REVIEW

Hyperbaric Oxygen Therapy in the Management of Dermal Filler-Induced Vascular Occlusion: A Scoping Review

by LUISA V. RAMIREZ, MD; EBONY P. GAYLE, MD; ANA ORMAZA, MD; SAMI K. SAIKALY, MD; ELISHA SINGER, MD; THAIS PINCELLI, MD; DEANNA C. MENAPACE, MD; COURTNEY L. JAMES-NEWSOME, MD; ROY S. ROGERS III, MD; ALISON J. BRUCE, MBChB; LEILA TOLAYMAT, MD; and MIGUEL A. ARISTIZABAL-TORRES, MD

Drs. Ramirez and Gayle are with ADEI, Aesthetics & Dermatology Institute, Bogota, Colombia. Dr. Ormaza is with the Department of Dermatology, Eastern Virginia Medical School, Norfolk, Virginia. Dr. Saikaly is with Rising Sun Dermatology & Cosmetics Center, St. Augustine, Florida. Drs. Singer, Pincelli, Bruce, Tolaymat, and Aristizabal-Torres are with the Department of Dermatology, Mayo Clinic, Jacksonville, Florida. Dr. Menapace is with the Department of Otolaryngology, Division of Facial Plastic Surgery, Mayo Clinic, Jacksonville, Florida. Dr. James-Newsome is with the Department of Emergency Medicine, Mayo Clinic, Jacksonville, Florida. Dr. Rogers is with the Department of Dermatology, Mayo Clinic, Scottsdale, Arizona.

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BACKGROUND: Hyperbaric oxygen therapy (HBOT) has been used in ischemic and inflammatory conditions due to its ability to enhance tissue oxygenation and support wound healing. Filler-induced vascular occlusion (FIVO) is a rare but potentially devastating complication of dermal filler injections that may result in skin necrosis or vision loss. HBOT has been increasingly reported as an adjunctive intervention in FIVO, but its reported use has not been systematically summarized. **OBJECTIVE:** To summarize the existing literature on HBOT use in FIVO, describe reported clinical contexts and treatment parameters, and identify gaps in current knowledge. **METHODS:** This scoping review was conducted in accordance with Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews guidelines. A comprehensive search of PubMed, MEDLINE, Embase, and Google Scholar identified case reports, case series, experimental studies, and reviews describing FIVO cases managed with HBOT. Articles published in English or Spanish between January 2011 and May 2025 were included. Data were synthesized descriptively. **RESULTS:** Twenty-four studies met inclusion criteria, consisting primarily of case reports and small case series. HBOT was most often used as an adjunct to established therapies, including hyaluronidase, antiplatelet agents, vasodilators, and thrombolytic therapy. Reported HBOT protocols varied substantially, with treatment pressures of 2.0 to 3.0 atmospheres absolute and session durations of 60 to 120 minutes. Outcomes were heterogeneous and frequently confounded by multimodal management. **CONCLUSION:** HBOT has been reported as an adjunctive intervention in selected cases of FIVO, but evidence remains limited to low-level observational data, highlighting the need for standardized protocols and prospective studies. **KEYWORDS:** Hyperbaric medicine, hyperbaric oxygen therapy, hyperbaric oxygen treatment, filler-induced vascular occlusion, derma filler, filler complications

Dermal fillers are widely used in minimally invasive cosmetic facial rejuvenation and have gained global popularity due to their biocompatibility, accessibility, and ease of use.[1,2] According to the 2023 Procedural Statistics Report by the American Society of Plastic Surgeons, approximately 25.4 million minimally invasive procedures were performed.[3] Among these, dermal fillers accounted for 3,441,534 procedures, representing a 4% increase from 2022.[3] This notable rise in frequency may also be accompanied by a corresponding increase in complications, underscoring the need for clinicians to have a broad and readily accessible vascular occlusion protocol.

Dermal fillers may be composed of various materials, including hyaluronic acid, poly-L-lactic acid, calcium hydroxyapatite, polycaprolactone, and others.[4,5] While generally considered safe, dermal fillers can occasionally lead to serious complications, most notably filler-induced vascular occlusion (FIVO).[6,7] FIVO refers to the obstruction of blood flow in areas treated with dermal fillers and is estimated to occur in approximately 0.01% to 0.05% of cases.[8] Although rare, FIVO can result in significant aesthetic and physiological consequences.[9,10] Despite its severity, FIVO remains underreported and insufficiently documented, emphasizing the importance of improved awareness, prevention strategies, and treatment protocols.[8,11]

Several treatment options for FIVO have been described, including vasodilators, hyaluronidase, antiplatelet agents, and corticosteroids. These therapies have demonstrated varying degrees of efficacy in restoring perfusion to ischemic tissues.[12,13] Given the potential limitations of current approaches, further exploration of adjunctive treatments is warranted. One such modality is hyperbaric oxygen therapy (HBOT), which may enhance oxygen delivery and thereby promote tissue viability in affected

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CORRESPONDENCE: Miguel A. Aristizabal-Torres, MD; Email: aristizabal.miguel@mayo.edu

TABLE 1. Search strategies for databases

DATABASE	SEARCH DATE	SEARCH TERMS
PubMed	May 2025	("hyperbaric oxygen therapy"[tiab] OR HBOT[tiab] OR "hyperbaric oxygen"[tiab]) AND ("dermal filler"[tiab] OR "filler-induced vascular occlusion"[tiab] OR FIVO[tiab] OR "vascular occlusion"[tiab] OR "skin ischemia"[tiab] OR "skin necrosis"[tiab])
Embase	May 2025	('hyperbaric oxygen therapy':ti,ab OR HBOT:ti,ab OR 'hyperbaric oxygen':ti,ab OR 'hyperbaric oxygen therapy'/exp) AND ('dermal filler':ti,ab OR 'filler induced vascular occlusion':ti,ab OR 'vascular occlusion':ti,ab OR 'skin ischemia':ti,ab OR 'skin necrosis':ti,ab)
MEDLINE	May 2025	("hyperbaric oxygen therapy" OR HBOT OR "hyperbaric oxygen") AND ("dermal fillers" OR "filler-induced vascular occlusion" OR "vascular occlusion" OR "skin ischemia" OR "skin necrosis")
Google Scholar	May 2025	"hyperbaric oxygen therapy" OR HBOT AND "dermal filler" OR "filler-induced vascular occlusion" OR "vascular occlusion"

[tiab] is a search abbreviation that searches within the citation's title and/or abstract.
/exp is a search abbreviation that expands the search to all other related terms.

areas. Owing to its ability to improve tissue oxygenation, reduce ischemia, and stimulate healing, HBOT has been proposed as a potential adjuvant treatment to minimize long-term sequelae in FIVO. However, the current evidence remains limited, primarily derived from case reports and small case series.[14–16] Therefore, the objective of this scoping review is to map the existing literature on the use of HBOT in FIVO, describe reported clinical contexts and treatment parameters, and identify knowledge gaps.

METHODS

Review design and reporting framework. This scoping review was conducted and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews (PRISMA-ScR) guidelines.

Eligibility criteria. Eligible sources of evidence included published case reports, case series, experimental studies, and review articles describing cases of FIVO in which HBOT was used as part of the management strategy. Articles published in English or Spanish between January 2011 and May 2025 were considered.

Studies were excluded if HBOT was not part of the treatment regimen for FIVO, if HBOT was used exclusively for non–filler-related dermatologic conditions (eg, chronic ulcers, scars, or graft compromise unrelated to dermal fillers), if insufficient clinical detail regarding HBOT use was provided, or if the report was unrelated to dermal filler complications.

Information sources and search strategy. A comprehensive literature search was performed using PubMed, MEDLINE, Embase, and Google Scholar. Search terms included combinations of keywords and subject headings related to HBOT and dermal filler complications, including "hyperbaric oxygen therapy," "HBOT," "dermal fillers," "filler-induced vascular occlusion," "vascular occlusion," "hyaluronic acid," "skin ischemia," "skin necrosis," "calcium hydroxyapatite," and "filler complications" (**Table 1**). Reference lists of included articles were manually reviewed to identify additional relevant sources. The literature search was conducted by L.V.R., E.P.G., A.O., and M.A.A.-T.

Study selection. All records identified through database searching were exported to a reference management system, and duplicate records were removed. Titles and abstracts were screened independently by 4 reviewers (L.V.R., E.P.G., A.O., M.A.A.-T.) to identify potentially relevant articles. Full-text articles were subsequently assessed for eligibility by 5 reviewers (L.V.R., E.P.G., A.O., R.S.R, M.A.A.-T.). Discrepancies were resolved through discussion and consensus, with consultation of a senior author (S.K.S., E.S., D.C.M., C.L.J.-N., R.S.R., A.J.B., L.L.T., M.A.A.-T.) when necessary. Reasons for exclusion at the full-text stage were documented.

Data charting process. Data were extracted using a standardized data charting form developed for this review. Extracted variables included author and year of publication, study design, type of dermal filler, clinical manifestations of FIVO (cutaneous and/or ocular), timing of HBOT initiation relative to symptom onset, number of HBOT sessions, chamber type (monoplace or multiplace), atmospheric pressure applied, duration of each session, concomitant therapies, and reported clinical outcomes. Extracted data were reviewed by all authors to ensure accuracy and completeness.

Synthesis of results. Data were synthesized descriptively to map patterns of HBOT use in the management of FIVO. Given the heterogeneity of study designs, interventions, and outcomes, no quantitative synthesis or meta-analysis was performed. Consistent with PRISMA-ScR guidance, no formal risk of bias assessment was undertaken.

RESULTS

Search results. The database search identified a total of 294 records across PubMed, MEDLINE, Embase, and Google Scholar. After removal of 55 duplicate records, 239 unique articles remained. Following exclusion of studies that addressed nondermatologic indications for HBOT or were unrelated to dermal filler complications, 144 records were screened based on title and abstract.

Seventy-eight full-text articles were assessed for eligibility. Four reports could not be retrieved. Of the remaining articles, 50 were excluded due to lack of relevance to FIVO or insufficient information regarding the use of HBOT. Ultimately, 24 studies met inclusion criteria and were included in this scoping review. The study selection process is summarized in the PRISMA-ScR flow diagram (**Figure 1**).

Characteristics of included sources. The 24 included studies consisted predominantly of case reports and small case series, with a limited number of narrative reviews and experimental reports. Publication years ranged from 2011 to 2025. Most reports described female patients undergoing cosmetic facial filler procedures, with hyaluronic acid being the most commonly implicated filler material.

Clinical presentations included cutaneous ischemia, skin necrosis, and, less frequently, ocular complications such as visual impairment or retinal ischemia. HBOT was consistently reported as an adjunctive intervention and was administered alongside established therapies, including hyaluronidase, antiplatelet agents, vasodilators, corticosteroids, and thrombolytic therapy. The extracted data from the included studies are presented in **Table 2**.

Overview of mapped evidence. Across the included sources, authors consistently described FIVO as resulting from either intravascular injection or external compression, leading to ischemia that may progress to tissue necrosis, ocular ischemia, or neurologic injury depending on the regions involved and the extent of anatomical compromise. The following

subsections summarize key concepts and clinical features reported in the literature to contextualize the use of HBOT.

Key concepts and clinical context. Dermal fillers are commonly used to enhance facial volume and to improve the appearance of rhytides.[17] Despite their widespread use and generally favorable safety profile, these agents carry a known risk of vascular complications.[18,19] Inadvertent intra- or perivascular injection may occur due to several leading factors such as injection technique, anatomical variations, and the physical characteristics of the filler material itself, including its composition, particle size, viscosity, and volume.[20,21] When a filler is unintentionally applied into or near a blood vessel, it can lead to mechanical obstruction of blood flow, which may result in tissue congestion or hypoxia and, in more severe cases, necrosis of the overlying skin and soft tissue.[6,22]

The risk of vascular compromise is heightened by the complex anatomy of the facial arterial system, which features an extensive network of anastomoses and collateral vessels.[23] These interconnections allow filler material to propagate unpredictably within the vasculature, even beyond the initial site of injection.[24] Once within the arterial system, intravascular filler particles can migrate and occlude smaller vessels, causing ischemia in more distal or even contralateral regions.[25,26] Embolization of filler material into critical vascular territories, such as the ophthalmic or cerebral arteries, can result in severe outcomes, including retinal ischemia, cerebral infarction, and stroke.[27,28]

FIVO has been described as a pathophysiological process comprising 4 progressive stages. The first stage, vasocannulation, involves mechanical penetration of the arterial wall by the needle or cannula. This is followed by vasoinoculation, in which the filler material enters the arterial lumen. Vasodissemination, the third stage, entails the dissemination of filler through anastomotic pathways to more distal vascular territories. Finally, vasoocclusion results in the obstruction of blood flow, culminating in tissue ischemia and necrosis.[29,30]

In addition to direct intravascular injection, vascular compromise may also occur through external compression. Large boluses of filler can exert extrinsic pressure on adjacent vessels, particularly in areas with limited tissue compliance, leading to perivascular obstruction.[31,32] Moreover, fillers may induce vasospasm, activate the coagulation cascade, and stimulate platelet aggregation and thrombosis.[20] These vascular responses are often accompanied by an inflammatory reaction mediated by cytokines, notably interleukin (IL) 6 and IL-8, which can further impair perfusion and oxygen delivery at the tissue level.[33]

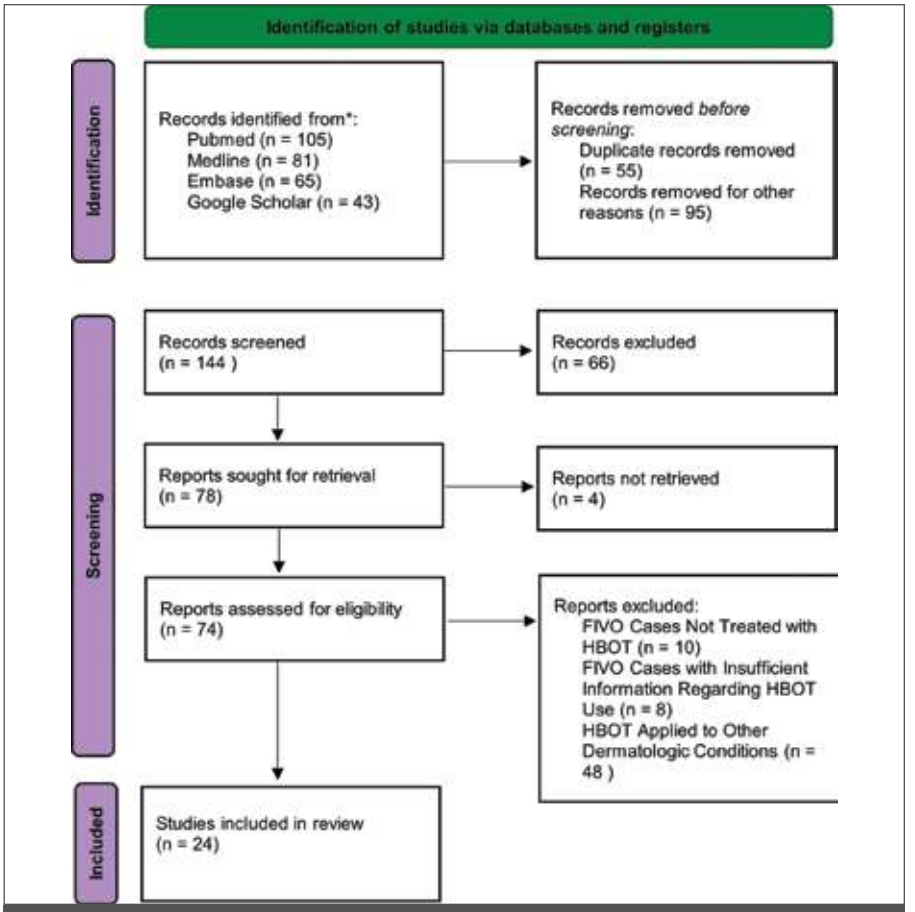

FIGURE 1. Preferred Reporting Items for Systematic Reviews and Meta-Analyses Extension for Scoping Reviews flow diagram illustrating the sources of evidence for this scoping review

Certain anatomical sites are recognized as being at a particularly high risk for vascular events due to their proximity to critical arteries and limited collateral circulation. These include the nasolabial folds, nasal dorsum, glabella, and forehead.[34,35] Risk is also influenced by procedural factors, including the volume of filler injected; volumes exceeding 0.1 mL are associated with increased complication rates.[17,36] The choice of delivery instrument can also increase risk. Needle-based injections and the use of small-diameter cannulas increase the likelihood of vascular penetration.[37,38] Additionally, prior scarring or fibrosis in the treatment area may distort normal anatomy and elevate the risk of vascular injury.[21,39]

Clinical manifestations of filler-induced vascular complications. Early signs and symptoms of FIVO that may precede ischemic skin necrosis include localized blanching, acute pain, livedo reticularis, and delayed capillary refill.[20,22,40,41] In contrast, venous congestion is more often characterized by a gradual onset of dusky, violaceous discoloration with a reticulated pattern, typically accompanied by mild swelling rather than acute pain. Bruising (ecchymosis), which may be mistaken for early vascular compromise, generally presents post procedure with well-demarcated purplish discoloration, is nonblanching, and lacks associated blanching or severe pain (**Table 3**).[42] Distinguishing these entities at the earliest stage is essential, as unrecognized or inadequately treated arterial ischemia may progress to epidermal and dermal disruption, culminating in full-thickness skin necrosis (**Figures 2A and 2B**).[21,28,43]

In addition to cutaneous complications, FIVO may result in devastating ophthalmic and

TABLE 2. Published case reports and case series on the use of HBOT in FIVO

AUTHOR	DEMOGRAPHICS	CLINICAL REMARKS	TIME TO TREATMENT	TREATMENT PARAMETERS	SESSIONS	OUTCOME	SEQUELAE/ COMPLICATIONS
Tsai et al[64]	43-year-old male	HA injection; hyperemia in left nasolabial fold and gingiva; violaceous discoloration on nasal tip	Not specified	HBOT administered daily at 2 to 2.5 ATA for 80 to 120 minutes	15	Complete recovery	None
Tsai et al[64]	23-year-old female	CH filler injection in area with previous filler rhinoplasty; full-thickness necrosis of nasal tip	Not specified	HBOT administered daily at 2.5 ATA for 80 to 90 minutes	2	Full-thickness necrosis of the nasal tip, requiring hospitalization for surgical debridement	None
Hwang[48]	43-year-old female	Fat injection; central retinal vein and cilioretinal artery occlusion, visual impairment in the left eye	Not specified	HBOT administered daily for 2 hours at 253 kPa (2.5 ATA) over a 14-day period	14	Left visual acuity returned to normal (from 20/200 to 20/20)	None
Henderson et al[68]	37-year-old female	Self-injected HA on both sides of the face near the temples; hearing loss in the left ear and ischemic changes on the face	16 hours post procedure	HBOT administered twice daily: the first 2 sessions at 3.0 ATA for 90 minutes, followed by 4 sessions at 2.4 ATA for 90 minutes, with air breaks every 30 minutes	6	After 3 days, the patient showed visible improvement with significantly reduced ischemic discoloration, and her hearing returned to baseline	None
Hong et al[70]	43-year-old female	HA filler injection into both nasolabial folds; necrosis of the left nasal ala	Within the first week post procedure	First HBOT session at 2.8 ATA for 135 minutes, followed by 42 sessions at 2.0 ATA for 110 minutes over 1 year	43	The wound fully healed with no further changes reported requiring surgical debridement	None
Ling[40]	41-year-old female	HA injections along nasal bridge; ischemia, blisters, edema, erythema, and dark-blue discoloration of the nose	1 week post procedure	HBOT administered at 2.0 ATA for 30 minutes	4	2 weeks post injection, blisters healed with minimal residual scarring	None
Uittenbogaard et al[22]	46-year-old female	CH injection for hollow temples; discoloration and numbness	7 days post procedure	HBOT in a multiplace chamber at 2.5 ATA for 90 minutes, including three 5-minute air breaks, administered once daily, 5 days per week	10	Resolution of pain, discoloration and numbness	None
Kruize et al[2]	43-year-old male	HA filler applied to the chin and nasolabial folds; facial artery occlusion	6 days post procedure	HBOT administered at 2.4 ATA for 80 minutes: first 2 days, twice daily; next 6 days, once daily	10	Wounds completely healed without any scarring	None
Kruize et al[2]	55-year-old female	HA injected into nose and nasolabial folds; occlusion of posterior buccal branch of maxillary artery	8 days post procedure	HBOT at 2.4 ATA for 80 minutes administered twice daily for the first 7 days; once daily during the following week, including the weekend	21	Wounds fully recovered	None
Zeltzer et al[35]	21-year-old female	HA injection into upper lip; leading to pain and ischemia	11 days post procedure	HBOT administered at 2.5 ATA (≈253 kPa) from Day 11 to 17: 70 minutes with 100% oxygen	6	Contracture and hypertrophy scar	Contracture and hypertrophic scar
Worley et al[25]	60-year-old female	HA injections in nasal crease, maxilla, and nasolabial folds; followed by keratitis diagnosis	Not specified	HBOT administered for 90 minutes	9	Keratitis nearly resolved	None
Alves et al[6]	39-year-old female	10% PMMA; resulting in hyperemia from chin to left nasal ala, suggesting facial artery occlusion	Within 24 hours post procedure	HBOT at 2.4 ATM in a multiplace chamber with for 90 minutes	5	Complete recovery	None

ATA: atmospheres absolute; CH: calcium hydroxyapatite; FIVO: filler-induced vascular occulsion; HA: hyaluronic acid; HBOT: hyperbaric oxygen therapy; PMMA: polymethylmethacrylate

TABLE 2 CONTINUED. Published case reports and case series on the use of HBOT in FIVO

AUTHOR	DEMOGRAPHICS	CLINICAL REMARKS	TIME TO TREATMENT	TREATMENT PARAMETERS	SESSIONS	OUTCOME	SEQUELAE/ COMPLICATIONS
Alves et al[6]	47-year-old female	10% PMMA injected through right oral commissure; followed by ischemic signs	3 days post procedure	HBOT at 2.4 ATA in a multiplace chamber for 90 minutes	7	Complete recovery	None
Simman and Bach[26]	30-year-old female	HA injection into chin; ischemia and mild smile asymmetry	2 days post procedure	HBOT at 2.5 ATA, discontinued due to COVID-19 infection	5	Complete skin remodeling and improvement in smile asymmetry after 3 weeks	None
Simman and Bach[26]	53-year-old female	HA injection into nasal tip; nasal tip skin necrosis	10 days post procedure	HBOT at 2.5 ATA, duration not specified	25	Improvement after 3 weeks with only minor residual pigmentation	Pigmentary disorder over the affected area
Oley et al[74]	32-year-old female	HA injections into nose; subsequent vascular occlusion	Not specified	HBOT sessions at 2.4 ATA for 90 minutes, twice weekly	Not specified	Noticeable improvement in the appearance of the nose after 1 month	None
Jalilian et al[7]	55-year-old female	HA filler injected bilaterally into nasolabial folds and chin; resulting in necrosis	5 days post procedure	HBOT at 2.5 ATA with 10-minute air breaks every 30 minutes, conducted in a dual-place chamber for 90 minutes	14	Improvement in skin lesions and near-complete resolution of mucosal necrosis, with some hypoesthesia on the left upper lip and a few small scars	Treatment discontinued due to middle ear barotrauma, which resolved favorably within a few days without treatment
Rodriguez-Valera and Nieto-Lopez[65]	Female, age not specified	HA injections into pyriform fossa and medial cheek fat; discoloration of nasolabial folds	4 hours post procedure	HBOT administered up to 3 ATA	5	Complete wound healing with no scarring	None
Rodriguez-Valera and Nieto-Lopez[65]	Male, age not specified	HA injection; necrosis in the middle cheekbone area	7 days post procedure	HBOT administered up to 3 ATA	5	Complete wound healing	None
Friedman et al[51]	38-year-old female	HA injection into left glabella and medial left eyebrow at home; sudden vision loss in the left eye	10 hours post procedure	HBOT session at 2.0 ATA for 90 minutes	3	Vision fully recovered to 20/20	None
Johnson-Arbor[41]	51-year-old female	PMMA injection into nasolabial folds; dusky discoloration and livedo changes in the right nasolabial area	Within the first week post procedure	Not specified	6	Improvement of discoloration	None
Madero Perez et al[14]	39-year-old female	HA injection to reduce glabellar wrinkles; leading to supratrochlear artery obstruction	6 days post procedure	HBOT in single-person chamber at 2.0 ATA for 45 minutes	10	At 3 months, minimal skin hyperpigmentation was observed, with no trophic changes or other symptoms	None
Stevens and Lewis[28]	Not specified	HA injection into glabellar wrinkles; supratrochlear artery obstruction	Not specified	HBOT at 284 kPa (2.8 ATA) over 7 days	9	Recovery	None
Cheng et al[47]	42-year-old female	Subdermal PDLLA injections to both cheeks, nasolabial folds, and forehead; sudden onset of right eye vision loss	3 days post procedure	HBOT administered twice daily for 14 days	28	Right eye visual acuity was limited to hand motion	Mild cataract formation

ATA: atmospheres absolute; CH: calcium hydroxyapatite; FIVO: filler-induced vascular occulsion; HA: hyaluronic acid; HBOT: hyperbaric oxygen therapy; PDLLA: poly-d,l-lactic acid; PMMA: polymethylmethacrylate

TABLE 3. Clinical differences among arterial ischemia, venous congestion, and bruising secondary to dermal fillers

FEATURE	ARTERIAL ISCHEMIA	VENOUS CONGESTION	BRUISING (ECCHYMOSIS)
Onset	Minutes after injection	Gradual (hours)	Delayed (hours to days)
Pain	Acute, severe pain disproportionate to clinical appearance; not universally present	Mild discomfort or pressure	Often only at time of injury
Color changes	Blanching → mottled/livedo reticularis	Dusky, violaceous, reticulated	Purplish/blue discoloration
Blanching	Present in early phase	Absent	Absent
Capillary refill	Delayed or absent	Normal or slightly delayed	Normal
Temperature	Cool to touch	Normal to slightly cool	Normal
Progression	Rapid → necrosis if untreated	Rarely progresses to necrosis	Resolves spontaneously
Associated findings	Possible paresthesia, tissue firmness	Mild swelling	Localized swelling possible

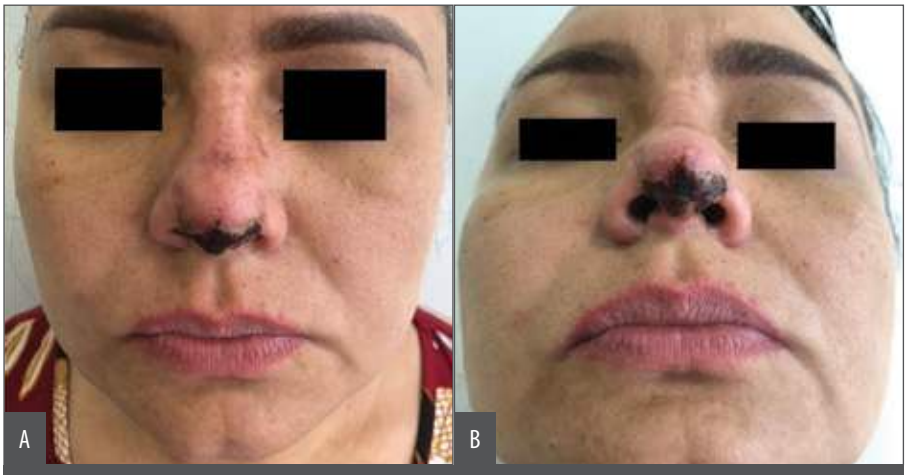

FIGURE 2. Cutaneous necrosis of the nasal area secondary to filler-induced vascular occulsion. (Courtesy of Dr. German Espinosa, all rights reserved.)

neurologic outcomes. A recent comprehensive literature review identified 365 cases of partial or complete vision loss following dermal filler injections.[44] These complications were most frequently associated with injections in high-risk anatomical regions, particularly the glabella, forehead, and nasal dorsum, where critical vessels are closely linked to the ophthalmic arterial system.[34,45,46] The underlying mechanisms of vision loss included occlusion of the ophthalmic artery, central retinal artery, branch retinal arteries, and posterior ciliary arteries.[47,48]

Clinical manifestations of filler-associated ocular ischemia included sudden-onset partial or complete vision loss, visual field deficits, afferent pupillary defects, ophthalmoplegia, blepharoptosis, and, in some cases, neurologic symptoms such as ptosis, nausea, vomiting, and headache.[27] Rarely, patients exhibited stroke-like symptoms, brain infarction, hemiplegia, or limb weakness due to retrograde embolization of dermal fillers from the injection area into the internal carotid artery and cerebral circulation. This may progress to postinfarction hemorrhage and carries a high risk of severe brain injury or death.[44,49] Vision loss typically occurred immediately or within 10 minutes following the injection, highlighting the need for prompt recognition and urgent intervention. Outcomes ranged from irreversible vision loss to partial or, in some cases, complete visual recovery.[44,50,51]

Management of filler-induced vascular occlusion. Prompt recognition and intervention are critical in cases of FIVO to minimize tissue ischemia and prevent irreversible complications.[11] The immediate priority is to stop injection of the filler at the first sign of vascular compromise. Warm compresses should be applied to promote vasodilation and improve local tissue perfusion. In any case of stroke or visual symptoms, urgent referral to a neurologist, ophthalmologist, or retinal specialist is mandatory. Multidisciplinary management, including neurology, ophthalmology, and plastic surgery, may be necessary for optimal outcomes.[52] Although the standard therapeutic window for effective ocular reperfusion is generally considered to be up to 90 minutes, some reports suggest that retinal ischemia may become irreversible in as little as 10 to 15 minutes.[29,34,44]

For FIVO involving hyaluronic acid fillers, prompt administration of hyaluronidase is the cornerstone of treatment.[5] Enzymatic degradation of the filler may be achieved through multiple high-dose intradermal or subcutaneous injections in the affected area. In severe cases, including vision loss, hyaluronidase has been administered into the retrobulbar, supratrochlear, or periorbital regions, and in some cases, both hyaluronidase and thrombolysis have been delivered via intra-arterial injection.[40,53–56] A report described a 27-year-old woman with complete vision loss following nasal hyaluronic acid injection. Intra-arterial hyaluronidase led to partial recovery, and subsequent thrombolysis with alteplase and hyaluronidase achieved ophthalmic artery recanalization and further improvement.[57]

Adjunctive therapies may include systemic or topical vasodilators such as nitroglycerin paste or oral tadalafil or sildenafil to enhance microcirculation, although care must be taken to avoid excessive vasodilation, which may inadvertently promote migration of filler material to other vascular territories.[31,58,59] Additional pharmacologic interventions include the use of antiplatelet agents such as aspirin, corticosteroids to mitigate inflammation, and anticoagulants such as low-molecular-weight heparin. In select cases, thrombolytic agents like alteplase have been considered.[20,21,23] Ultrasound assessment may be performed following emergency intervention to evaluate vascular competence in potentially compromised areas.[23]

HBOT is an emerging and scientifically supported adjunctive treatment (**Table 2**). It is particularly beneficial in improving tissue oxygenation in both early and delayed presentations of vascular occlusion.[7,15] HBOT may help reduce tissue hypoxia, modulate

inflammation, and support neovascularization, especially when used alongside conventional therapies.[26]

HBOT in the setting of filler-induced vascular occlusion. HBOT represents a valuable adjunctive modality in the treatment of FIVO, particularly when tissue ischemia is prolonged. Under physiological conditions, oxygen is primarily transported in the blood bound to hemoglobin, with only a small fraction dissolved in the plasma.[15,60] The therapeutic effect of HBOT can be understood through Henry's law, which states that the amount of gas dissolved in a liquid is directly proportional to the pressure applied to it.[14,16] Accordingly, breathing 100% oxygen under elevated atmospheric pressure increases the concentration of dissolved oxygen in plasma, potentially exceeding the oxygen-carrying capacity of hemoglobin.[61–63]

At sea level (1 atmosphere absolute [ATA]), nearly all oxygen is bound to hemoglobin, with plasma containing only about 0.32% dissolved oxygen.[15,64] Breathing 100% oxygen at 1 ATA increases this value to approximately 2.09%, and, at 3 ATA, the dissolved oxygen concentration can reach 6.8%, over 21 times higher than normobaric conditions.[25,62] Arterial oxygen tensions during HBOT may rise to levels as high as 2,000 mm Hg.[14] This hyperoxygenation promotes oxygen delivery even in areas with impaired microcirculation, thereby preventing tissue hypoxia and necrosis.[41,65]

HBOT is delivered in specialized single- or multiperson chambers where patients inhale 100% oxygen in a pressurized environment.[66] The minimum therapeutic pressure for HBOT is typically 1.4 ATA, though elective treatments are generally conducted at 2 to 3 ATA for durations of 60 to 120 minutes, depending on clinical indications.[62,67,68] In dermatology, plastic surgery, and aesthetic medicine, HBOT has been used successfully for ischemic ulcers, compromised flaps and grafts, posthair transplantation recovery, and keloid scars.[2,22] Its primary goal is to improve oxygenation in hypoxic tissues, reduce inflammation, enhance regeneration, and prevent necrosis.[61,69]

Tissue oxygen pressures of at least 30 mm Hg are considered necessary for adequate wound healing.[64] HBOT raises the partial pressure of oxygen in the blood and tissues, supporting oxygen-dependent cellular processes and the recovery of mitochondrial function even when arterial perfusion is inadequate, as in cases of FIVO.[33,39,45] Additionally, HBOT exerts effects on erythrocytes by promoting osmotic fluid loss, leading to shrinkage and deformation of red blood cells.[27] This may enhance microvascular flow and tissue oxygenation.[60,64]

Another relevant mechanism is the Robin Hood effect, wherein hyperoxia-induced vasoconstriction occurs in normoxic tissues but not in hypoxic regions.[51,65] This differential response leads to a redistribution of blood flow toward ischemic areas. Similarly, HBOT reduces interstitial edema by decreasing capillary hydrostatic pressure, consistent with Starling's law, while elevating plasma oncotic pressure, thereby promoting reabsorption of fluid from the interstitial space.[14,60,66]

HBOT also demonstrates bactericidal and immunomodulatory effects. High oxygen concentrations are toxic to anaerobic bacteria and improve leukocyte function by enhancing phagocytosis and oxidative burst capacity.[51,70,71] Inflammatory signaling is also modulated through suppression of proinflammatory cytokines, such as IL-1, IL-6, IL-8, and tumor necrosis factor α, while levels of the anti-inflammatory cytokine IL-10 are increased.[33,68,72]

The upregulation of hypoxia-inducible factor (HIF) 1, a transcription factor critical to wound healing, is also another key mechanism.[60] HIF-1 promotes keratinocyte migration and epithelial regeneration and induces the production of several prorepair growth factors, including vascular endothelial growth factor, platelet-derived growth factor, and stromal cell-derived factor.[33,40,73] These signaling molecules enhance neovascularization, erythropoiesis, osteogenesis, fibroblast activation, and collagen synthesis, key processes for tissue recovery in hypoxic or ischemic environments.[2,14,47,61]

HBOT has been successfully used for the salvage of compromised skin flaps and grafts.[16,69] The European Underwater and Baromedical Society recommends initiating HBOT immediately for such cases, using pressures of 2 to 2.5 ATA for 60 to 120 minutes per session.[17] Initial treatment typically includes 2 to 3 sessions daily, followed by 1 to 2 sessions per day until clinical improvement is observed or necrosis resolves.[26,66]

In ophthalmology, HBOT has also been applied in the treatment of central retinal artery occlusion (CRAO) related to filler embolization.[15,65] The therapy improves oxygenation of the retina and macula, particularly in hypoperfused regions affected by interstitial edema.[15,25] In the first 10 minutes of HBOT, vasoconstriction may occur, but this is soon followed by vasodilation mediated by increased nitric oxide production. The choroidal circulation, which supplies approximately 60% of the oxygen delivered to the retina, plays a critical role during CRAO. HBOT can augment oxygen delivery from choroidal and collateral vessels to maintain retinal metabolism until reperfusion occurs.[27,34] Adequate oxygenation of the inner retinal layers is critical to preserving vision, and early HBOT may help mitigate ischemic injury.

Although HBOT is considered an adjunctive therapy, its timely initiation is crucial for maximizing benefit in both cutaneous and ocular FIVO.[73] Incorporating HBOT into a multidisciplinary management plan may significantly improve clinical outcomes in patients at risk of tissue necrosis or permanent vision loss.[68,70]

Although generally well tolerated, HBOT carries potential risks due to the elevated pressures and increased oxygen concentrations. The most common adverse effects are related to barotrauma and include ear and sinus discomfort, middle ear injuries such as tympanic membrane rupture, and, less frequently, sinus squeeze or barotitis.[67,68] More serious complications include arterial gas embolism, pulmonary barotrauma, and, in rare instances, lung collapse, pulmonary oxygen toxicity, or pulmonary edema.[66]

One of the key mechanisms underlying HBOT-related toxicity is oxidative stress from excessive production of reactive oxygen species. This can contribute to cellular and tissue damage, including ocular effects such as lens toxicity and cataract development, particularly after prolonged exposure or cumulative treatment sessions exceeding 100.[45,47] Although uncommon, central nervous system oxygen toxicity may also occur and can manifest as tonic-clonic (grand mal) seizures during or shortly after treatment.[62]

To mitigate the risk of adverse events, treatments are generally limited to pressures below 3 ATA and durations of less than 2 hours per session.[42] Careful patient selection, pretreatment screening (eg, for existing pulmonary or otolaryngologic conditions, such as pneumothorax and eustachian tube

dysfunction, respectively), and close monitoring during therapy are essential for safety.[73]

Another critical concern is the risk of fire due to the high oxygen concentrations in HBOT chambers. To mitigate this risk, the US National Fire Protection Association mandates strict operational and engineering safety standards in all HBOT facilities.[16,65] These guidelines include rigorous protocols for ignition source control, patient attire, chamber construction materials, and staff training.

Contraindications to HBOT. HBOT is contraindicated in cases of untreated pneumothorax due to the risk of gas expansion during decompression, according to Boyle's law.[66] The use of certain chemotherapeutic agents, such as doxorubicin or cisplatin, is also considered an absolute contraindication because of the potential for enhanced oxidative tissue damage.[45,65]

HBOT can lead to fluid shifts from the third space into the intravascular compartment under elevated pressure, potentially exacerbating congestive heart failure.[6] Additionally, the vasoconstrictive effects of hyperoxia may increase afterload, further complicating cardiac function.[51] As such, HBOT should be administered with caution in patients with known heart failure.[51,62]

Other relative contraindications include pregnancy, epilepsy, upper respiratory infection, chronic obstructive pulmonary disease, asthma, pulmonary bullae, optic neuritis, claustrophobic individuals, and recent thoracic surgery.[69,72]

GUIDELINES AND FUTURE DIRECTIONS

HBOT has emerged as a valuable adjunct in the management of FIVO and has been incorporated into treatment protocols reported in multiple case reports and case series (**Table 2**). However, despite encouraging clinical outcomes, further rigorous controlled studies are needed to establish optimal timing, dosing, and treatment parameters that influence efficacy. Available data suggest that HBOT should ideally be initiated within 24 hours of suspected FIVO to maximize therapeutic benefit. In cases involving ocular compromise, particularly CRAO, time is even more critical.[55] The retina can typically tolerate no more than 90 to 100 minutes of ischemia before irreversible damage occurs, underscoring the importance of immediate ophthalmologic intervention and the potential use of subsequent HBOT.[15,25,45]

In the studies reviewed, the majority of reported cases involved female patients, predominantly in the third to fifth decades of life. The timing of intervention varied considerably across cases, ranging from within the first 24 hours to as late as 11 days postprocedure. In several reports, the exact timing of treatment initiation was not specified. Most cases demonstrated complete clinical improvement without residual sequelae. However, some cases reported persistent complications, including scarring, hypoesthesia, hyperpigmentation, or visual acuity impairment, in which treatment was initiated 11, 5, 3, and 6 days after the procedure, respectively. These findings suggest that earlier initiation of therapy may be associated with greater clinical benefit and a reduced risk of long-term sequelae. HBOT was generally administered on a daily basis at pressures ranging between 2.0 and 3.0 ATA, with session durations of 60 to 120 minutes, most commonly 90 minutes. The total number of sessions varied depending on clinical response, disease severity, and patient compliance. Importantly, in most cases, patients did not develop functional or anatomical sequelae; however, treatment was multimodal, with HBOT used as an adjunct to other therapies such as hyaluronidase, antiplatelet agents, vasodilators, and thrombolytic therapy.

Based on the author's clinical experience, a preliminary protocol of 5 to 15 consecutive sessions of HBOT at 2 to 3 ATA, with 100% oxygen administered for at least 60 minutes per session, combined with daily clinical assessments of the affected area, may offer therapeutic benefit.

Despite these promising mechanistic and clinical findings, HBOT use in FIVO is limited by several challenges, and it is important to acknowledge the presence of several confounding factors when interpreting its role. There is no standardized treatment protocol specific to FIVO, and most current recommendations are extrapolated from protocols for compromised grafts, flaps, or other ischemic injuries. The optimal timing, pressure, session duration, and number of treatments remain undefined, and practice patterns vary widely across institutions. The available evidence is largely limited to case reports, small case series, and retrospective analyses, with a paucity of prospective studies or randomized controlled trials to confirm efficacy, determine cost effectiveness, and define patient selection criteria. Furthermore, most reported cases of FIVO have been managed using a multimodal therapeutic approach, including hyaluronidase, antiplatelet agents, thrombolytic therapy, and HBOT administered concurrently. As a result, clinical improvement cannot be attributed solely to HBOT, making it difficult to determine its isolated therapeutic effect. Consequently, HBOT has been applied as an adjunctive treatment, with reported benefits observed in combination with other interventions rather than as a monotherapy. HBOT should not replace first-line interventions such as prompt hyaluronidase administration and urgent ophthalmologic referral in the case of ocular compromise; its role as an adjunctive therapy warrants further clarification. Additional barriers include limited access in nonhospital or aesthetic practice settings, which can delay initiation, as well as the high cost of HBOT, limited insurance coverage, and the absence of robust data on long-term outcomes.

Future research should prioritize the development of standardized, evidence-based HBOT protocols for FIVO, the conduct of multicenter prospective studies or randomized trials to assess efficacy and safety, and the identification of patient- and procedure-specific factors predictive of treatment response. Such efforts are essential to optimize clinical outcomes and establish the definitive role of HBOT in managing filler-induced vascular complications.

CONCLUSION

The management of FIVO remains a complex, time-sensitive challenge that demands both technical expertise and rapid clinical decision-making. Successful outcomes depend on a thorough understanding of facial vascular anatomy, recognition of high-risk injection zones, and immediate initiation of appropriate diagnostic and therapeutic measures. Early diagnosis and prompt intervention are critical to minimizing ischemic injury and preserving tissue viability.

As a scoping review, this study does not aim to determine efficacy but rather to summarize how HBOT has been reported and applied in clinical practice. By analyzing reported patient outcomes in cases where HBOT was used as an adjunctive treatment, this review seeks to clarify its potential therapeutic contribution within a multimodal management approach. In doing so, this study aims to synthesize currently fragmented evidence, highlight existing knowledge gaps, and encourage further investigation into HBOT as a supportive intervention. Given the increasing and widespread use of facial fillers in aesthetic practice, along

with the rising incidence of associated vascular complications, a clearer understanding of adjunctive therapies is clinically relevant.

Recognizing the limitations of this study, the available evidence is derived exclusively from case reports and case series, and no well-designed prospective or randomized studies currently exist. As such, this review represents a systematic synthesis of the existing literature rather than definitive evidence of efficacy, underscoring the need for future well-designed prospective studies to better define the role of HBOT in FIVO managemen and optimal treatment parameters, assess safety, and clarify the role of HBOT in managing vascular complications related to dermal fillers. Ultimately, this review intends to inform clinical decision-making, promote future research, and support strategies that may help reduce functional and anatomical sequelae associated with FIVO.

While HBOT is not a substitute for first-line measures such as hyaluronidase administration and urgent ophthalmologic referral, preliminary clinical observations support its role as a valuable adjunct in both early and select delayed presentations of FIVO. Its potential to improve oxygen delivery, attenuate inflammation, and support tissue repair warrants consideration in refractory or severe cases. Clinicians should be aware of the location and accessibility of nearby HBOT facilities to avoid delays in initiation.

Until standardized, evidence-based protocols are established, the integration of HBOT into the therapeutic algorithm for FIVO should be guided by multidisciplinary collaboration, individualized treatment planning, and ongoing clinical reassessment.

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