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Hyperbaric Oxygen Therapy in Liver and Kidney Transplantation

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Abstract

Ischemia-reperfusion injury remains a major determinant of early and late graft outcomes after liver and kidney transplantation. Hyperbaric oxygen therapy (HBOT), the delivery of 100% oxygen at pressures above 1 atmosphere absolute (ATA), increases dissolved plasma oxygen independent of hemoglobin and has antioxidant, anti-inflammatory, and mitochondria-protective effects in experimental models. This narrative review summarizes the physical principles and safety of HBOT, the biologic rationale for its use in transplantation, and the preclinical and clinical evidence in liver and kidney recipients. Literature was identified through a structured search of PubMed/MEDLINE (Medical Literature Analysis and Retrieval System Online), Embase, and the Cochrane Library and synthesized qualitatively. Animal models of hepatic and renal ischemia-reperfusion injury consistently show protection, but benefit depends on timing and is greatest when oxygen is delivered during ischemia or as preconditioning, a window that recipient HBOT rarely reaches. Clinical experience is limited to case reports and small series, mostly describing salvage treatment of hepatic artery thrombosis, and no randomized trial of recipient HBOT exists in either organ. Oxygenation of the isolated graft by machine perfusion, by contrast, has entered clinical practice with randomized evidence, and hyperbaric preservation remains experimental. HBOT also has an established adjunctive role in conditions that affect transplant recipients, such as necrotizing soft-tissue infections and compromised wounds. Clinical application to the graft is limited by the mismatch between the protective window and post-transplant delivery, recipient instability, interactions with immunosuppression, chamber access, and the lack of standardized protocols. Recipient HBOT therefore remains biologically plausible but clinically unproven. Its most realistic roles are as a selective salvage therapy for arterial complications, an adjunct for established indications, and a possible complement to ex vivo graft oxygenation, to be tested in prospective studies that define timing, dose, and patient selection.

Categories: Preventive Medicine, Transplantation, Medical Physics
Keywords: hepatic artery thrombosis, hyperbaric oxygen therapy, ischemia-reperfusion injury, kidney transplantation, liver transplantation, machine perfusion

Introduction And Background

Liver and kidney transplantation are definitive therapies for end-stage organ disease, yet ischemia-reperfusion injury (IRI) continues to shape early and long-term graft outcomes despite advances in surgical technique and perioperative care [1-3]. IRI begins with metabolic depletion, oxidative stress, and mitochondrial dysfunction during procurement and preservation, and is amplified at reperfusion [4,5]. In the liver, IRI contributes to early allograft dysfunction, vascular complications, and ischemic cholangiopathy; in the kidney, it manifests as delayed graft function (DGF), acute tubular necrosis, and a higher risk of rejection [6-8]. Because injury accumulates across several phases, from donor management through cold storage to reperfusion and recovery, interventions differ in when and where they can act.

Hyperbaric oxygen therapy (HBOT) is the administration of 100% oxygen at pressures above 1 atmosphere absolute (ATA). It increases dissolved plasma oxygen independent of hemoglobin [9,10] and has shown immunomodulatory and cytoprotective effects in experimental settings [11,12]. These properties have prompted interest in HBOT as an adjunct in transplantation, although its clinical role is undefined. At the same time, oxygenation of the graft itself has moved into routine practice through machine perfusion, which raises the question of what, if anything, systemic hyperbaric oxygen can add.

This narrative review summarizes the physical principles and safety of HBOT, the biologic rationale for its use against IRI, and the preclinical and clinical evidence in liver and kidney transplantation. It also examines hyperbaric approaches to organ preservation, the use of HBOT for post-transplant complications beyond IRI, the practical barriers to clinical application, and priorities for future research. Machine perfusion is discussed separately, as a comparator supported by a different level of evidence.

Literature search strategy

Although this is a narrative review, the literature search was structured, and its methods are reported to promote transparency. PubMed/MEDLINE (Medical Literature Analysis and Retrieval System Online), Embase, and the Cochrane Library were searched from inception to [month year], supplemented by Google Scholar and manual screening of the reference lists of key articles. Search terms combined "hyperbaric oxygen", "hyperbaric oxygenation", or "HBOT" with "liver transplantation", "kidney transplantation", "ischemia-reperfusion injury", "hepatic artery thrombosis", "organ preservation", and "machine perfusion".

Eligible publications were English-language preclinical studies, clinical reports, and reviews evaluating HBOT or hyperbaric oxygenation in liver or kidney transplantation, hepatic or renal IRI, liver regeneration, or organ preservation. Randomized trials, meta-analyses, and large cohort studies of oxygenated machine perfusion were included as a comparator, and selected publications on the physiology, safety, accepted indications, and immunologic effects of HBOT were included where relevant to transplant recipients. Conference abstracts without full text, non-English publications, duplicate reports, and studies of HBOT in other organs without relevance to IRI or transplant recipients were excluded. Titles and abstracts were screened, and full texts of potentially relevant articles were assessed, by two authors independently, with disagreements resolved by discussion. Given the heterogeneity of the evidence, findings were synthesized qualitatively and no quantitative pooling was attempted.

Review

Pathophysiology of IRI in liver and kidney grafts

Phases of Injury

IRI in transplantation unfolds across distinct phases, each with its own dominant mechanisms. Donor instability and, after donation after circulatory death (DCD), a period of warm ischemia start the process before procurement. Cold storage then slows but does not stop metabolism: adenosine triphosphate (ATP) is progressively depleted, ion pumps fail, and metabolic intermediates accumulate [2,4]. Rewarming during implantation and the restoration of blood flow at reperfusion convert this latent injury into active damage through oxidative stress, release of damage-associated molecular patterns, and recruitment of innate immune cells [5,13]. The magnitude of injury therefore depends not only on the duration of each phase but also on the metabolic state of the graft when oxygen is reintroduced (Figure *1*).

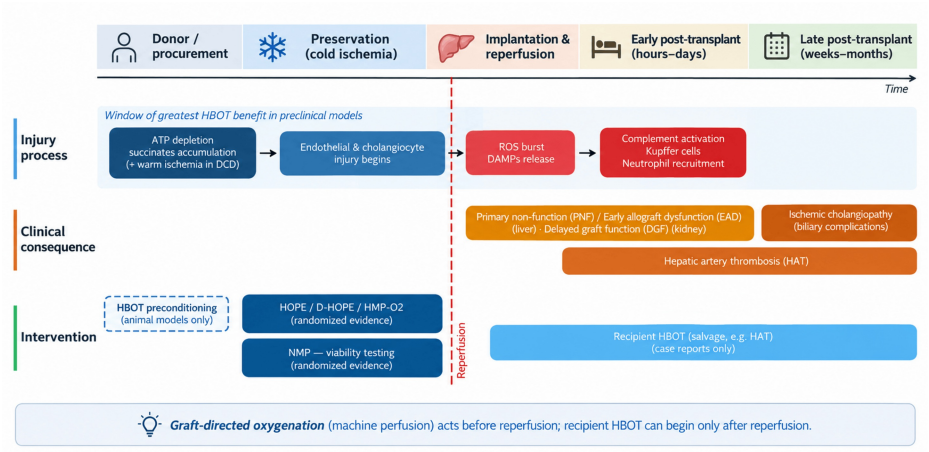


FIGURE 1: Timeline of ischemia-reperfusion injury and the windows in which graft oxygenation strategies act

Injury begins during procurement and preservation, when ATP is depleted and succinate accumulates, and is amplified at reperfusion by a burst of reactive oxygen species and innate immune activation. Machine perfusion oxygenates the graft before reperfusion, whereas recipient HBOT can begin only after implantation. In preclinical models, HBOT is most protective when given during ischemia or as preconditioning, that is, before reperfusion (to the left of the red dashed line), a window rarely accessible in clinical transplantation.

DAMPs: damage-associated molecular patterns; DCD: donation after circulatory death; DGF: delayed graft function; D-HOPE: dual hypothermic oxygenated perfusion; EAD: early allograft dysfunction; HAT: hepatic artery thrombosis; HBOT: hyperbaric oxygen therapy; HMP-O2: oxygenated hypothermic machine perfusion (kidney); HOPE: hypothermic oxygenated perfusion; NMP: normothermic machine perfusion; PNF: primary non-function; ROS: reactive oxygen species.

Image Credit: Authors; an initial draft was generated in Python (matplotlib library; Python Software Foundation, Wilmington, Delaware, United States) with the assistance of Claude (Anthropic, PBC, San Francisco, California, United States), and the final figure was rendered with ChatGPT (OpenAI, San Francisco, California, United States).

Liver-Specific Features

In the liver, sinusoidal endothelial cells and cholangiocytes are more vulnerable to cold ischemia than hepatocytes. Kupffer cell activation, complement, and neutrophil infiltration amplify injury after reperfusion, and loss of the endothelial glycocalyx impairs sinusoidal flow [5,14,15]. Clinically, IRI contributes to early allograft dysfunction, primary non-function, and ischemic cholangiopathy, the latter particularly after DCD donation [16]. Beyond the early period, hepatic IRI has been linked to longer-term graft injury and, in recipients transplanted for hepatocellular carcinoma, to tumor recurrence [3].

Kidney-Specific Features

In the kidney, the proximal tubular epithelium, with its high metabolic demand, bears the brunt of ischemic injury. Tubular cell death through apoptosis and regulated necrosis, complement activation, and endothelial dysfunction combine to produce DGF [7,8,17]. DGF prolongs hospitalization and dialysis dependence, and IRI increases graft immunogenicity, contributing to acute rejection and chronic graft injury [8]. Kidneys from older and DCD donors are most susceptible, which explains why they are the focus of preservation trials [18].

These organ-specific features define what an oxygen-based intervention would need to achieve: limit ATP depletion and succinate accumulation before reperfusion, protect endothelial and biliary or tubular cells, and blunt the inflammatory response that follows.

Principles and practice of HBOT

At sea level, nearly all arterial oxygen is carried by hemoglobin, and dissolved oxygen contributes only about 0.3 mL/dL. Because the amount of gas dissolved in plasma is proportional to its partial pressure, breathing 100% oxygen in a pressurized chamber raises arterial oxygen tension more than 10-fold and increases dissolved oxygen in proportion [9]. At 3.0 ATA, dissolved oxygen alone approaches the resting oxygen extraction of most tissues, independent of hemoglobin concentration or red-cell flow [9,10]. This hemoglobin-independent reserve is the physiologic basis for the use of HBOT in severe anemia when transfusion is not possible, where it has been proposed as a temporary bridge [19].

With the patient breathing 100% oxygen at 3.0 ATA, arterial PO2 approaches 2,200 mmHg and dissolved oxygen contributes about 6.6 mL/dL. At a hemoglobin of 5 g/dL, this equals roughly half of total arterial oxygen content; at 10 g/dL, it is still about one-third. In anemic or poorly perfused recipients, arterial PO2 is therefore the main modifiable determinant of oxygen content short of transfusion.

Delivery and Accepted Indications

HBOT is delivered in monoplace chambers, which treat one patient pressurized with oxygen, or multiplace chambers, in which patients breathe oxygen by mask or hood while attendants remain inside. Multiplace chambers allow ventilation, invasive monitoring, and resuscitation and are therefore preferred for critically ill patients [20]. Treatment pressures range from 2.0 to 3.0 ATA depending on the indication, with sessions of 60 to 120 minutes given once or twice daily [9,21]. The European Committee for Hyperbaric Medicine recommends HBOT for conditions including carbon monoxide poisoning, decompression illness, necrotizing soft-tissue infections, compromised skin grafts and flaps, and late radiation injury; neither organ transplantation nor hepatic artery thrombosis appears among accepted indications [22]. Any transplant application is therefore off-label and investigational.

Safety profile

HBOT is generally well tolerated, but its adverse effects are relevant to transplant recipients. Middle-ear barotrauma is the most frequent complication and is usually mild. Central nervous system oxygen toxicity with seizures is uncommon, occurring in roughly one in 2,000 to one in 10,000 treatments. Reversible myopia develops with prolonged courses, and pulmonary edema has been reported in patients with reduced left ventricular function. Hypoglycemia can occur in patients with diabetes, and an unvented pneumothorax is an absolute contraindication [22]. Each of these risks is magnified in recently operated, immunosuppressed recipients, a point developed further below.

Biologic rationale for HBOT in transplantation

Oxygen Delivery and Microcirculatory Support

HBOT raises plasma oxygen tension many-fold, which may matter most in grafts with endothelial injury, microvascular dysfunction, and capillary no-reflow [5,8]. Because dissolved oxygen diffuses independently of red-cell flow, HBOT may support ischemic tissue even when perfusion is impaired, including tissue supplied by thrombosed or narrowed arteries.

Oxidative Stress and Inflammatory Modulation

Although hyperoxia can generate reactive oxygen species (ROS), controlled HBOT exposure appears to induce adaptive antioxidant responses. Experimental and clinical studies show reduced proinflammatory cytokines and modulation of NF-κB (nuclear factor kappa-light-chain-enhancer of activated B cells)-dependent signaling [11,23,24], effects that may blunt innate immune activation after reperfusion. Whether HBOT meaningfully modulates the transplant-specific pathways described above, including complement activation, Kupffer cell priming, selectin- and ICAM-1 (intercellular adhesion molecule-1)-mediated leukocyte adhesion, and glycocalyx shedding, is unknown [15,16].

Mitochondrial Preservation and Cell Death

Mitochondrial dysfunction is central to IRI. During ischemia, succinate accumulates and is rapidly re-oxidized at reperfusion, driving a burst of mitochondrial ROS [14]. In liver and kidney models, HBOT has been associated with preserved ATP production, reduced mitochondrial permeability transition, and less apoptosis, correlating with reduced tissue injury and better functional recovery [25,26]. Regulated necrosis, including necroptosis, also contributes to renal IRI [18], but whether HBOT acts on these pathways has not been established.

Adaptive Immunity and Alloimmune Responses

A recurring hypothesis is that HBOT might dampen alloimmune responses and so reduce rejection. Experimental data do not support HBOT as an immunosuppressant on its own: in a highly immunogenic mouse skin-graft model, two different HBOT regimens did not delay rejection, and the authors suggested that any effect might only appear in combination with conventional immunosuppression [27]. In contrast, peri-transplant HBOT improved graft function, graft insulin content, and, in one regimen, beta-cell mass after islet transplantation in diabetic mice, consistent with relief of early graft hypoxia rather than immunomodulation [28]. These observations suggest that the most plausible benefit of HBOT in solid-organ transplantation lies in limiting hypoxic and reperfusion injury, not in preventing rejection.

HBOT in liver transplantation

Preclinical Evidence

Animal models provide the strongest support for HBOT in hepatic IRI; key preclinical and clinical studies are summarized descriptively in Table *1*. In a rat liver transplant model, post-transplant HBOT reduced hepatocyte necrosis and apoptosis and improved microvascular architecture and regeneration [25]. Preconditioning studies in hepatic IRI show similar protection, mediated in part by heme oxygenase-1 and heat shock protein 70 [29-31]. In a model comparing exposure windows, the effect of HBOT depended on when it was given [32] (Table *1*), a finding discussed further below. Heterogeneous pressures, durations, and endpoints make translation difficult.

Study (author, year)	Setting	Design	Intervention	Key findings
Tran et al., 2012 [25]	Liver, preclinical	Rat orthotopic liver transplant	HBOT 2.5 ATA, 90 min, at 12 and 48 h post-transplant	At 12 h: necrosis 18.7% vs 2.4%, apoptosis 6.4% vs 1.6%, ALT 1,068 vs 370 IU/L; improved microvascular density
Chaves et al., 2016 [32]	Liver, preclinical	Rat hepatic IRI	HBOT during ischemia, reperfusion, or both	Injury score lower with HBOT during ischemia only (79.0 vs 135.0); higher with HBOT during reperfusion (151.3)
Liu et al., 2011 [29]	Liver, preclinical	Rat hepatic IRI	HBOT preconditioning 2.5 ATA, 90 min	HO-1 upregulation; lower MDA; improved antioxidant capacity
Kurir et al., 2004 [33]	Liver, preclinical	Rat partial hepatectomy	HBOT before or after resection	Faster early recovery of liver mass and lower lipid peroxidation with HBOT pretreatment
Ijichi et al., 2006 [34]	Liver, preservation	Rat liver cold storage	Hyperbaric oxygen during static cold storage	Less preservation injury and ATP depletion
Giannone et al., 2012 [35]	Liver, preservation	Rat liver, 24-h preservation	Hyperbaric (2 ATA) hypothermic machine perfusion	Near-normal ultrastructure, preserved glycogen, less oxidative stress, higher eNOS expression; no transplantation
Castro e Silva et al., 2006 [36]	Liver, clinical	Case series	HBOT for HAT and functional cholestasis	Normalization of transaminases and bilirubin; retransplantation avoided
Grover et al., 2006 [37]	Liver, clinical	Pediatric case report and review	HBOT plus heparin for HAT after failed thrombolysis	Recanalization after 6 sessions; graft functioning at 1 year
Franchello et al., 2010 [38]	Liver, clinical	Case report (n=1)	20 HBOT sessions for late HAT after split-liver transplant	Arterial flow restored, collaterals developed, ischemic areas resolved; no retransplantation at 18 months
Bekker et al., 2009 [39]	Liver, clinical	Systematic review	HAT incidence and outcomes	Early HAT 2.9% in adults and 8.3% in children
Nesovic Ostojic et al., 2021 [26]	Kidney, preclinical	Rat renal IRI	HBOT preconditioning or treatment	Improved creatinine and urea; less oxidative injury; HO-1 upregulation; preserved tubules
Ramalho et al., 2012 [40]	Kidney, preclinical	Rat renal IRI	HBOT preconditioning or treatment	Improved creatinine and urea; less oxidative injury; HO-1 upregulation; preserved tubules
Kanowski et al., 2025 [41]	Kidney, clinical	Controlled study in patients treated for hearing loss or tinnitus (n=23; 15 received HBOT)	10 HBOT sessions	No adverse effect on renal function; no transplant recipients

TABLE 1: Key preclinical and clinical evidence for HBOT, hyperbaric preservation, and machine perfusion in liver and kidney transplantation

ATA: atmospheres absolute; COR: controlled oxygenated rewarming; D-HOPE: dual HOPE; DBD: donation after brain death; DCD: donation after circulatory death; EAD: early allograft dysfunction; eNOS: endothelial nitric oxide synthase; HAT: hepatic artery thrombosis; HO-1: heme oxygenase-1; HOPE: hypothermic oxygenated perfusion; IRI: ischemia-reperfusion injury; MDA: malondialdehyde; NMP: normothermic machine perfusion; NMP-ILT: ischemia-free liver transplantation; RCT: randomized controlled trial.

DCD grafts sustain warm ischemia with amplified endothelial activation and cholangiocyte vulnerability, the substrate of ischemic cholangiopathy [16]. Most HBOT models use controlled ischemia paradigms that do not reproduce DCD injury, a key gap given the growing use of these grafts.

Liver Regeneration and Partial Grafts

Regeneration is central to split and living donor liver transplantation, in which a partial graft must grow rapidly to meet the recipient's metabolic demand. In rats undergoing partial hepatectomy, HBOT pretreatment was associated with faster early recovery of liver mass, lower lipid peroxidation in the remnant liver, and better-preserved lobular architecture, without biochemical signs of added injury [33]. Whether HBOT can support regeneration of partial grafts, or protect against small-for-size injury, has not been studied in transplant models and represents a specific research opportunity.

Clinical Experience: Hepatic Artery Thrombosis (HAT)

Clinical data are limited to case reports and small series, most describing HBOT as salvage therapy for HAT. Early HAT occurs in approximately 3% of adult and 8% of pediatric recipients and carries substantial graft loss and mortality [39,42]. Because the biliary tree depends almost entirely on hepatic arterial inflow, HAT often presents with bile leaks, biliary strictures, or hepatic abscesses, and urgent revascularization or retransplantation remains standard management [39,42]. When revascularization fails or is not feasible, and a donor organ is not immediately available, there is no established therapy to protect the ischemic biliary tree, and this gap is where HBOT has been tried. Reports describe normalization of liver tests, recanalization, and avoidance of retransplantation after HBOT, including after failed revascularization [36,37] (Table *1*). HBOT has also been used for late HAT: in a split-liver recipient with arterial occlusion nearly two years after transplantation, 20 sessions were followed by restoration of intrahepatic arterial flow, development of collaterals, and resolution of ischemic areas, with retransplantation avoided at 18 months [38]. Proposed mechanisms include enhanced oxygen diffusion to the biliary tree, promotion of collateral circulation, and mitigation of biliary ischemia [37,43]. These observations are uncontrolled and subject to publication bias, and HBOT is not part of standard HAT management algorithms [39,42].

Other Clinical Applications

An integrative review identified only six relevant studies of HBOT before and after liver transplantation. It described fewer episodes of encephalopathy and less pruritus in waiting-list patients and suggested protection against early hepatocyte injury after transplantation, but the underlying evidence was of low level [44]. No randomized trial has evaluated recipient HBOT in liver transplantation. Logistical barriers and the low incidence of hard endpoints such as primary non-function have hindered trial design, and translational effort has shifted toward ex vivo graft oxygenation.

Hyperbaric Oxygen Applied to the Graft: Experimental Preservation

The idea of applying hyperbaric oxygen to the graft rather than the recipient is not new. Combined hypothermia and hyperbaric oxygenation was explored for kidney preservation decades ago; one canine study combined cooling to 4°C, 3 ATA of oxygen, and initial plasma perfusion to preserve kidneys for 24-48 hours before reimplantation [45]. Interest declined with the success of simple cold storage solutions but has re-emerged experimentally. In rat livers, hyperbaric oxygen applied during static cold storage reduced preservation injury and ATP depletion [34]. A hyperbaric hypothermic perfusion system at 2 ATA preserved near-normal hepatocyte ultrastructure and glycogen, reduced oxidative stress, and increased endothelial nitric oxide synthase expression over 24 hours, effects attributed specifically to the hyperbaric component [35]. Neither the cold-storage study by Ijichi et al. [34] nor the perfusion study by Giannone et al. [35] included transplantation, and no hyperbaric preservation device has entered clinical testing

Persufflation, the delivery of gaseous oxygen through the graft vasculature, is a related approach studied in the heart, liver, kidney, intestine, and pancreas. Concern about gas embolism after transplantation has limited its adoption, and it remains experimental [46].

HBOT in kidney transplantation

Preclinical Studies

Renal IRI models consistently show renoprotection with HBOT, including improved creatinine and urea, reduced oxidative injury, preserved tubular architecture, and heme oxygenase-1 induction [26,40,47-49] (Table *1*). These findings suggest relevance to extended-criteria and DCD kidneys, which are most susceptible to DGF.

Clinical Evidence

Clinical experience with HBOT in kidney recipients is essentially absent. The absence is notable because DGF, the clinical expression of renal IRI, is common after DCD and extended-criteria transplantation and has no specific preventive therapy once the kidney is implanted [8]. A small controlled study of patients receiving HBOT for otologic indications found no adverse effect on renal function, but it did not include transplant recipients [41].

Timing of oxygen delivery: the central translational problem

Across liver and kidney models, the most consistent determinant of benefit is when oxygen is delivered relative to reperfusion (Figure *1*). Early HBOT reduced neutrophil sequestration and hepatic injury, whereas delayed exposure offered little protection [50]. In a direct comparison of exposure windows, HBOT during ischemia was protective, whereas HBOT during reperfusion worsened hepatic injury [32]. Preconditioning regimens, in which oxygen is given before ischemia, account for most of the positive liver and kidney studies [26,29-31,49]. This pattern is consistent with the underlying mechanism: succinate accumulates during ischemia and drives a burst of mitochondrial ROS within minutes of reperfusion [13], so an intervention must act before or at reperfusion to prevent it.

In clinical transplantation, this window is largely inaccessible to recipient HBOT. Donor preconditioning is rarely feasible; the graft cannot be exposed to hyperbaric oxygen in the recipient before implantation, and transfer to a chamber after surgery takes hours, by which time the reperfusion burst and innate immune activation have already occurred. This mismatch between the protective window in experimental models and the earliest practical point of clinical delivery explains much of the gap between preclinical promise and clinical translation. It is also the main reason graft-directed oxygenation during preservation has advanced while recipient HBOT has not.

Machine perfusion as a comparator: graft-directed oxygenation

Machine perfusion oxygenates the isolated graft during preservation, before reperfusion, and is discussed here as a comparator rather than as a form of HBOT. The two bodies of evidence differ fundamentally in quality. Machine perfusion has been evaluated in multicenter randomized controlled trials and meta-analyses with prespecified clinical endpoints, whereas clinical evidence for recipient HBOT consists of uncontrolled case reports and small series. HBOT findings in this review are therefore presented descriptively (Table *1*), and the effect sizes reported for machine perfusion (Table *2*) should not be read as comparable to, or as a benchmark for, the HBOT observations.

Study (author, year)	Organ	Design and level of evidence	Comparison	Key findings
Tingle et al., 2023 [51]	Liver	Meta-analysis of RCTs	HOPE vs static cold storage	4 trials, 482 recipients: graft loss HR 0.45; ischemic cholangiopathy in DCD OR 0.31; serious adverse events in extended-criteria DBD OR 0.45
Parente et al., 2023 [52]	Liver	Meta-analysis, 7 RCTs (n=1,017)	HOPE and NMP vs static cold storage	EAD reduced with HOPE (RR 0.48) and NMP (RR 0.50); HOPE reduced major complications, retransplantation, and graft loss
Jaber et al., 2025 [53]	Liver	Network meta-analysis, 12 RCTs (n=1,628)	HOPE/D-HOPE, NMP, NMP-ILT, COR vs static cold storage	HOPE/D-HOPE: EAD RR 0.53, graft failure RR 0.38, biliary complications RR 0.52
Nguyen et al., 2025 [54]	Liver	Single-center retrospective cohort (n=1,086)	NMP vs static cold storage	Lower graft failure (HR 0.22) and mortality (HR 0.31); shorter ICU stay
Jochmans et al., 2020 [18]	Kidney	Paired RCT, 106 DCD kidney pairs	Oxygenated vs non-oxygenated hypothermic perfusion	12-month eGFR not different (primary analysis); graft failure 3% vs 10%; acute rejection 14% vs 26%

TABLE 2: Randomized and large-cohort evidence for oxygenated machine perfusion, included as a comparator

These data come from randomized trials and meta-analyses and are not directly comparable with the descriptive HBOT evidence in Table 1.

COR: controlled oxygenated rewarming; D-HOPE: dual hypothermic oxygenated perfusion; DBD: donation after brain death; DCD: donation after circulatory death; EAD: early allograft dysfunction; HOPE: hypothermic oxygenated perfusion; NMP: normothermic machine perfusion; NMP-ILT: ischemia-free liver transplantation; RCT: randomized controlled trial.

A Cochrane meta-analysis found that hypothermic oxygenated perfusion (HOPE) improves graft survival and reduces serious adverse events compared with static cold storage [51]; in DCD livers, it also reduced ischemic cholangiopathy [51,55] (Table *2*). Oxygenation at low temperature is thought to restore mitochondrial energy stores and limit succinate-driven ROS generation before the graft is reperfused

[13,55]. Normothermic Machine Perfusion (NMP) maintains physiologic metabolism and allows viability assessment before transplantation. Meta-analyses and large cohorts show less early allograft dysfunction and improved graft outcomes relative to static storage [52-54] (Table *2*). In the COMPARE trial, 106 pairs of kidneys from DCD donors aged 50 years or older were randomized to oxygenated or non-oxygenated hypothermic machine perfusion. The primary endpoint, 12-month eGFR, did not differ in the primary analysis, but oxygenation reduced graft failure (3% vs 10%) and biopsy-proven acute rejection (14% vs 26%) [18] (Table *2*). Machine perfusion thus delivers oxygen within the protective window, whereas recipient HBOT can begin only after reperfusion (Figure *1*). The two strategies are compared qualitatively in Table *3*.

Feature	Recipient HBOT	Machine perfusion (HOPE, NMP, HMP-O2)
Target	Whole recipient (systemic)	Isolated graft (ex vivo)
Timing relative to reperfusion	After implantation and transfer to chamber	Before reperfusion, during preservation
Main mechanism	Raised dissolved plasma oxygen; antioxidant and anti-inflammatory signaling	Restored mitochondrial energy; reduced succinate-driven ROS; viability assessment (NMP)
Evidence in liver transplantation	Animal models; case reports for HAT	Multiple RCTs and meta-analyses
Evidence in kidney transplantation	Animal models; no clinical efficacy data	RCT (COMPARE) and other trials
Patient-related constraints	Unstable recipients, ventilation, vasopressors, dialysis; limited monitoring in chamber	None for the recipient; donor-graft logistics only
Safety concerns	Barotrauma, CNS and pulmonary oxygen toxicity; possible interaction with calcineurin inhibitor neurotoxicity	Device failure; perfusion-related graft injury (rare)
Infrastructure	Hyperbaric chamber with critical care capability; few transplant centers	Perfusion device and trained staff; increasingly available
Standardization	No agreed pressure, duration, number of sessions, or start time	Established protocols for each device
Current status	Investigational; selective salvage use	Integrated into routine practice at many centers

TABLE 3: Recipient HBOT versus ex vivo machine perfusion as strategies for graft oxygenation

CNS: central nervous system; HAT: hepatic artery thrombosis; HBOT: hyperbaric oxygen therapy; HMP-O2: oxygenated hypothermic machine perfusion; HOPE: hypothermic oxygenated perfusion; NMP: normothermic machine perfusion; RCT: randomized controlled trial; ROS: reactive oxygen species.

HBOT for post-transplant complications beyond IRI

Transplant recipients are exposed to several complications for which HBOT is already an accepted adjunct outside transplantation, and in these settings the evidence base is borrowed from other populations rather than absent. Necrotizing soft-tissue infections and compromised skin grafts or flaps are recognized indications [21], and both occur in immunosuppressed recipients with impaired wound healing.

Invasive mucormycosis is an uncommon but often fatal opportunistic infection in immunosuppressed patients, and standard antifungal therapy has limited efficacy. In a review of 28 published cases treated with adjunctive HBOT, survival was 94% among patients with diabetes but only 33% among those with hematologic malignancy or bone marrow transplantation, and longer HBOT courses were associated with better survival [56]. These data are retrospective and highly selected, but they support considering HBOT as part of multidisciplinary management of selected invasive fungal infections, particularly when tissue necrosis is extensive.

In these indications, HBOT is used for the complication rather than for the graft, so its benefit does not depend on reaching the protective window around reperfusion. This is arguably the most realistic current role for HBOT in transplant programs with access to a chamber.

Practical considerations if HBOT is used in transplant recipients

When HBOT is considered for a transplant recipient, whether for HAT, a compromised wound, or a

necrotizing infection, several practical steps reduce risk. The decision should involve the transplant team, the hyperbaric physician, and critical care, with treatment delivered in a multiplace chamber if the patient requires ventilation or close monitoring [20].

Before the first session, an unvented pneumothorax must be excluded, which is relevant after liver transplantation given the frequency of right-sided pleural effusions and thoracic drains. Pre-treatment glucose should be checked in patients with diabetes, and HBOT should be used cautiously in patients with significantly reduced left ventricular function because of the risk of pulmonary edema. Slow compression reduces middle-ear barotrauma, and intubated or sedated patients may require tympanostomy to equalize pressure [22]. For the wound-related indications most relevant to transplant recipients, such as compromised skin grafts and flaps, recommended regimens of 2.0 to 2.5 ATA for 60 to 120 minutes provide a reasonable reference [21].

During a course of treatment, immunosuppressant levels, neurologic status, and glycemic control should be monitored closely, given the overlapping risks of calcineurin inhibitor neurotoxicity and oxygen toxicity [22,57]. The goals of treatment, the number of planned sessions, and objective response criteria, such as Doppler arterial flow, liver tests, biliary imaging, or wound status, should be defined in advance. Recording these elements consistently, even in single cases, would make future reports more informative than the current literature.

Limitations of clinical application

Transferring critically ill recipients to a hyperbaric chamber is complex, particularly early after surgery. Transport of ICU patients to standalone facilities is feasible but resource-intensive [20], and monoplace chambers limit access for invasive monitoring and resuscitation. HBOT carries recognized risks, including middle-ear barotrauma, central nervous system oxygen toxicity with seizures, and pulmonary oxygen toxicity with repeated sessions [9,22]. Optimal pressure, session number, timing relative to reperfusion, and interactions with immunosuppression remain undefined.

Beyond logistics, several features of transplant practice limit where HBOT can realistically be applied (Table *3*). The timing mismatch has been discussed above; the remaining constraints are practical:

Recipient Condition

The patients most likely to benefit, such as those with early allograft dysfunction, HAT, or DGF, are often the least stable: ventilated, on vasopressors, coagulopathic, or receiving renal replacement therapy. Each of these complicates chamber treatment, and monitoring and resuscitation are restricted during a session.

Drug Interactions

Calcineurin inhibitors cause neurotoxicity in a substantial minority of recipients, and seizures are a recognized complication [57]. Whether this risk is additive with hyperbaric oxygen toxicity has not been studied. Post-transplant diabetes adds a risk of hypoglycemia during treatment [22]. The effect of repeated hyperoxia on immunosuppressant handling and on rejection is also unknown.

Access and Cost

Few transplant centers have an on-site multiplace chamber with critical care capability. Repeated sessions consume staff time and chamber capacity without reimbursement for this indication.

No Standard Protocol or Endpoint

Reported regimens vary in pressure (2-3 ATA), session length, number of sessions, and start time. There is no agreed clinical endpoint against which to judge response, which makes case-level benefit hard to attribute to HBOT rather than to concurrent interventions.

Competition With Established Tools

Machine perfusion now addresses graft oxygenation before reperfusion with randomized evidence, and revascularization or retransplantation remain the standard for HAT. HBOT must therefore show added value on top of these, a higher bar than benefit over no treatment.

Future directions

Progress requires studies designed around the biology described above rather than further uncontrolled case reports. Table *4* summarizes priorities.

Question	Suggested study design	Key endpoints
Does adjunctive HBOT improve graft salvage in HAT?	Prospective multicenter registry or pragmatic trial alongside standard revascularization	Graft survival free of retransplantation; biliary complications; safety events
Can hyperbaric oxygenation enhance ex vivo perfusion?	Preclinical and early-phase studies of hyperbaric HOPE or NMP	Mitochondrial function, ATP content, biliary injury markers, post-transplant function
Does HBOT protect DCD grafts and the biliary tree?	DCD-specific large-animal transplant models	Ischemic cholangiopathy, bile duct histology, graft survival
Does HBOT support regeneration of partial grafts?	Split and living-donor transplant models, including small-for-size settings	Graft volume gain, hepatocyte proliferation, liver function
Can HBOT reduce DGF in high-risk kidneys?	Feasibility study in recipients of DCD or extended-criteria kidneys	DGF incidence, dialysis-free days, eGFR at 12 months
Is HBOT safe with modern immunosuppression?	Prospective safety cohort with standardized monitoring	Seizures, calcineurin inhibitor levels, glycemic events, rejection

TABLE 4: Research priorities for hyperbaric oxygen in liver and kidney transplantation

DCD: donation after circulatory death; DGF: delayed graft function; eGFR: estimated glomerular filtration rate; HAT: hepatic artery thrombosis; HBOT: hyperbaric oxygen therapy; HOPE: hypothermic oxygenated perfusion; NMP: normothermic machine perfusion.

Prospective registries or pragmatic trials of HBOT as an adjunct to revascularization in HAT, the setting with the most clinical experience, could define safety, timing, and patient selection with standardized endpoints such as graft survival free of retransplantation and biliary complications. Hyperbaric oxygenation could be integrated into ex vivo perfusion, the one setting in which oxygen can be delivered before reperfusion; preclinical hyperbaric perfusion data provide a starting point [35]. DCD-specific and partial-graft models are needed to test whether HBOT protects the biliary tree and supports regeneration under clinically relevant injury. Finally, any recipient-level trial should prespecify immunosuppressant levels, neurologic events, and glycemic control as safety outcomes, given the overlap between HBOT toxicity and transplant pharmacology.

Mechanistic biomarkers, including succinate, mitochondrial injury markers, and perfusate or serum markers of endothelial and biliary injury, could link HBOT exposure to biologic effect and help select patients. Collaboration between transplant centers and hyperbaric units, many of which already manage critically ill patients, will be necessary for adequately powered studies.

Strengths and limitations of this review

This review has several distinctive features. It examines liver and kidney transplantation together, exposing a shared pattern of strong preclinical signal and minimal clinical translation. It frames recipient HBOT against ex vivo preservation, including hyperbaric and oxygenated machine perfusion, as routes to the same goal, graft oxygenation, distinguished by where and when oxygen is delivered. It quantifies the dissolved-oxygen contribution across hemoglobin levels, making the physiologic argument concrete for clinicians. It identifies the timing of exposure relative to reperfusion as the unifying explanation for the gap between animal and human data, highlights the absence of DCD-specific and partial graft models as key research gaps, and separates graft-directed uses of HBOT from established adjunctive uses for post-transplant complications. Finally, it offers practical guidance for clinicians and proposes realistic niches and a research agenda rather than routine use.

The review also has limitations. Although the search was structured and eligibility criteria were predefined, this is a narrative rather than a systematic review; no protocol was registered, and no formal risk-of-bias or certainty assessment was performed, so selection bias cannot be fully excluded. A meta-analysis of HBOT data was not feasible. Preclinical studies differed in species and strain, ischemia model (in situ clamping or transplantation, partial or total, warm or cold), ischemia duration, HBOT pressure (2-3 ATA), session length and number, timing relative to ischemia and reperfusion, and outcome measures, and clinical data consisted of single cases or small series without comparators. Pooled estimates from such data would have had no meaningful interpretation, so the evidence was synthesized qualitatively. Most of the HBOT evidence comes from rodent models, which limits extrapolation to humans. Clinical HBOT reports are prone to publication bias, and clinical data in kidney recipients are essentially absent. Evidence for HBOT in post-transplant infections is extrapolated from mixed populations. Comparisons between HBOT and machine perfusion are indirect, because no study has tested them head-to-head or in combination. Finally, machine perfusion is evolving quickly, and newer trials may change the comparative picture.

Conclusions

HBOT is biologically plausible as an adjunct against IRI, acting through hemoglobin-independent oxygen delivery, inflammatory modulation, and mitochondrial preservation. Preclinical liver and kidney models consistently show protection, but benefit depends on timing and is greatest during ischemia or as preconditioning, a window that recipient HBOT rarely reaches. Clinical evidence is confined to case reports, mainly of salvage therapy for hepatic artery thrombosis, and no randomized trials exist. Practical barriers, including recipient instability, chamber access, possible interactions with immunosuppression, and the absence of standardized protocols, further limit its use. Ex vivo oxygenation by machine perfusion, by contrast, has randomized evidence of benefit and is now central to graft preservation, and hyperbaric oxygenation of the isolated graft is a clinically untested extension of it. In the near term, HBOT is best regarded as a selective salvage therapy for arterial complications and an adjunct for established indications such as necrotizing infections, while prospective studies define whether it has a place in protecting the graft itself.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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