

Presentation of Two Cases with Hyperbaric Oxygen Therapy Use in Plastic Surgery

 Ayşe İrem İskenderoğlu,¹  Mehmet Emin Elbüken,²  Ali Haydar İskenderoğlu³

¹Department of Plastic Surgery, University of Health Sciences, Hamidiye Faculty of Medicine, Haydarpaşa Numune Health Application and Research Center, Istanbul, Turkey

²Department of Hyperbaric Medicine, Oksipol Hyperbaric Oxygen Treatment Center, Istanbul, Turkey

³Department of Plastic Surgery, Eren Hospital, Istanbul, Turkey

Abstract

Hyperbaric oxygen therapy is a treatment method that involves breathing 100% oxygen in a specially manufactured pressure chamber under a certain pressure and for a certain period of time.

In this article, we present two patients, one with Buerger's disease and the other with osteomyelitis, who were treated with hyperbaric oxygen therapy.

Hyperbaric oxygen therapy is an important adjunct therapy that is generally applied in chronic wounds that do not heal despite surgical procedures and other medical treatments. With this method, in addition to the transported amount with hemoglobin, the oxygen transported in the liquid phase also reaches the problematic tissues. At the same time, due to the antiedema effect of hyperbaric oxygen therapy, it provides an additional contribution to the oxygenation of the tissues. We have obtained successful results by using hyperbaric oxygen therapy in 2 different patients: patient chronic osteomyelitis and with Burger's disease

Keywords: Burger's disease; chronic osteomyelitis; hyperbaric oxygen.

Hyperbaric oxygen therapy (HBOT) is a treatment method that involves breathing 100% oxygen in a specially manufactured pressure chamber under a certain pressure and for a certain period of time. HBOT is an important adjunct therapy that is generally applied to chronic wounds that do not heal despite surgical procedures and other medical treatments. With this method, in addition to the oxygen transported with hemoglobin, the oxygen in the liquid phase also reaches the problematic tissues. At the same time, due to the antiedema effect of hyperbaric oxygen therapy, it contributes additionally to the oxygenation

of the tissues^[1]. In this article, we present two patients, one with Buerger's disease and the other with osteomyelitis, who were treated with hyperbaric oxygen therapy.

Case Report

Case 1 – A 30-year-old female patient presented with an open wound and fistula in the right jaw region of her face. HBOT treatment was planned for the patient who had a deficiency in the right corpus region of the mandible and osteomyelitis of the mandibular bone in the panoramic x-ray. The patient received 40 sessions of HBOT (under 2.5 ATA

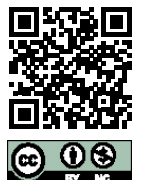
Correspondence (İletişim): Ayşe İrem İskenderoğlu, M.D. Sağlık Bilimleri Üniversitesi Hamidiye Tıp Fakültesi, Haydarpaşa Numune Sağlık Uygulama ve Araştırma Merkezi, Plastik Cerrahi Kliniği, İstanbul, Turkey

Phone (Telefon): +90 216 542 32 32 **E-mail (E-posta):** iremmert@gmail.com

Submitted Date (Başvuru Tarihi): 19.02.2019 **Accepted Date (Kabul Tarihi):** 11.10.2019

Copyright 2021 Haydarpaşa Numune Medical Journal

OPEN ACCESS This is an open access article under the CC BY-NC license (<http://creativecommons.org/licenses/by-nc/4.0/>).



and in 2-hour sessions). During the treatment, antibiotics, analgesics and anti-inflammatory drugs were given when necessary. At the end of 40 sessions, the fistulized open wound on the patient's skin was closed. In the panoramic mandible x-ray, it was observed that the existing bone defect was gone and osteomyelitis was resolved (Fig. 1).

Case 2 – A 35-year-old male patient with a diagnosis of Buerger's disease was admitted with severe circulatory disorders in his lower extremities and a necrotic open wound in the dorsolateral region of the right foot. The patient's right fifth toe was amputated. 110 sessions of HBOT (under 2.5 ATA and in 2-hour sessions) were applied to the patient who did not benefit from debridements, antibiotic therapy and wound care previously. The wound developed granulation tissue and was repaired with a skin graft (Fig. 2).

Discussion

Hyperbaric oxygen is a systemic-effective treatment method in which patients breathe 100% oxygen in a closed cylindrical chamber under pressure increased up to 2, 2.5, 3 ATA above normal atmospheric pressure (1 ATA 760 mmHg) [2]. With the effect of increased pO₂, oxygen pressure in the hypoxic tissue is increased as a result of increased oxygen perfusion from the normally functioning capillaries to the

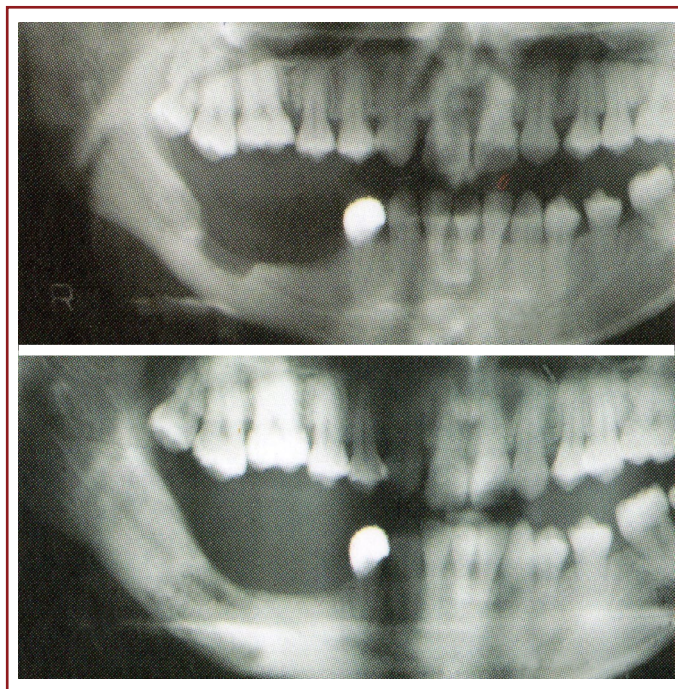


Figure 1. Upper: Bone defect due to chronic osteomyelitis in the right corpus region of the mandible, panoramic X-ray image. (Case 1) Lower: Panoramic mandible X-ray image taken after 40 sessions of hyperbaric oxygen therapy. It is observed that the bone defect in the right corpus is filled. (Case 1)



Figure 2. Upper Left: Necrotic open wound on the dorsum of the right foot in a patient with Buerger's disease. (Case 2)

Upper Right: Patient with Buerger's disease, open wound with granulation tissue on the dorsum of the right foot after 110 sessions of hyperbaric oxygen therapy. (Case 2)

Lower: Patient with Buerger's disease, right dorsum of foot after grafting. (Case 2)

hypoxic wound area^[3]. With HBOT applied in 1 or 2 sessions daily, an increase in collagen production and fibroblast division is provided, which provides support for capillary proliferation. Thus, in an infected open amputation stump, the formation of a rich vascular bed in bone or tissue is accelerated. Healing speed is increased.

Buerger's disease is a non-atherosclerotic inflammatory disease involving small and medium vessels of the upper and lower extremities segmentally. Winimarter defined this disease as "Endarteritis Obliterans" in 1878. It was defined as "Obliterating Thromboangiitis" (TAO) by Buerger in 1908^[4]. Despite various studies, the pathogenesis of this disease has not been fully elucidated. However, today, with the increase of smoking and the increase in air pollution due to rapid advances in technology, it emerges as a vascular pathology that creates a serious public health problem both in terms of diagnosis, treatment and prophylaxis, and as a result of the workforce loss it causes. TAO is primarily a disease of peripheral vessels. The most common involvement is in the lower extremities. Revascularization is often not possible in these patients due to diffuse and distal involvement of the vessels^[4]. Tissue hypoxia develops due to

distal arterial occlusion. In 25% of cases, all four extremities are involved in the disease. The most common localizations are; a. tibialis anterior, a. tibialis posterior, a. plantaris in the lower extremity; and in the upper extremity, a. radialis, a. ulnaris, a. palmaris, and digital arteries. Amputation frequency is higher than amputation rates (70%) due to atherosclerotic ischemia. Intermittent claudication is the first finding in patients.

There is no specific treatment for thromboangiitis obliterans. To date, a medical treatment method that is generally accepted to be effective has not been described. Many controversial treatment options have been tried, and the success rates of the treatment methods vary depending on the severity of the existing disease and the continuation of smoking after treatment. Recently, good results have been reported after recanalization with endovascular therapy, especially in cases with Rutherford Grade III and IV, and autogenous bone marrow-derived cell therapies are also applied at the experimental stage^[5,6]. The most important and only proven fact to prevent disease progression and amputation is to quit smoking, not to use tobacco products in any way, and to avoid smoking places (shared or passive smoking). Extremities should be protected from heat/cold exposure, infections and traumas^[4]. Ischemic ulcerations and pain should be treated. Treatment with calcium channel blockers, antiplatelet agents, pentoxifylline, iloprost (prostacyclin analogue) can be tried. Antibiotic therapy and analgesics are used when necessary. Sympathectomy can be performed. If the patient is suitable for reconstruction, surgical intervention is performed. However, if all treatment methods fail, amputation is inevitable.

In Buerger's disease, while oxygen supply to the tissue decreases as a result of ischemia, there is an increase in tissue metabolism products and CO₂ retention. With HBOT, the low TcPO₂ of hypoxic/ischemic tissues with low perfusion is increased. Thus, it is aimed to correct the cellular metabolism that is impaired due to hypoxia, to reduce the frequency of new gangrene formation and the size of the existing gangrene. It has been suggested that HBO treatment gradually increases the exercise limit in these patients, thus increasing the walking distance and helping the treatment of intermittent claudication^[4]. HBOT treatment is applied to contribute to the healing of wounds due to vascular insufficiency, to help save the extremity in cases secondary to arterial trauma, in cases where surgical intervention is planned or surgical intervention is unsuccessful^[7]. HBOT improves local circulation by reducing the accumulation of algogenic polypeptides, reduces pain by reducing hypoxia and increasing the endorphin sensitivity of receptors. In

addition, it increases exercise capacity by improving blood flow properties, improving perfusion of the ischemic extremity and biochemical deterioration due to exercise.

Chronic osteomyelitis is a bone infection that persists for more than 6-12 weeks despite adequate surgery and antibiotic therapy^[8]. Chronic infection can continue for life. The role of HBOT in the treatment of chronic osteomyelitis is to improve the hypoxic environment of infected bone to normal or above normal levels by increasing oxygen levels. It has been reported that hypoxia occurs in 3 ways in osteomyelitic bone: Excessive O₂ consumption by microorganisms, excessive O₂ consumption by inflammatory cells, impaired local perfusion due to tissue edema. HBO treatment has been shown to have a direct suppressive effect on anaerobic bacteria (About 15% of chronic non-hematogenous osteomyelitis are caused by anaerobes). HBOT is effective in acutely reducing tissue edema, reducing intra-compartmental pressure, relieving and curing the destructive effects of inflammatory reactions^[9].

HBOT stimulates new collagen formation, angiogenesis and increases osteoblastic activity in hypoxic bone and surrounding tissue in the long term^[9].

Conclusion

With HBOT applied in one or two sessions per day in accordance with the protocols made according to the patient's condition and disease, an increase in collagen production and fibroblast division, which provides support for capillary proliferation, is achieved^[10]. Thus, in an infected open amputation stump, the formation of a rich vascular bed in the grafted bone or tissue is accelerated. With the increase in vascular enrichment and tissue pO₂ pressure, the healing rate increases in regional perfusion failure, infected diabetic ulcer, decubitus ulcer, crush-type injuries, osteomyelitis, compartment syndrome and comminuted infected fractures.

With HBOT, there is an increase in the phagocytosis abilities of hypoxic and incompletely functioning leukocytes in the infected tissue, as well as an increase in the rate of killing the bacteria in the phagocytes.

Indications for hyperbaric oxygen therapy are decompression sickness, air or gas embolism, carbon monoxide and cyanide poisoning, acute smoke inhalation, gas gangrene, necrotizing infections of soft tissue (subcutaneous, muscle, fascia), crush injuries, compartment syndrome and other acute traumatic ischemias, wounds with delayed healing (diabetic and non-diabetic), chronic refractory osteomyelitis, excessive blood loss, radiation necrosis, skin flaps and

grafts suspected to survive, thermal burns, brain abscess, anoxic encephalopathy, sudden hearing loss, retinal artery occlusion, acute osteomyelitis of skull bones, sternum and vertebrae and tuberculous osteomyelitis^[11]. We have also obtained successful results by using hyperbaric oxygen therapy in 2 different patients with chronic osteomyelitis and Burger's disease.

Informed Consent: Written informed consent was obtained from the patient for the publication of the case report and the accompanying images.

Peer-review: Externally peer-reviewed.

Conflict of Interest: None declared.

Authorship Contributions: Concept: A.I.I.; Design: A.I.I., A.H.I.; Data Collection or Processing: M.E.E., A.I.I., A.H.I.; Analysis or Interpretation: M.E.E.; Literature Search: A.I.I., A.H.I.; Writing: A.I.I., M.E.E., A.H.I.

Financial Disclosure: The authors declared that this study received no financial support.

References

1. Çimşit M. Hiperbarik Tıp. İstanbul: Eflatun Yayınevi; 2009: 141–3.
2. Çimşit M. Hiperbarik Oksijenin Kullanım Alanları. *Tıbbi Ekoloji ve Hidroklimatoloji Dergisi*, Hiperbarik Oksijenizasyon Özel Sayısı 1984;2:8–15.
3. Jain KK. Physical, Physiological, and Biochemical Aspects of Hyperbaric Oxygenation. In: Jain KK (editor). *Textbook of hyperbaric medicine*. 3th ed. Seattle, Toronto, Bern, Göttingen: Hogrefe & Huber Publishers, 1999: 9–20.
4. Hemsinli D, Kaplan ST, Kaplan S, Yildirim F. Hyperbaric oxygen therapy in the treatment of fontaine stage iv thromboangiitis obliterans. *Int J Low Extrem Wounds* 2016;15:366–70. [\[CrossRef\]](#)
5. Firat A, Igus B. Endovascular recanalization of thromboangiitis obliterans (buerger's disease) in twenty-eight consecutive patients and combined antegrade-retrograde intervention in eight patients. *Cardiovasc Intervent Radiol* 2019;42:820–8.
6. Guo J, Guo L, Cui S, Tong Z, Dardik A, Gu Y. Autologous bone marrow-derived mononuclear cell therapy in Chinese patients with critical limb ischemia due to thromboangiitis obliterans: 10-year results. *Stem Cell Res Ther* 2018;9:43. [\[CrossRef\]](#)
7. Uzun G, Yıldız Ş, Aktaş Ş. Hyperbaric oxygen therapy in the management of nonhealing wounds in patients with critical limb ischemia. *Future Med* 2008;5:99–108. [\[CrossRef\]](#)
8. Mader JT, Brown GL, Guckian JC, Wells CH, Reinartz JA. A mechanism for the amelioration by hyperbaric oxygen of experimental staphylococcal osteomyelitis in rabbits. *J Infect Dis* 1980;142:915–22. [\[CrossRef\]](#)
9. Park M. Effects of hyperbaric oxygen in infectious diseases: basic mechanisms. In: Kindwall EP, Whelan HT (editors). *Hyperbaric Medicine Practice*. 2nd Revised ed. New York: Best Publishing Company, 2002:205–44.
10. Hunt KT, Zederfelt B, Goldstick TK. Oxygen and healing. *Am J Surg* 1969;118:521–5. [\[CrossRef\]](#)
11. Feldmeier JJ. Hyperbaric Oxygen Indications and Results. In: Feldmeier JJ, editor. *The Hyperbaric Oxygen Therapy Committee Report UHMS*. Kensington, Maryland 2003:87–100.