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Hyperbaric Oxygen Therapy: A Quick Guide for Physicians and Surgeons

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Abstract

Hyperbaric oxygen therapy (HBO2) is a useful, and often underutilized, treatment modality for a variety of conditions. Providing 100% oxygen at increased atmospheric pressures oxygenates ischemic tissues, decreases edema, lessens reperfusion injury, stimulates angiogenesis, promotes wound healing, and improves fibrosis of irradiated tissues. As a result, HBO2 may significantly improve patient outcomes in carbon monoxide poisoning, crush injury and impending compartment syndrome, bone and soft tissue necrosis secondary to delayed radiation injury, problem wounds, central retinal artery occlusion, and idiopathic sudden sensorineural hearing loss. Given the likelihood of initial patient presentation or necessity of expedient intervention, it is imperative for physicians and surgeons to be able to recognize such opportunities where HBO2 referral is appropriate. Timely referral is important for successful outcomes.

Key Points

Doctors and other providers should be aware of the utility of hyperbaric oxygen therapy for the following conditions:

1. Carbon Monoxide Poisoning
2. Crush Injury and Skeletal Muscle Compartment Syndrome
3. Delayed effects of therapeutic radiation including osteoradionecrosis, radiation cystitis, enteritis, proctitis and skin wounds in irradiated fields.
4. Central Retinal Artery Occlusion
5. Sudden Sensorineural Hearing Loss
6. Problem wounds due to diabetes and vascular disease

Introduction

Hyperbaric oxygen therapy (HBO2) as defined by the Undersea & Hyperbaric Medical Society (UHMS), is "an intervention in which an individual breathes near 100% oxygen intermittently while inside a hyperbaric chamber that is pressurized to greater than sea level pressure (1 atmosphere absolute, or ATA)" [1] Patients are enclosed within a chamber that is pressurized up to three times normal atmospheric pressure. HBO2 is commonly known by health care providers for treating decompression sickness ("the bends") in divers; however, there are a total of fourteen indications approved by the Undersea & Hyperbaric Medical Society (UHMS) and generally reimbursed by The Centers for Medicare & Medicaid Services (CMS) and other insurers in the USA.

These indications are:

1. Gas Embolism
2. Carbon Monoxide Poisoning
3. Clostridial Myositis and Myonecrosis (Gas Gangrene)
4. Crush Injury, Compartment Syndrome and Other Acute Traumatic Ischemia's
5. Decompression Sickness
6. Arterial Insufficiencies including:
    a. Problem Wounds
    b. Central Retinal Artery Occlusion
7. Severe Anemia
8. Intracranial Abscess
9. Necrotizing Soft Tissue Infections
10. Osteomyelitis (Refractory)
11. Delayed Radiation Injury (Soft Tissue and Bony Necrosis)
12. Compromised Grafts and Flaps
13. Acute Thermal Injury
14. Idiopathic Sudden Sensorineural Hearing Loss

Physicians and surgeons encounter a broad spectrum of patients that may need referral for hyperbaric therapy, either as the primary treatment modality or as an adjunct to pharmacologic or surgical interventions. Out of the fourteen indications for HBO2, we have decided to focus on the following based on the likelihood of initial patient presentation or necessity of expedient intervention.

Additional information and resources may be found at the Undersea & Hyperbaric Medical Society: https://www.uhms.org/

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Carbon Monoxide Poisoning

Carbon Monoxide (CO) is a colorless, odorless gas that can be inhaled when an individual is exposed to combustion products in a poorly ventilated or enclosed area. CO poisoning is a clinical diagnosis, based upon the patient's history and reported symptoms. Carboxyhemoglobin (COHb) may be used to confirm exposure, but the relatively short half-life of COHb may result in normal COHb levels [2-4].

The cardiovascular and central nervous systems are the most susceptible following CO exposure, resulting in cardiac injury and often long-lasting neurological sequelae including motor weakness, peripheral neuropathies, hearing and vision changes, and Parkinsonian-like syndromes [5-7]. Infants, children, pregnant women, elderly, and those with heart disease are particularly at risk for more serious illness.

CO induced injuries are due to hypoxic stress from COHb formation as well as systemic oxidative stress [8]. However, it is important to keep in mind that COHb levels are not correlated with symptomatology or very predictive of the development of central nervous system sequelae and long-term patient outcome [9-12]. Therefore, COHb should not be used alone to determine necessity of hyperbaric referral.

Signs and symptoms may present long-after the initial exposure. Patients may complain of nonspecific symptoms, such as: headaches, dizziness, fatigue, and sleep disturbances. In addition, neurological symptoms including neuropsychological and affective symptoms may arise. This symptomatology can present weeks to months later and patients may have deficits lasting for years. Therefore, patient follow-up is crucial [13].

Patients with signs or symptoms of CO poisoning should be placed on supplemental oxygen to increase tissue oxygenation and hasten the dissociation of CO from hemoglobin [3]. The addition of hyperbaric oxygen accelerates this dissociation, treats tissue hypoxia, reduces the harmful inflammatory response that can occur in damaged tissue, and protects against oxidative stress [14,15]. Despite various findings in differing studies and reviews such as the Cochrane review, which discusses a trial that has been under scrutiny for using inadequate dosing of HBO2, HBO2 has been shown to decrease the incidence of cognitive and cerebrovascular abnormalities and improve long term neurological outcomes [16-19]. This treatment is recommended by the UHMS and is rated as a Class I – Strong recommendation (American Heart Association Classification) [13].

HBO2 consultation should be considered in patients with signs of significant CO poisoning (e.g. severe acidosis, cardiovascular dysfunction or injury, loss of consciousness, neurological problems, or COHb ≥ 25%), with the optimal benefit occurring with the least delay [20-22]. The most optimal time to HBO2, to prevent delayed neurologic sequence, is within 48 hours of exposure [23].

Crush Injury and Skeletal Muscle Compartment Syndrome

For both crush injuries and skeletal muscle compartment syndrome, trauma and subsequent tissue hypoxia are involved in a vicious cycle that ultimately lead to limb threatening damage. Following the initial insult, bleeding or edema within the compartment can collapse the microcirculation leading to tissue ischemia and hypoxia. Hypoxic cells leak intracellular water leading to further edema and third spacing of fluid. This ischemia-edema cycle continues until compartment syndrome is fully established and emergent fasciotomy is needed.

If delivered early, HBO2 may benefit the patient through several mechanisms. First, HBO2 offsets tissue hypoxia by increasing oxygen tensions in plasma as well as tissue fluids. This increases the diffusion distance of oxygen from the capillary to the cell [24,25]. Secondly, HBO2 reduces edema by inducing vasoconstriction, reducing capillary inflow and decreasing hydrostatic pressure in the capillary bed. It does this while maintaining fluid outflow with resorption of fluid at the capillary level further reducing fluid built up within the compartment [26-31]. Lastly, HBO2 can mitigate oxidative reperfusion injury by interfering with neutrophil adhesion to the endothelium and providing an oxygenated environment to produce oxygen radical scavengers that are responsible for reducing reactive oxygen species [32,33].

The best argument can be made for patients in the impending compartment syndrome stage. When the patient begins to develop signs and symptoms associated with compartment syndrome (e.g. pain, hyperesthesia, weakness, discomfort with passive stretching of toes, or tautness of the compartment) compartment pressure measurements should be made by the provider caring for the patient in any location (e.g. covering in an Emergency Department in a rural facility). In the impending stage, the patient has not reached the threshold requiring fasciotomy. Consideration and consultation for HBO2 should be made at this time, given the opportunity to intervene in the edema-ischemia cycle and potentially prevent the compartment syndrome advancing to requiring fasciotomy [34,35].

Additionally, the cost effectiveness of HBO2 is evident. It has been reported that when HBO2 was started during the impending stage of compartment syndrome, the total costs were 75 percent less than having to complete HBO2 following a fasciotomy procedure [36]. It is estimated that the cost savings for one patient to undergo a fasciotomy in the impending stage would be equivocal to ten patients undergoing HBO2 in the impending stage and halting their progression [13]. In addition, following crush injury, the same mechanisms may reduce healthcare costs through decreasing complications, reducing tissue loss, and morbidity. This can then improve patient outcomes, mental outlook, and ability to function [13,36].

Delayed Radiation Injury – Soft Tissue and Bony Necrosis

Radiation therapy is associated with several acute, subacute, and delayed complications following treatment. Many of the acute and subacute complications are self-limiting in nature or are treated symptomatically [37]. The delayed complications of radiation therapy may develop months or even years following radiation exposure. These delayed injuries may be precipitated by an additional insult such as surgery within the irradiated area [38]. These injuries, especially those that manifest months to years later with bony or soft tissue necrosis, require multi-disciplinary management in which medical providers play a crucial role in identifying patients at risk.

The mechanism of delayed radiation injury is not well understood at this time. However, it manifests itself as vascular obliteration and stromal fibrosis in the irradiated field [39]. HBO2's ability to combat these changes following radiation is multifactorial. Mechanisms include stimulating angiogenesis, recruiting stem cells, improving oxygenation, and reducing fibrosis of irradiated tissues [40-42].

The largest and best studied application for HBO2 in radiation injury is in the treatment and prevention of osteoradionecrosis of the mandible. In osteoradionecrosis of the mandible, tooth extraction is a common precipitating factor and can necessitate subsequent mandibular resection and reconstruction. Pre- and post-surgical HBO2 has been shown to drastically decrease the occurrence and severity of mandibular necrosis and is an opportunity for providers to recommend a consult for HBO2 [43-45]. Recently this practice has

come into question in the HOPON Trial, however, the study displayed low incidence and was performed on patients receiving lower doses of radiation than in previous literature [46]. Additionally, HBO2 has been considered and utilized for radiation injury at other soft tissue sites, including injuries to the head and neck, radiation cystitis, and radiation proctitis [47]. Early utilization of HBO2 for delayed radiation effects is important for best outcomes.

Given that the sequelae of radiation injury manifest themselves months to years following exposure, practitioners should be cognizant of the advantages of hyperbaric oxygen in the treatment and prevention of complications secondary to radiation therapy.

Enhancement of Healing in Selected Problem Wounds-Arterial Insufficiencies

Non-healing, problem wounds (e.g., arterial insufficiency ulcers, pressure ulcers, and venous stasis ulcers) are an ever-growing challenge for healthcare providers. The chronicity, recurrence rates, and subsequent complications from these wounds represent an increased cost and burden to healthcare systems [48]. The addition to HBO2 to standard wound-care management optimizes the environment for improved healing by reducing the microcirculation impairment and optimizing the local inflammatory response.

The positive healing effect has been best studied and widely implemented in the treatment of infected, ischemic diabetic foot ulcers. Hyperbaric oxygen can mitigate the hypoperfusion, hypoxia, and prevalence of infection that is common in these non-healing wounds [49]. It does this by increasing the partial pressure of oxygen dissolved in the plasma, which subsequently increases the diffusion distance of oxygen at the tissue level. This increase in available oxygen reduces tissue hypoxia and provides an oxygen-rich environment optimal for neutrophils, fibroblasts, and macrophages to carry out necessary repair or immune functions [50-55]. In addition, hyperbaric oxygen has been shown to stimulate angiogenesis and promote tissue growth [56-61].

This practice has been studied for more than 50 years and has continued to be evaluated. Despite varying outcomes in wound healing, promising results have continued to prevail and HBO2 has shown to decrease amputation [62-64]. Therefore, patients with problematic hypoxic lower-extremity wounds (i.e. wound PO2 < 40 mmHg) such as diabetic foot ulcers, hyperbaric oxygen is a valid adjunctive therapy and referral to HBO2 should be strongly considered.

Central Retinal Artery Occlusion

Central retinal artery occlusion (CRAO) is an emergent condition resulting in sudden, painless vision loss that is associated with an overall poor prognosis.

In patients who present with sudden painless vision loss, evaluation of visual acuity along with fundoscopic exam should be performed and documented. In addition, an ophthalmologist should be consulted emergently. Additional diagnostic work-up is necessary to screen and identify predisposing conditions that may help guide further decision making. However, prompt supplementation of oxygen to ischemic retina is of the utmost importance and should not be delayed while awaiting the arrival of consultations or further diagnostic testing [13,65].

If oxygen supplementation at normal atmospheric pressure is ineffective at restoring vision, HBO2 consultation and hyperoxygenation via HBO2 should be initiated. The timing of reoxygenation is essential in CRAO, with improved outcomes in patients who receive proper treatment within 90 minutes of symptom onset. Although, good outcomes have been reported as late as 24 hours after vision loss [66-71]. However, even with optimal treatment, the patient outcome is largely dependent upon the severity of the CRAO, the vessel occluded, the degree and location of the occlusion, as well as the underlying etiology of the occlusion [72,73]. Overall, recent publications have displayed improvement in visual acuity with timely HBO2 onset [74,75].

Idiopathic Sudden Sensorineural Hearing Loss

Idiopathic sudden sensorineural hearing loss (ISSHL) is defined as a loss of ≥ 30 dB occurring within three days over at least three contiguous frequencies [76]. This may present as a patient who complains of sudden unilateral hearing loss, tinnitus, aural fullness, and vertigo [77,78].

The etiology and pathophysiology of ISSHL remains unclear; however, it is now known that perilymphatic oxygen tension is significantly decreased in patients who present with ISSHL. This results in decreased oxygen delivery to the cochlea and associated structures (in particular, the stria vascularis and the organ of Corti) [79,80]. The need for improved oxygen delivery is the primary rational for utilizing HBO2 in treating ISSHL. HBO2 greatly increases arterial perilymphatic oxygen concentration, increasing oxygen delivery to the cochlea and associated structures [80-82]. In addition, there are other potential benefits of HBO2 including blunting of ischemia-reperfusion injury, edema reduction, and anti-inflammatory effects.

Patients who present with sudden sensorineural hearing loss should be evaluated by an otolaryngologist and audiologist in a timely manner. Those determined to have ISSHL may benefit from the addition of an HBO2 consultation and HBO2 as adjunctive therapy, with the best outcomes within two weeks of symptom onset and initiated as soon as possible [77].

Author disclosure of interest

The authors report no conflict of interest. Conflicts may include, but are not limited to, personal, professional, or financial relationships with manufacturers of products mentioned in the manuscript or manufacturers of competing products. No such potential, perceived, or real conflicts of interest exist for either author.

Author Independence

Authors have full access to and control of this paper. The decision to submit this manuscript for publication rests with the authors and no others. No organizations provided support for this effort.

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