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Editorial

Role of hyperbaric oxygen therapy in periodontitis and implants

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1. Introduction

Oxygen (O_2) makes up 20.8% of atmospheric air and is the universe's third most prevalent element, following hydrogen and helium. It is an essential component of all major biomolecules in living creatures, as well as inorganic substances. All organisms' survival depends on oxygen equilibrium. As a result, organisms devised a method to maintain homeostasis by coordinating oxygen levels within internal compartments. When these systems fail and the intracellular oxygen concentration falls, a stress state known as hypoxia develops.

Oxygen is a necessary chemical for survival. Mammals, including humans, rely on oxygen for electron transport, oxidative phosphorylation, and energy generation. Variations in tissue oxygen demands are caused by a variety of physiological or pathological conditions, which means that the tissues in question must be able to adapt to diverse O2 environments, including hypoxia.

Hyperbaric oxygen therapy was found to enhance oxygen distribution at the base of the pocket, which is harmful to periodontal infections, particularly anaerobic microbes. Cultivation of plaque microorganisms from chronic periodontitis sites yields large percentages of anaerobic (90%) bacterial species. HBO2 stimulates the production of oxygen-free radicals, which oxidize proteins and membrane lipids, damage deoxyribonucleic acid, and impair bacterial metabolic processes. It also aids the oxygen-dependent peroxidase system, which helps leukocytes fight germs. HBO2 also facilitates the oxygen-dependent transport of certain drugs through bacterial cell walls. In this way, HBOT inhibits bacterial growth. HBOT, on the other hand, would provide adequate oxygen intake to ischemic tissues, allowing for a speedy recovery of cell metabolism. Periodontal pockets have very low oxygen tension (pO2 5-27 mmHg) when compared to ambient pO2 (155 mmHg), arterial blood pO2 (95 mmHg), and venous blood pO2 (20-40 mmHg). Fibroblast and leukocyte function are greatly reduced when pO2 is $\leq$30 mmHg. HBO2 promotes collagen synthesis and capillary development. HBO2 stimulates fibroblast proliferation and collagen creation while the patient is in the hyperbaric chamber. It also boosts leukocytes' bactericidal action. HBOT improves gingival microcirculation and increases gingival blood flow.

Thus, HBOT in periodontal tissues has been shown to have both a negative effect on periodontal microbes and a positive effect on periodontal healing by increasing oxygen tension in the pocket.

Schlagenhauf et al. performed repeated subgingival oxygen irrigations on previously untreated periodontal patients. They concluded that repeated oxygen insufflations produced in a significant clinical improvement in periodontal baseline conditions that outperformed the control group.[1]

Chen et al. investigated the effects of HBO2 on aggressive periodontitis (AgP) and subgingival obligate anaerobes in Chinese patients and concluded that HBO2 inhibits the growth of subgingival obligate anaerobes,

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facultative anaerobes, and Bacteroidesmelaninogenicus, promoting peridontium healing and potentially aiding in the treatment of AgP. HBO2 therapy combined with SRP appears to be considerably more effective in the treatment of AgP. The effects can continue more than two years.[2]

2. In Implants

Dental implants provide a way to replace missing teeth. Patients who have had radiotherapy or surgery may benefit from implant-based repair.

Hyperbaric oxygen has been found to influence angiogenesis, bone metabolism, and bone turnover. In terms of radiotherapy, HBO2 can thereby offset some of the deleterious effects of irradiation while also stimulating osseointegration.

The particular processes at the cellular level via which HBO2 acts remain unknown. It has recently been demonstrated that HBO2 and basic fibroblast growth factor (bFGF) work together in irradiated bone. Bone marrow radioprotection, activation of oxygen radical scavengers, and cytokine production are all potential factors in bFGF and HBO2 bone protection.

Hyperbaric oxygen and bFGF can also increase insulin-growth factor levels, which have been shown to stimulate bone proliferation and differentiation. They may also impact bone progenitor cells by boosting DNA synthesis, stimulating bone forming enzymes, or influencing membrane receptors. HBO has also been demonstrated to impact the interface between titanium implants and bone, which may differ from the cellular effect. Oxygen under hyperbaric settings may potentially have a role in osseointegration by influencing bone cell metabolism, implant interface, and the capillary network in the implant bed.

Increases oxygen delivery to periodontal tissues by breathing pure oxygen under pressure, which has been demonstrated to enhance healing and angiogenesis, albeit its routine usage in periodontitis is limited and frequently reserved for refractory or systemic-influenced cases (e.g., diabetic periodontitis).

The main constraint is high cost and limited availability.

1. Local oxygen-releasing medications boost oxygen availability in periodontal pockets, inhibiting anaerobic bacteria and promoting healing.[3]

Agent	Mechanism	Delivery	Benefits	Status
Carbamide peroxide	Breaks into $H_2O_2 \rightarrow O_2$ release	Gel, tray system	Antimicrobial, Promotes healing	Clinically used
Pzone/Ozonated Gels	$O_2 \rightarrow O_2$ + radicals (Antimicrobial)	Gel, Gas, Irrigation	Anti-Inflammatory biofilm disruption	Widely used
Stabilized chiorine Dioxide	Slow oxygen release	Rinse, Gel	Safe, Reduces halitosis, Antimicrobial	Over-the-counter
Mg/Calcium peroxide	Moisture-activated sustained Q_2 release	Coating, paste (Research)	Lon-Acting Oxygenation	Eperimental

3. Conclusion

Periodontal disorders and hypoxia interact in a reciprocal manner. It is widely established that periodontal disorders impair tissue feeding while creating oxygen deficiency. Furthermore, emerging evidence suggests that hypoxia caused by environmental and systemic variables can be regarded an etiological component for periodontal disease, influencing disease progression. The studies reported in this review focused on the activities of HIF and transcriptional regulatory proteins. Interpreting hypoxia in periodontal tissues can shed light on disease causation and prognosis, leading to possible treatment opportunities and associations with inflammatory disorders.

4. Conflict of Interest

None.

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