytes, driven by diffusion along steep partial pressure gradients [9,13,15]. This physiological mechanism constitutes the fundamental rationale for the

use of HBOT in ischemic conditions where arterial blood flow is impaired, including acute retinal arterial ischemia.

OXYGEN DIFFUSION FROM THE CHOROIDAL CIRCULATION

In retinal artery occlusion, the choroidal circulation typically remains preserved despite interruption of retinal arterial inflow. Under hyperbaric conditions, the substantial increase in arterial oxygen tension leads to markedly elevated oxygen levels within the choroidal vasculature and outer retina. Experimental and physiological studies have demonstrated that hyperoxia significantly enhances retinal tissue oxygenation, thereby increasing the diffusion gradient toward ischemic inner retinal layers [2,6,17,18]. This choroidal-to-retinal diffusion of oxygen constitutes the principal physiological justification for the use of HBOT in retinal arterial occlusion.

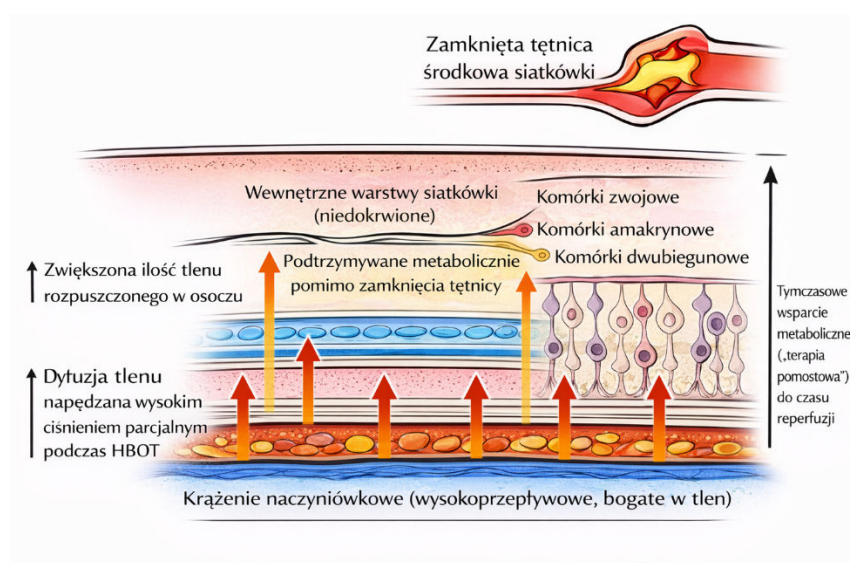


Fig. 1 Mechanism of oxygen delivery to the inner retinal layers during HBOT in retinal artery occlusion.

IMPROVED OXYGENATION OF INNER RETINAL LAYERS

Ischemic injury following retinal artery occlusion predominantly affects the inner retinal layers, particularly retinal ganglion cells, which are highly sensitive to oxygen deprivation. Experimental primate models have shown that retinal survival following complete arterial occlusion is strictly time-dependent, with irreversible structural damage occurring after prolonged ischemia [10]. By substantially increasing tissue oxygen availability through diffusion from the choroid, HBOT may temporarily sustain inner retinal metabolism and delay the progression of ischemia-induced cellular injury. Proposed mechanisms include preservation of mitochondrial function, attenuation of excitotoxic glutamate release, reduction of oxidative stress, and modulation of apoptotic signaling pathways that contribute to retinal ganglion cell death [10,14,18].

IMPORTANCE OF OXYGEN PARTIAL PRESSURE

Oxygen delivery to ischemic retinal tissue is governed by diffusion physics and depends directly on the partial pressure gradient between blood and tissue. Under normobaric conditions, supplemental oxygen produces only modest increases in arterial oxygen tension and is often insufficient to support ischemic inner retinal layers when retinal arterial perfusion is severely compromised or absent. In contrast, HBOT generates arterial oxygen tensions that may exceed 1,000 mmHg, substantially increasing the diffusion distance of oxygen from the choroid toward the inner retina [9,15]. This physiological principle explains why treatment pressure, session duration, and early initiation of HBOT are considered critical determinants of potential therapeutic efficacy in retinal artery occlusion [9,13].

HYPERBARIC OXYGEN THERAPY AS A „BRIDGE THERAPY” TO REPERFUSION

Acute retinal artery occlusion is increasingly conceptualized as an ocular manifestation of ischemic stroke, requiring urgent diagnostic evaluation and time-sensitive intervention [3]. Within this framework, HBOT may be regarded as a temporary supportive or “bridge” therapy aimed at preserving retinal viability until spontaneous reperfusion occurs or until partial restoration of perfusion through recanalization or collateral circulation is achieved. This concept is consistent with contemporary clinical guidelines, which acknowledge that although high-level evidence is limited, HBOT may be considered in selected patients - particularly when treatment can be initiated early after symptom onset [3,11,13]. Anatomical modifiers such as incomplete occlusion or the presence of a cilioretinal artery may further influence the effectiveness of this bridging effect by providing residual oxygen delivery to the macular region [2,6].

SAFETY PROFILE AND CONTRAINDICATIONS

HBOT is generally regarded as a safe therapeutic modality when administered according to established protocols and under appropriate medical supervision. Reported adverse effects are usually mild and reversible, and can be broadly categorized into pressure-related complications and oxygen-related toxicity [15,19].

Pressure-related complications arise during compression and decompression phases of treatment and primarily involve air-filled cavities, including the middle ear, paranasal sinuses, teeth, and lungs. Middle ear barotrauma is the most frequently reported adverse event and may range from transient discomfort to tympanic membrane injury in rare cases. Sinus and dental barotrauma have also been described, while pulmonary barotrauma is uncommon but potentially serious. In this context, untreated pneumothorax constitutes an absolute contraindication to HBOT, as pressurization may result in

expansion of trapped intrathoracic air and life-threatening cardiopulmonary compromise [19,20].

Oxygen-related adverse effects reflect the pharmacological action of hyperoxia. Central nervous system oxygen toxicity is rare at pressure levels typically used in clinical HBOT but may present with prodromal symptoms such as visual disturbances, tinnitus, nausea, irritability, or muscle twitching, and in rare cases may progress to generalized tonic-clonic seizures. These events are usually self-limited and resolve with prompt reduction of inspired oxygen concentration [19,20]. Pulmonary oxygen toxicity, characterized by cough, chest discomfort, and decreased pulmonary compliance, is more commonly associated with prolonged or repeated exposure and is infrequently observed in short-term treatment protocols [20].

Several additional systemic and ocular effects have been reported. Transient myopic shift, attributed to reversible changes in lens refractive properties under hyperoxic conditions, is a recognized but typically self-limiting phenomenon. Cataract progression has been described primarily in patients undergoing long-term HBOT and is unlikely to be clinically relevant in the limited number of sessions usually applied for acute retinal ischemia [15,21]. Psychological factors such as claustrophobia may also limit tolerability in some patients, particularly during treatment in monoplace chambers [19].

Relative contraindications to HBOT include severe chronic obstructive pulmonary disease, uncontrolled asthma, acute upper respiratory tract infection, and the presence of implanted medical devices that are not pressure-compatible. Prior exposure to certain chemotherapeutic agents, such as bleomycin or doxorubicin, warrants caution due to increased susceptibility to pulmonary oxygen toxicity [19,21]. Given that patients with retinal artery occlusion are often elderly and have multiple vascular comorbidities, careful patient selection and individualized risk-benefit assessment are essential before initiating HBOT.

METHODS

SEARCH STRATEGY

A narrative literature review was conducted using the PubMed database. The search was limited to English-language publications published between January 2016 and December 2025. The search strategy combined terms related to hyperbaric oxygen therapy and acute retinal ischemia, including *"hyperbaric oxygen therapy," "hyperbaric oxygen," "HBOT," "central retinal artery occlusion," "branch retinal artery occlusion," "CRAO,"* and *"BRAO"*. Reference lists of relevant articles were also manually screened to identify additional eligible studies.

STUDY SELECTION AND ELIGIBILITY CRITERIA

Studies were eligible for inclusion if they reported the use of HBOT in patients with acute retinal ischemia caused by CRAO or BRAO. Case reports, case series, prospective cohort studies, and retrospective observational studies were included.

Studies were excluded if they primarily involved retinal vein occlusion, inflammatory or vasculitic retinal

disorders, congenital retinal vascular anomalies, traumatic or iatrogenic retinal ischemia, or mixed retinal vascular pathologies in which outcomes attributable to CRAO or BRAO could not be clearly separated. Studies without sufficient information on visual outcomes or timing of HBOT were also excluded.

DATA EXTRACTION

From each included study, the following data were extracted: author and year of publication, study design, type of retinal artery occlusion (CRAO or BRAO), number of treated eyes, time from symptom onset to initiation of HBOT, details of the HBOT protocol (pressure, session duration, and number of sessions), and reported visual outcomes. In comparative studies, only data from patients treated with HBOT were extracted and analyzed.

OUTCOME MEASURES AND DATA SYNTHESIS

Visual acuity was the primary outcome of interest. For consistency, visual acuity values were standardized to logMAR units for tabular presentation and descriptive analysis. Snellen or decimal visual acuity values were converted to logMAR where applicable. Very low vision categories were converted using predefined logMAR equivalents (counting fingers = 1.9, hand motion = 2.3, light perception = 2.7, no light perception = 3.0). Given the heterogeneity of study designs, patient populations, treatment protocols, and outcome reporting, a quantitative meta-analysis was not performed. Instead, findings were synthesized descriptively, with results summarized in tabular form to facilitate comparison across studies.

SUMMARY OF INCLUDED STUDIES

HBOT evidence table.

Author	Study type	Condition	Eyes (n)	Time to HBOT from symptom onset	HBOT protocol (ATA, session duration, number of sessions)	Main outcome
Doğruya et al. (2025) [22]	Retrospective comparative	CRAO	7	5.85 ± 10.71 days	100% O ₂ , 2.4 ATA, 120 min/session; 2 sessions/day for 1 week, then 1 session/day for 6 days (total 20 sessions)	VA improved in 2 of 7 eyes, remained stable in 4 eyes, and worsened in 1 eye.
Kim et al. (2018) [23]	Case report	CRAO	1	10 h	100% O ₂ up to 2.8 ATA, 140 min/session; 1 session/day for 3 days	VA improvement
Elder et al. (2017) [24]	Retrospective case series	ARAO (CRAO, BRAO)	31	3-25.5 h	100% O ₂ , 2.0-2.8 ATA for 60-120 min; median 4 sessions (range 1-7)	VA improvement in 29%; all responders treated within 10 h
Thoongsuwan et al. (2025) [25]	Retrospective comparative cohort	CRAO (including cilioretinal sparing)	17	11.6 ± 6.8 h	Day 1: 100% O ₂ , 2.0 ATA, 180 min/session Days 2-5: 100% O ₂ , 2.4 ATA, 90 min/session	A higher rate of visual improvement in HBOT group compared with the non-HBOT group
Chiabo et al. (2024) [26]	Prospective non-comparative cohort	CRAO and BRAO	31	≤7 days (32% <12 h)	100% O ₂ , 2.5 ATA, 90 min/session; twice daily until FA-confirmed revascularisation (mean 34 sessions, range 13–56)	Clinically significant VA improvement (≥0.3 logMAR) in 48.4% of eyes
Zarzecki et al. (2025) [27]	Case series	BRAO	3	Case 1 – 36 h Case 2 – 48 h Case 3 – 5 h	100% O ₂ , 2.5 ATA, 90 min/session, once daily; 10–15 sessions	All eyes recovered full functional VA and complete resolution of visual field defects
Hadanny et al. (2016) [28]	Retrospective cohort	CRAO	128	7.8 ± 3.8 h	100% O ₂ , 2.0–2.4 ATA, 90 min; 3 sessions in first 24 h, then daily until no further improvement (mean 4 sessions)	2/3 achieved clinically meaningful VA improvement; absence of cherry-red spot strongly predicted response to HBOT
Au & Chong (2025) [20]	Prospective cohort	CRAO (cilioretinal-sparing excluded)	26	13.2 ± 7.5 h	100% O ₂ ; 1 session at 2.8 ATA (~140 min), then 9 sessions at ≤2.4 ATA (~120 min)	Significant VA improvement
Bhuvaneshwari et al. (2025) [29]	Case report	CRAO	1	7 h	100% O ₂ ; 8 sessions (pressure and duration not specified)	VA improvement
Soares et al. (2017) [30]	Case reports	CRAO	2	Case 1 – 6 h Case 2 – 4 h	100% O ₂ at 2.4 ATA; 90 min/session Case 1: 2 sessions/day for 3 days + 1 session on day 5 (total 7). Case 2: 2 sessions/day for 2 days, then 1/day for 5 days (total 9)	VA improvement with normalization of retinal perfusion on FA
Tang et al. (2016) [31]	Case report	CRAO	1	24 h	Two sessions daily for 5 days (total 10 sessions); each session ~4h (pressure not specified)	Partial visual improvement, early ocular neovascularization and neovascular glaucoma
Khallouli et al. (2020) [32]	Case report	CRVO with CLRAO	1	2 days	100% O ₂ , 2.5 ATA, 2 h sessions; total 30 sessions	VA fully recovered
Blegen et al. (2021) [33]	Case series	CRAO	4	6-22 h	100% O ₂ , 2.0-2.8 ATA; 90–278 min/session; 7–9 sessions	2/4 eyes developed early ocular neovascularization, particularly in DM patients

Tab. 1 cont

HBOT evidence table.

Author	Study type	Condition	Eyes (n)	Time to HBOT from symptom onset	HBOT protocol (ATA, session duration, number of sessions)	Main outcome
Feng et al. (2020) [36]	Case report	BRAO	1	Within days	100% O ₂ , 2.0 ATA, 110 min/session, once daily for 10 days	VA fully recovered
Uslu & Kanra (2020) [37]	Case report	BRAO	1	3 days	100% O ₂ , 2.4 ATA, 120 min/session; 2 sessions/day for 2 weeks then 1/day; total 20 sessions	VA improvement
Wang et al. (2023) [38]	Case report	Multiple BRAO	1	5 days	40 HBOT sessions over 2 months (pressure not specified)	VA improvement
Xiong et al. (2020) [39]	Case report	CRAO	1	24 h	Pressure and session duration not specified	VA improvement
Rozenberg et al. (2022) [40]	Retrospective comparative study	CRAO	121	9.1 ± 5 h	3 sessions within 24 h (8 h apart), while the first session was at 2.4 ATA and the rest of the sessions at 2 ATA of 100% oxygen, 90 min/session	Significant BCVA improvement vs conservative treatment
Maldonado et al. (2024) [41]	Retrospective cohort	CRAO	114	12.2 ± 6 h	100% O ₂ , 2.4 ATA, 90 min/session; median 7 sessions (range 2-19); twice daily for first 72 h, then daily	VA improvement
Lee et al. (2024) [42]	Retrospective comparative study	CRAO	29	Mean 86.7 ± 135.8 h (≤7 days)	100% O ₂ ; 2.8 ATA, 90 min twice daily (first 3 days), then 120 min once daily; up to 14 days	Significant BCVA improvement vs conservative treatment
Yang et al. (2025) [43]	Retrospective comparative study	CRAO	39	Median 6 days	100% O ₂ ; 2.0-2.4 ATA, 90 min/session once daily; ~10 sessions, extended if improvement	Greater BCVA improvement and higher rate of significant visual recovery vs conservative treatment, despite delayed treatment

VA = visual acuity; h = hour; ARAO = acute retinal arterial occlusion; CRAO = central retinal artery occlusion; BRAO = branch retinal artery occlusion; CLRAO = cilioretinal artery occlusion; CRVO = central retinal vein occlusion; FA = fluorescein angiography; DM = diabetes mellitus

In studies including a control group, data presented in this table refer exclusively to patients treated with hyperbaric oxygen therapy.

Patients characteristics and visual outcomes.

Author	Mean age (years)	Comorbid diseases	Baseline VA (logMAR)	Final VA (logMAR)
Doğruya et al. (2025) [22]	65.2 ± 9.9	HTN, DM, CAD	3.1	3.1
Kim et al. (2018) [23]	81	HF, AF, prior renal infarction	2.3	0.1
Elder et al. (2017) [24]	70	HTN, DM, carotid disease	2.5	0.5 (responders)/3.0 (non-responders)
Thoongsuwan et al. (2025) [25]	53.8 ± 22.3	HTN, DM, dyslipidemia, obesity, CVD	2.3 ± 0.4	1.4
Chiabo et al. (2024) [26]	68.3	HTN, dyslipidemia, DM	1.51	1.10
Zarzecki et al. (2025) [27]	41.3	HTN, post-carotid aneurysm embolization	0.1	0.0
Hadanny et al. (2016) [28]	66.4 ± 13.1	HTN, DM, CVD	2.14 ± 0.50	1.61 ± 0.78
Au & Chong (2025) [20]	67.5 ± 13.3	Not mentioned	2.0 ± 0.3	1.5 ± 0.7
Bhuvaneswari et al. (2025) [29]	52	Carotid artery occlusion, probable polycythemia vera	2.7	0.52
Soares et al. (2017) [30]	65	HTN, DM, CVD	1.9	0.05
Tang et al. (2016) [31]	70	HTN, dyslipidemia, DM, CVD	2.0	1.0
Khallouli et al. (2020) [32]	48	Hormonal fertility treatment	0.15	0.0
Blegen et al. (2021) [33]	70.8	DM, HTN, dyslipidemia, CAD	2.3	1.9
Chowdhary et al. (2020) [34]	25	None	1.9	1.10
Wu & Wu (2021) [35]	49	HTN, DM, CAD	1.0	3.0
Feng et al. (2020) [36]	42	DM, diabetic retinopathy, hypercoagulable state after high-altitude exposure	0.3	0.0
Uslu & Kanra (2020) [37]	77	AF, HTN	0.52	0.05
Wang et al. (2023) [38]	23	Postpartum	2.3	0.52
Xiong et al. (2020) [39]	43	None reported	3.0	0.52
Rozenberg et al. (2022) [40]	69 ± 12	HTN, DM, dyslipidemia	2.89 ± 0.98	2.15 ± 1.07
Maldonado et al. (2024) [41]	68.9 ± 13	HTN, dyslipidemia, CVD, DM, obesity	2.12 ± 0.74	1.60
Lee et al. (2024) [42]	65.7 ± 15.2	DM, HTN, dyslipidemia	2.03 ± 0.74	1.55 ± 0.97
Yang et al. (2025) [43]	67.5 ± 11.9	HTN, DM, CVD, dyslipidemia	2.3	1.9

LogMAR = logarithm of the Minimum Angle of Resolution, VA = visual acuity; HTN = hypertension; DM = diabetes mellitus; CAD = coronary artery disease; HF = heart failure; AF = atrial fibrillation; CVD = cardiovascular disease

In studies including a control group, data presented in this table refer exclusively to patients treated with hyperbaric oxygen therapy

CLINICAL EVIDENCE AND VISUAL OUTCOMES OF HBOT

VISUAL OUTCOMES IN CRAO

In patients with CRAO, baseline visual acuity is typically poor, most commonly ranging from counting fingers to hand motion or worse at presentation. Across published series, visual improvement following HBOT is variable and strongly time-dependent. When HBOT is initiated within the first hours after symptom onset, reported improvement generally corresponds to approximately 2 to 4 Snellen lines, or an improvement of roughly 0.2–0.4 logMAR in responders. In some early-treated cases, more substantial gains have been described, including recovery to functional visual acuity; however, such outcomes remain uncommon. Patients treated outside the early therapeutic window demonstrate a markedly lower likelihood of meaningful visual recovery. In late-presenting CRAO, HBOT is frequently associated with stabilization rather than improvement of visual acuity, and many patients show no measurable change from baseline. Importantly, the presence of anatomical modifiers, such as incomplete arterial occlusion or a cilioretinal artery supplying the macula, appears to increase the probability of visual improvement, even when treatment initiation is delayed. Overall, the clinical evidence suggests that HBOT may confer modest but potentially clinically meaningful visual benefit in selected CRAO patients, particularly when therapy is initiated early.

VISUAL OUTCOMES IN BRAO

Compared with CRAO, patients with BRAO generally present with better baseline visual acuity and a more favorable natural history. Across reported studies, visual improvement following HBOT in BRAO is more consistent and of greater magnitude. Responders frequently demonstrate improvement of approximately 2 to 5 Snellen lines, corresponding to 0.2–0.5 logMAR, particularly when the macula is partially involved and treatment is initiated promptly. In some cases, near-complete visual recovery has been reported, reflecting both residual perfusion and the smaller ischemic burden characteristic of BRAO. Even when HBOT is initiated later, patients with BRAO appear more likely than those with CRAO to experience partial improvement, supporting the concept that residual arterial flow and collateral oxygen delivery play a critical role in therapeutic responsiveness.

METHODOLOGICAL HETEROGENEITY AND DETERMINANTS OF VARIABILITY

The substantial heterogeneity in visual outcomes observed across the analyzed studies is a consequence of differences in study design, inclusion criteria, timing of HBOT initiation, and treatment protocol intensity. Most studies were retrospective, with limited standardization of visual acuity measurement and inconsistent definitions of clinically meaningful improvement.

Treatment protocols varied considerably with respect to chamber pressure (2.0–2.8 ATA), session duration (60–180 minutes), number of sessions, and frequency during the first 24–72 hours. Such variability limits direct comparison between studies and precludes quantitative synthesis.

Additionally, patient selection bias may have influenced reported outcomes, as individuals treated earlier or referred to centers with hyperbaric facilities may represent a subgroup with better prognostic characteristics. The absence of randomized controlled trials further constrains causal interpretation.

These methodological considerations underscore the need for standardized treatment pathways and prospective comparative studies to better define the therapeutic role of HBOT in acute retinal ischemia.

OVERALL STUDY CHARACTERISTICS AND OUTCOMES

Patients with retinal artery occlusion were frequently burdened with vascular comorbidities, including hypertension, diabetes mellitus, hyperlipidemia, and other cardiovascular diseases.

In CRAO, the effectiveness of HBOT was clearly dependent on treatment timing and anatomical factors. The greatest benefit was observed in patients treated early and in cases of incomplete occlusion or preserved macular perfusion via a cilioretinal artery.

In BRAO, the therapeutic response appeared more predictable, with visual improvement occurring more frequently and to a greater extent than in CRAO.

Available comparative studies suggest a higher likelihood of visual improvement with HBOT compared with conservative management; however, interpretation of these findings is limited by methodological heterogeneity. Adverse events were infrequent and generally mild.

DISCUSSION

The present review synthesizes the available clinical evidence on HBOT in acute retinal ischemia due to CRAO and BRAO. Overall, the available literature suggests that HBOT is associated with clinically meaningful visual improvement in a subset of patients, with the most consistent signal observed when treatment is initiated early after symptom onset. Patients treated later in the disease course were less likely to experience measurable visual gains, with outcomes more often limited to preservation of baseline visual acuity. These findings are congruent with experimental data on time-dependent retinal survival and with the physiological rationale of HBOT, namely enhancement of oxygen diffusion from the choroidal circulation to metabolically vulnerable inner retinal layers.

A second observation across the included evidence is the difference in outcomes between CRAO and BRAO. Patients with BRAO generally present with better baseline visual acuity, smaller ischemic burden, and residual perfusion from adjacent territories, which together likely contribute to more favorable and more consistent visual recovery compared with CRAO. In CRAO, outcomes remain highly heterogeneous. Several factors may modulate response, including baseline visual acuity, completeness of occlusion, and anatomical modifiers such as cilioretinal artery sparing, which may preserve macular oxygenation and increase the probability of recovery even when HBOT initiation is delayed. Notably, a small number of studies reported improvement beyond conventional “early” time windows, suggesting that retinal viability may be preserved in selected patients with incomplete ischemia or

residual perfusion; however, these observations should be interpreted cautiously due to the non-randomized nature of the evidence.

Comparative cohort studies included in this review provide indirect context regarding HBOT relative to conservative management and selected interventional strategies. In several comparative analyses, HBOT-treated patients more frequently achieved visual improvement than conservatively managed controls. Nevertheless, differences in baseline characteristics, timing to treatment, and protocol intensity substantially limit causal inference, and the observed effects may reflect selection bias favoring early presenters and centers with ready access to hyperbaric chambers. Similarly, comparisons with thrombolytic approaches remain inconclusive, given heterogeneity in treatment windows, patient selection, and outcome definitions across studies.

From a safety perspective, HBOT was generally well tolerated in the included reports, with few serious adverse events and predominantly mild complications when reported. Importantly, ocular neovascular complications observed after CRAO are consistent with the natural history of severe retinal ischemia and were not clearly attributable to HBOT. In clinical practice, the use of HBOT is primarily limited by organizational factors -

including restricted availability of hyperbaric chambers, delays in patient qualification and transport, and the lack of standardized treatment protocols. These circumstances hinder rapid initiation of therapy, influence treatment outcomes, and limit the ability to directly compare results across centers.

Taken together, the current evidence supports HBOT as a potentially beneficial adjunct in selected patients - particularly when treatment can be initiated promptly - while underscoring the need for standardized pathways and higher-quality comparative studies.

LIMITATIONS

The available evidence is largely based on retrospective studies and case series, with no randomized controlled trials, which limits causal inference. Marked heterogeneity in patient selection, treatment timing, HBOT protocols, and outcome definitions precludes quantitative synthesis. Potential selection and publication bias further limit generalizability. These constraints underscore the need for prospective, standardized studies.

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