Injury Extra (2005) **36**, 499—502

ELSEVIER

www.elsevier.com/locate/inext

CASE REPORT

The treatment of deep frostbite with hyperbaric oxygen

Hakan Ay [a], Senol Yildiz [a], Gunalp Uzun [a,*], Emrullah Solmazgul [b], Kadir Dundar [c], İsmail Yildirim [d]

[a] *Gülhane Military Medical Academy, Haydarpaşa Training Hospital, Department of Underwater and Hyperbaric Medicine, 81100 Kadıköy/İstanbul, Turkey*
[b] *Gülhane Military Medical Academy, Haydarpaşa Training Hospital, Department of Internal Medicine, 81100 Kadıköy/İstanbul, Turkey*
[c] *Gülhane Military Medical Academy, Department of Underwater and Hyperbaric Medicine, 06018 Etlik/Ankara, Turkey*
[d] *Gülhane Military Medical Academy, Department of Emergency Medicine, 06018 Etlik/Ankara, Turkey*

Accepted 25 April 2005

Introduction

Frostbite is the acute freezing of the skin and tissues and usually develops following exposure to temperatures below −2 °C for more than 1 h without proper clothing.[2,8] The severity of frostbite is related to vascular insufficiency, altitude, duration of exposure, tissue wetness, previous exposure to cold injury, age and other factors.[9] Thromboxanes and prostaglandins also play an important role in frostbite injuries.[5]

Frostbite is classified in four degrees, with superficial frostbite comprising the 1st and 2nd degrees and deep frostbite the 3rd and 4th degrees.[11] Superficial frostbite affects the skin and subcutaneous tissues; deep frostbite also affects bones, joints and tendons.[1] After rewarming, marked tissue oedema becomes evident after 3 h and lasts 5 days or more.[5] Necrotic skin forms black and dry eschar within 9—15 days.[5] Prolonged cold exposure, refreezing of partially thawed tissue and slow rewarming predispose to greater tissue loss.[1]

Effective treatment of frostbite has not yet been exactly defined.[5] Adjunctive therapies including antiprostaglandin agents, vasodilators, thrombolysis, sympathectomy and improved rewarming techniques are all used in the early phases of frostbite.[11] Radiological assessment may be used to determine tissue viability when amputation is required.[11]

Hyperbaric oxygen (HBO_2) is a form of therapy that has been used worldwide for the treatment of many disorders such as problem wounds. It is performed by means of the patient breathing 100% oxygen using an endotracheal tube, mask or hood in a completely isolated compression chamber by raising the ambient pressure to higher than 1 absolute atmosphere (ATA).[4]

Two patients suffering from deep frostbite were admitted to our department on the 14th and 6th

* Corresponding author. Tel.: +90 216 504 2738; fax: +90 216 412 5918.
E-mail address: gunalpu@yahoo.com (G. Uzun).

1572-3461
doi:10.1016/j.injury.2005.04.015

days post injury. We used HBO_2 as an adjunctive therapy in deep frostbite treatment.

Patients

Both patients were treated in a multiplace chamber (Galeazzi, Italy). A medical attendant was present with patients during HBO_2 therapy. Both the patients were treated by HBO_2 (2.4 ATA, total time at depth 60 min, breathing 100% O_2 using a mask), 6 days a week, on an in-patient basis. Wound care was performed daily. All wounds were treated locally with 2% nitrofurazone ointment (Furacin Cream) and vaseline gauze. Tetanus prophylaxis, analgesics and antibiotic prophylaxis were applied in all cases. Daily debridement was performed when required. No adverse effects of HBO_2 treatment were observed.

Case 1

A 50-year-old man in good general health suffered frostbite in December 2002 while standing in an outdoor temperature of −25 °C. Intense pain and cyanotic discoloration on the 1st, 2nd and 3rd toes of both feet occurred with thawing near an oven. On the 2nd day necrosis of the affected toes developed. The patient had been smoking 20—25 cigarettes per day for 35 years. One physician at the regional hospital recommended amputation of all the affected toes on the 5th day, but another physician at the regional hospital recommended HBO_2 therapy. The patient was admitted to our HBO_2 therapy center with erythema, oedema, and cold toes and feet on the 14th day post injury (Fig. 1). We determined there was 4th degree (deep) frostbite on the 1st, 2nd and 3rd toes of the patient's both feet. Thirty-four sessions of HBO_2 treatments were carried out over 40 days. Demarcation occurred on the 26th day of frostbite. An average of 12 days after the HBO_2 therapy the dark crust came off. Granulation tissue appeared. There was only slight tissue loss on the tips of the 1st and 2nd right toes and the tip of the 1st left toe (Fig. 2). All the affected fingers regained their sensitivity. Twelve months after the injury the patient reported no complaints at follow-up examination.

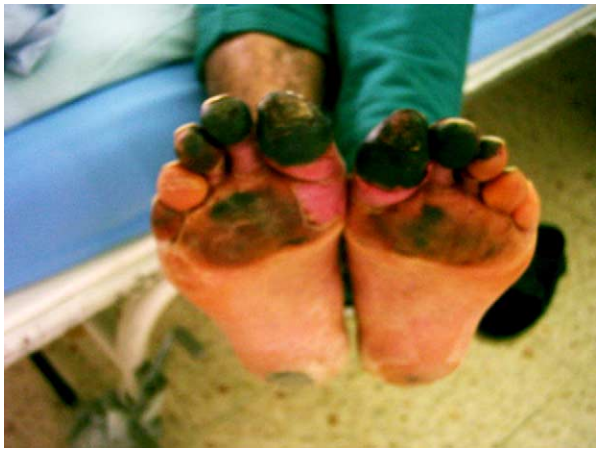

Figure 1 Appearance of the feet at presentation to our department 14 days after frostbite, revealing deep frostbite.

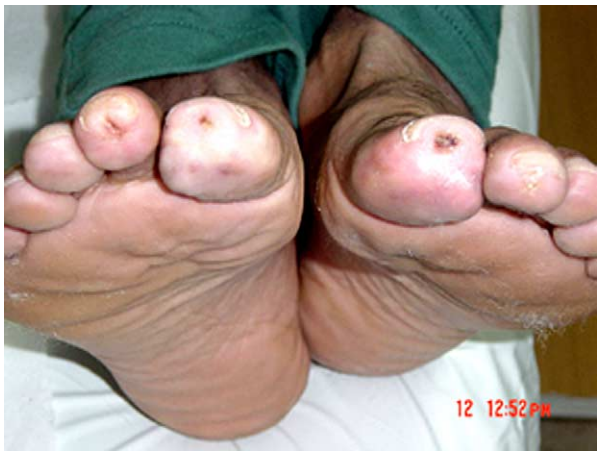

Figure 2 Appearance after 34 sessions of HBO treatment. No amputation required.

Case 2

A 45-year-old man in good general health suffered frostbite in October 2003 while mountain climbing in Turkey in an outdoor temperature of −28 °C. The patient complained of intense pain and discoloration of the feet after thawing out under a blanket.

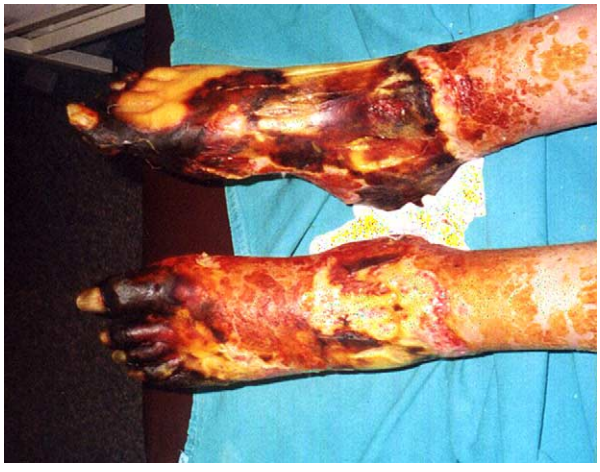

Figure 3 Appearance of the feet at presentation to our department 6 days after frostbite, revealing deep frostbite.

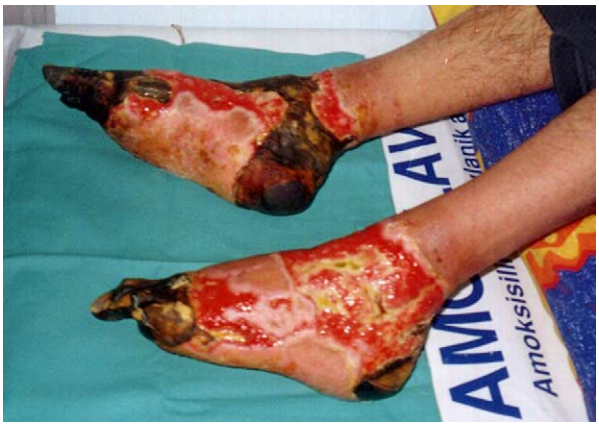

Figure 4 Appearance after 30 sessions of HBO treatment.

The patient received no medical treatment for 2 days. The surgeon at the regional hospital recommended below-knee amputation of both feet. The patient's wife then requested help from our department. Six days after contracting frostbite the patient was admitted to our hospital with widespread necrosis on the fingers and the plantar and dorsal surfaces of both feet (4th degree frostbite), (Fig. 3) and reported a complete lack of sensitivity in the affected areas. Examination revealed no 2-point discrimination in the feet. Demarcation occurred on the 20th day. Ten days after the HBO_2 treatment began granulation tissue appeared. Daily debridement was performed. The necrosis improved and was limited to small areas. Fourteen days after the HBO_2 treatment the dark crust came off (Fig. 4). Thirty sessions of HBO_2 treatment were carried out. The patient had, by the time of discharge, undergone bilateral transmetatarsal amputation. Skin grafts had been applied to the other affected sites at his regional hospital. It was suggested that the patient attend a follow-up examination 1 month later, but he failed to do so because of the distance between the two cities involved. This meant that we were unable to take final pictures of the injuries.The patient was able to walk.

Discussion

Frostbite was once mainly a military problem.[5] However, recent increasing participation in outdoor winter activities (skiing, mountain climbing, etc.) and homelessness have also made frostbite a civilian population problem.[14] Our patients were engaged in outdoor pursuits when frostbite was contracted and both were civilians.

The anatomical sites most susceptible to frostbite are the hands and feet, which constitute 90% of frostbite injuries. Other susceptible sites are the ears, nose, cheeks and penis. Adults aged between 30 and 49 are the most commonly affected.[14] Immediate treatment consists of rewarming the affected sites with warm water (40—42 °C), though not warmer than 44 °C, over 15—30 min.[3,5] Tetanus prophylaxis and analgesia have to be provided and the affected areas should be protected.[3] The extremity concerned must be elevated and splinted immediately to reduce oedema and promote tissue perfusion.[1]

There are only a few reports in the literature concerning the use of HBO_2 in patients with frostbite.[10,15,16] The first case of HBO_2 treatment in frostbite injury was reported by Ledingham.[10] Ward et al. studied patients who suffered frostbite while mountain climbing. HBO_2 treatment was initiated 5—10 days after injury.[16] Good results were observed in the treatment of frostbite injury using HBO_2.[10,12] Von Heimburg et al. published a case report in which a boy suffered deep frostbite to six fingers. HBO_2 treatment was performed for 14 days. No tissue loss ensued and all the affected areas regained sensitivity. X-ray examination after HBO_2 treatment showed no premature closure of the epiphyses or sclerosis of the metaphyses. Total recovery was observed.[15]

HBO_2 increases leukocyte bactericidal activity, resistance to infection, the growth of new capillaries, collagen synthesis and storage, and oxygen-sensitive fibroplast replication in wound healing[13,17] by increasing the normal level of tissue oxygen pressure by 10—15 times.[4] At the same time, HBO_2 regulates microcirculation and reduces oedema by causing vasoconstriction without the occurrence of hypoxia.[4]

Tissue damage in freezing occurs by two means: cellular damage and vascular insufficiency.[1] Slow freezing leads to the emergence of more ice crystals in the extracellular space than in the intracellular space.[5]

Extracellular ice crystals cause dehydration of cells, which leads to protein denaturation, inhibition of DNA synthesis, loss of cellular membrane integrity, and finally cellular death.[1,6] Cold causes increased blood viscosity and vasoconstriction leading to thromboembolism.[1] After rewarming endothelial injury, thrombosis, tissue oedema, increased compartment space pressure, bleb formation, and tissue death occur. Inflammatory leukocyte infiltration and liberation of free oxygen radicals, prostaglandins, and thromboxane may also play a role in frostbite.[5,11]

We used HBO_2 treatment in deep frostbite of both feet in two patients. Both patients were healthy individuals with no history of circulatory impairment, diabetes mellitus, drug abuse, alcohol con-

sumption, substance abuse. One patient was a heavy smoker, but gave up during the HBO_2 course (Case 1). Both patients improved with HBO_2 treatment.

Conclusion

In frostbite a certain number of cells are injured irreversibly but a large number of cells are injured reversibly, and these may recover and survive with appropriate treatments.[6] The aim of frostbite treatment must be to salvage more irreversibly injured cells.[5] In the early stages of freezing, HBO_2 can assist in the salvaging of a greater amount of tissue by increasing the viability of reversibly damaged cells neighbouring on necrotic tissue by preventing tissue hypoxia and reducing tissue oedema. In the late stage it shortens the duration of hospitalization and reduces the development of secondary infection by accelerating wound healing. At the same time HBO_2 assists the surgeon by permitting the clarification of the demarcation line and increases the success of surgical interventions planned for tissue defects.[7] Prospective clinical studies are needed to demonstrate the beneficial effects of HBO_2 in deep frostbite treatment.

References

1. Biem J, Koehncke N, Classen D, Dosman J. Out of the cold: management of hypothermia and frostbite. CMAJ 2003;168:305—11.
2. Britt LD, Dascombe WH, Rodriguez A. New horizons in management of hypothermia and frostbite injury. Surg Clin North Am 1991;71:345—70.
3. Greenwald D, Cooper B, Gottlieb L. An algorithm for early agressive treatment of frostbite with limb salvage directed by triple-phase scanning. Plast Reconstr Surg 1998;102: 1069—74.
4. Hammarlund C. The physiologic effects of hyperbaric oxygenation. In: Kindwall EP, Whelan HT, editors. Hyperbaric medicine practice. 2nd rev.ed. USA: Best Publishing Com; 2002. p. 37—69.
5. Heggers JP, McCauley RL, Phillips LG, Robson MC. Cold-induced injury: frostbite. In: Herndon DN, editor. Total burn care. London: WB Saunders; 1996. p. 408—14.
6. Hurley LA. Angioarchitectural changes associated with rapid rewarming subsequent to freezing injury. Angiology 1957;8:19—28.
7. Jain KK. Hyperbaric oxygen therapy in wound healing, plastic surgery, and dermatology. In: Jain KK, editor. Textbook of hyperbaric medicine. 4th rev.ed. Germany: Hogrefe & Huber Publishers; 2004. p. 147—67.
8. Knize DM, Weatherly-White RCA, Paton BC, Owens JC. Prognostic factors in the management of frostbite. J Trauma 1969;9:749—59.
9. Kober A, Scheck T, Fulesdi B, Lieba F, Vlach W, Friedman A. Effectiveness of resistive compared to passive warming in treating hypothermia associated with minor trauma: a randomized controlled trial. Mayo Clin Proc 2001;76:369—75.
10. Ledingham I. Some clinical and experimental applications of high pressure oxygen. Proc Roy Soc Med 1963;56:999—1002.
11. Murphy JV, Banwell PE, Roberts AH, McGrouther DA. Frostbite: pathogenesis and treatment. J Trauma 2000;48:171—8.
12. Okuboye JA, Ferguson CC. The use of hyperbaric oxygen in the treatment of experimental frostbite. Can J Surg 1968;11:78—84.
13. Tandara AA, Mustoe TA. Oxygen in wound healing—more than a nutrient. World J Surg 2004;28:294—300.
14. Valnicek SM, Chasmir LR, Clapson JB. Frostbite in the prairies: a 12-year review. Plast Reconstr Surg 1993;92:633—41.
15. von Heimburg D, Noah EM, Sieckmann UP, Pallua N. Hyperbaric oxygen treatment in deep frostbite of both hands in a boy. Burns 2001;27:404—8.
16. Ward MP, Garnham JR, Simpson BRJ, Morley GH, Winter JS. Frostbite: general observations and report of cases treated by hyperbaric oxygen. Proc Roy Soc Med 1968;61:787—9.
17. Warriner III RA, Hopf HW. Enhancement of healing in selected problem wounds. In: Feldmeier JJ, editor. Hyperbaric oxygen 2003: indications and results: The Hyperbaric Oxygen Commitee Report. Kensington,MD: Undersea and Hyperbaric Medical Society;2003.