Hyperbaric Management Of Frostbite

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Continuing Education Activity

Although evidence for treating frostbite with hyperbaric oxygen is sparse and limited to case reports and animal studies, the proposed mechanism of action is plausible. This activity reviews the presentation and evaluation of frostbite injury and the potential for effective treatment with hyperbaric oxygen therapy. This activity highlights the role of the interprofessional team in the care of affected patients.

Objectives:

- Review the indications for the use of hyperbaric oxygen therapy in the management of frostbite injury.
- Outline the contraindications to hyperbaric oxygen therapy in the treatment of frostbite injury.
- Describe the management of frostbite injury.
- Explain interprofessional team strategies for improving care coordination and communication to advance the treatment of frostbite injury and optimize patient outcomes.

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Introduction

Although evidence for treating frostbite with hyperbaric oxygen is sparse and limited to case reports and inadequate animal studies, there is a plausible mechanism of action to suggest effective treatment using hyperbaric oxygen, and it may have additional benefits if initiated as soon as possible after rewarming. All animal studies reviewed utilized some form of rapid freezing

of tissues involving deeper, and almost immediate, tissue destruction[1][2][3]. This presents a model that is unlike the slow progressive freezing seen in clinical human frostbite cases.

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Anatomy and Physiology

The primary mechanism of injury to human tissue in frostbite typically involves a prolonged, progressive insult to peripheral tissues. As the body cools, the first adaptive response is to maintain thermal homeostasis through vasoregulatory mechanisms to essentially shunt warmer fluid circulation to the body's critical internal organs, referred to as the "core," while restricting the cooled peripheral flow from reaching the core. Maximal peripheral vasoconstriction occurs at skin surface temperatures down to 15 C (59 F), reducing cutaneous flow from about 200 ml/min to 250 ml/min to 20 ml/min to 50ml/min. At temperatures lower than this, vasoconstriction must be interrupted to preserve metabolic function in the soft tissues. This occurs through a physiologic mechanism referred to as the hunting response, also known as the Lewis reaction, where a reflex vasodilation occurs in rhythmic bursts about three to five times per hour and lasts for about 5 to 10 minutes. Below 10 C (50 F), neuropraxia of the sensory nerves occurs, resulting in loss of cutaneous sensation (numbness). With dropping temperatures, blood viscosity increases causing a "sludging" of erythrocytes and thrombus formation. This leads to endothelial inflammation and the resulting cascade of chemotactic factors, including increased release of prostaglandins (PGF), thromboxane (TXA), cellular adhesion molecules (CAMs), matrix metalloproteinases (MMPs), and reactive oxygen species (ROS). Leukocyte adhesion is observed, indicating the initiation of endothelial inflammatory response and eventually the metabolites of arachidonic acid which are implicated as mediators of progressive dermal ischemia. Further congestion and stasis lead to circulatory collapse and endothelial plasma leakage with downrange tissue ischemia.[4]

Up to this point, these tissue changes are considered reversible; however, as cooling progresses below 0 C (32 F), negligible cutaneous flow occurs. Without circulation, skin temperature drops at a precipitous rate (more than 0.5 C per minute) and dermal tissues freeze. As would be expected, smaller blood vessels freeze before larger vessels and venous circulation freezes

before arterial. Further cooling initially leads to ice crystal formation in the extracellular fluid, then in the intracellular fluid, creating an osmotic fluid shift to the extracellular space. This leads to eventual cell death, which occurs from a combination of metabolic disinhibition from tissue hypoxia, compounded by cellular dehydration from the fluid shift, and likely further compounded by physical tissue destruction from possible abrasive mechanical forces exerted on the crystal-laden tissues.

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Indications

Hyperbaric oxygen therapy may be a therapeutic option either as a solo treatment or in combination with vasodilating, anticoagulation, or hemorrheologic agents such as pentoxifylline for the treatment of frostbite. The evidence is limited to case studies.[5][6][7][8][9][10][11][12][13][14]

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Contraindications

Hyperbaric oxygen therapy (HBO2) is relatively safe with the most common side effect by far related to barotrauma of the ears or sinuses. The only absolute contraindication is an untreated pneumothorax. Caution must be used in patients with respiratory infections, seasonal allergy symptoms, sinusitis, or recent surgeries or emphysema/chronic obstructive pulmonary disease with open gas pockets or blebs. HBO2 should not be administered in conjunction with doxorubicin, Sulfamylon, or disulfiram and for an extended time following discontinuation of either bleomycin or cisplatinum. Patients with asthma should be evaluated for air trapping but can be successfully treated with education and training. Patients with confinement anxiety may need sedation. Patients with implantable devices may need the device to be cleared by the manufacturer for effective delivery under pressure.

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Equipment

Use approved multiple or monoplane chambers with appropriately trained staff. The Undersea and Hyperbaric Medical Society (UHMS) provides

accreditation for facilities and staff to improve confidence in adherence to standards of safety and appropriate use.

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Personnel

All HBO2 treatments must be directly supervised by a physician trained in hyperbaric medicine. Physicians that have the minimum required 40-hour introductory course training should be precepted by physician Board Certified in UHM or have a certificate of additional training (CAQ) in UHM. Support personnel includes nursing staff, preferably a certified hyperbaric nurse (CHRN), and certified hyperbaric technicians (CHTs). For a multiple chamber treatment, one accompanies the patient as an inside attendant with one chamber "driver" and one outside observer. For monoplane chambers, one CHT can monitor two monoplane chambers.

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Preparation

Patients must be dressed in clothing that has a low potential for static discharge. Cotton/polyester fabric blends up to 60/40 are acceptable. To further reduce fire risk patients are screened for no oil or petroleum products in hair or skin, makeup, nail polish, or deodorants. All electronic devices, telephones, watches hearing aids, etc. are left out of the chamber. No chemical or gas heating devices are allowed in a chamber. Transdermal patches should be avoided or, at least, checked and covered. Diabetics should have glucose checks before and after treatment, following approved guidelines for treatment according to levels.

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Technique

There is no specific treatment protocol identified but a used regimen according to submitted case studies is 2.0 to 2.5 ATA for 90 to 110 minutes, with or without an air break, twice a day for the first eight to 12 treatments and continued daily until the maximum medical benefit is determined. This typically occurs within 12 to 20 treatments.

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Complications

The most commonly reported complications are related to barotrauma of the ears and/or more rarely the sinuses. There is a concern for oxygen toxicity which is reported very rarely, and no cases of recurrence or resulting sequelae have been reported. Another rare possible complication is barotrauma to parenchymal lung tissue from blebs or emphysematous bullae resulting in pneumothorax, but this also is a very rarely reported complication. One review of complications in 782 patients involving 11,376 HBO2 treatments showed 17% had experienced some ear pain or discomfort, but the barotraumatic injury was verified in only 3.8%of all patients. No barotrauma or teeth or sinuses were identified. Four patients experienced oxygen seizures, but none of these had recurrences or sequelae. Other known complications include a possible worsening of existing cataracts and a temporary change in refractory vision usually a self-limited increase in myopia resolving over several weeks post-treatment. Patients with confinement anxiety (claustrophobia) may require anxiolytic therapy.

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Clinical Significance

The pathophysiologic evidence indicates the primary mechanism of tissue damage in slow onset frostbite is due to a multifactorial vascular response. The final stage of intracellular ice crystal formation may seal the necrotic fate of the tissue, at that point preventing a chance for tissue regeneration. There is a recoverable phase if rewarming occurs before the final tissue destruction, but endothelial reperfusion injury can occur again within 4 to 8 hours post-rewarming. All treatment options are focused at restoration and maintenance of microvascular flow post-rewarming and reduction of inflammation, specifically aimed at endovascular inflammatory effects and prevention of reperfusion injury. Treatment options are then divided into three phases: (1) prethaw field care, (2) immediate rewarming phase, which is preferable in the hospital, and (3) postthaw phase, which may continue for several weeks to months.[15]

In the majority of cases, however, the postthaw phase treatment is delayed until the physical evidence of tissue damage is manifest when the wounds are staged and assessed, typically more than 24 to 48 hours post-rewarming. This is due to a lack of early recognition protocols and inability to identify true frostbite in the earliest stages.[16][17][16] This diagnostic dilemma sometimes leads to further loss of tissues that are hovering between stages of reversible to irreversible damage, either from effects of reperfusion injury or further vascular insult due to due persistent or progressive effects of inflammation.[18][19][18] In tissue damage by direct high thermal injury (burns), there is a penumbral area surrounding the immediately damaged tissue (central zone of coagulation) known as the zone of stasis where tissues are viable but prone to injury through vasoconstriction, vascular thrombosis, and endothelial fibrin deposition. Since observed tissue damage in frostbite is similar and the mechanism of destruction is related, low thermal injury, the treatment goal of reducing further penumbral tissue loss in phase 3, is time sensitive and prevention of further loss dependent on early intervention. Cauchy advocates early diagnosis and intervention through a new classification system used within the first 24 hours based on the topography of the presenting lesions, augmented by isotopic bone scans (99mTc-HMDP).[20]

Current treatment options are plagued by lack of high-level evidence provided by well-designed randomized control trials (RCTs), but all therapeutic strategies are aimed at inhibition of endovascular inflammation and early reperfusion. Vasodilators such as nitroglycerine or papaverine have been useful, but studies are not graded with use alone but when combined with thrombolytic agents such as tissue plasminogen activator (rTPA) or streptokinase in a stepwise approach it receives a 1B-C recommendation by ACCP classification criteria. Iloprost, a prostacyclin analogue with vasodilatory properties, also has received a 1B recommendation, but currently, the drug is not available in the United States.

Hyperbaric oxygen sustains the metabolic needs for injured tissue with the immediate reversal of hypoxia and reduces the endothelial inflammation and inhibition of reperfusion injury by reducing leukocyte adhesion. No randomized control trials have been performed to assess this theory, and it has not received an ACCP recommendation, but multiple case series have been presented with hyperbaric oxygen treatment initiated during various

time intervals following rewarming. All conclude that some level of tissue recovery occurred with decreased levels of amputation, but these results have not been matched to controls. Cases that initiate hyperbaric oxygen treatment earlier tend to have better results than delayed treatment, most likely from sustaining penumbral areas of injury. Several studies have combined hyperbaric treatment with vasodilating, anticoagulation or hemorrheologic agents such as pentoxifylline with reported success.[21]

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Enhancing Healthcare Team Outcomes

Adjuvant therapy with hyperbaric oxygen for the treatment of frostbite is reasonable, well tolerated and safe but the evidence supporting treatment is limited to level 4 evidence at best, primarily from case series with no better than historical outcomes to match. Nurses and clinicians involved in wound care should be aware of hyperbaric oxygen as a therapeutic option.

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Review Questions

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