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Hyperbaric Oxygen Therapy: A Treatment Modality For Avascular Necrosis Of Maxilla- A Clinical Review.

Dr. Heeral Mahlawat, MDS1, Dr. Ashith M.V, MDS2, Dr. Amoli Singh, BDS3,
Dr. Mustafa K., MDS4, Dr. Amrutha Varshini K., MDS5,

1Department of Orthodontics and Dentofacial orthopaedics, Manipal College of Dental Sciences, Mangalore.
Affiliated to Manipal Academy of Higher Education, Manipal, Karnataka. 576104
2 Department of Orthodontics and Dentofacial orthopaedics, Manipal College of Dental Sciences, Mangalore. Affiliated to Manipal Academy of Higher Education, Manipal, Karnataka. 576104
3 Department of Orthodontics and Dentofacial orthopaedics, Manipal College of Dental Sciences, Mangalore. Affiliated to Manipal Academy of Higher Education, Manipal, Karnataka. 576104
4 Department of craniofacial anomalies Kanachur Institute of Medial Sciences Deralakatte, Mangalore-575022
5 Department of craniofacial anomalies Kanachur Institute of Medial Sciences Deralakatte, Mangalore-575022
Corresponding author: Dr. Ashith M.V, MDS. Email: ashith.mv@manipal.edu

ABSTRACT

Oxygen has a vital role in regulating the normal body metabolism and is essential for all tissues. It enhances the vascularity of cells and increases their multiplication there by contributing significantly in wound healing. It is known to enhance the oxygenation of tissues along with having anti-bacterial and anti-inflammatory effects. Oxygen can also intensify the mechanisms involving tissue repair. HBOT (Hyperbaric oxygen therapy) is defined as a treatment modality where the patient is given 100% oxygen in a hyperbaric chamber through intermittent inhalation at a pressure higher than 1 absolute atmosphere for a specified period. The mechanism of action of HBOT is basically by increasing the amount of dissolved oxygen in the body drastically. This causes the oxygen levels in the plasma to increase and leads to better oxygen delivery. The growth of new blood vessels are encouraged and tissue homeostasis is restored. The function of white blood cells are enhanced along with the overall anti-bacterial effect. The aim of writing of this article is to clinically analyse the use of hyperbaric oxygen therapy during a complication post orthognathic surgery with a case discussion.

Keywords: Therapeutic drug, Oxygen therapy, Hyperbaric oxygen therapy

Correspondence:
Dr. Ashith M.V, MDS
Department of Orthodontics and Dentofacial orthopaedics, Manipal College of Dental Sciences, Mangalore. Affiliated to Manipal Academy of Higher Education, Manipal, Karnataka. 576104

*Corresponding author: Dr. Ashith M.V, MDS email-address: ashith.mv@manipal.edu

INTRODUCTION

Oxygen has a vital role in regulating the normal body metabolism and is essential for all tissues. It enhances the vascularity of cells and increases their multiplication there by contributing significantly in wound healing. Oxygen is required by all tissues. The following is an estimation of the percentage of oxygen used by each major tissue: brain (48%), heart (25%), kidneys (15%), skeletal muscle (50%) and skin (5%). Cytochrome C is responsible for metabolizing oxygen. Carbon dioxide and water are the main metabolites involved in the process. Oxygen is considered to be one the most common therapeutic agent that is used. Studies have shown that oxygen therapy can exhibit considerable pharmacological responses and can also affect the physiological processes. It is known to enhance the oxygenation of tissues along with having anti-bacterial and anti-inflammatory effects. Oxygen can also intensify the mechanisms involving tissue repair[1].

Several delivery systems are available that are capable of delivering 24 to 90% oxygen. There is enough data to show that normobaric hyperoxia plays a therapeutic role in the treatment of conditions such as asphyxia, cardiac arrest, cerebral ischemic stroke, drowning and head injuries[2-7].

Data is available on the effects of hypoxia and hyperoxia on the haemodyanamics of the central circulatory system. But literature lacks data on the role they play in the regional haemodynamics. Tissue hypoxia can activate vascular inflammatory processes which in turn can cause an inflammatory response at the systemic level causing multiple organ failure and organ dysfunction[8-12].

The endothelial cells are responsible for regulating the immune and coagulation systems., which in turn regulates the blood supply to the organs and tissues. Endothelial dysfunction can lead to improper functioning of the organ systems.

The site where inflammatory reactions occur in response to pathogens are the interstitial spaces which are hypoxic areas. These areas have restricted blood supply since the vessels are clogged with thrombi or fibrin and other immune cells[13]. The coagulation system gets activated when a rapid dysfunction occurs in the endothelial system. A circulatory dysfunction results from this and the oxygen supply is suppressed. The mitochondria receives inadequate amount of oxygen leading to energy depletion and failure of organs.

HBOT (Hyperbaric oxygen therapy) is defined as a treatment modality where the patient is given 100% oxygen in a hyperbaric chamber through intermittent inhalation at a pressure higher than 1 absolute atmosphere for a specified period[14]. The mechanism of action of HBOT is basically by increasing the amount of dissolved oxygen in the body drastically. This causes the oxygen levels in the plasma to increase and leads to better oxygen delivery. The growth of new blood vessels are encouraged and tissue homeostasis is restored. The function of white blood cells are enhanced along with the overall anti-bacterial effect. HBO therapy also presented as an option for the drug-resistant microorganisms[15].

The first mention of HBOT goes back to the 1600s. As a therapy, HBO therapy began to be administered in 1943. The gold standard for treatment of decompression sickness seen in military divers and aviators, since 1967 has been administration of hyperbaric oxygen with 100% oxygen, starting at 2.8 ATA[16].

The aim of writing of this article is to clinically analyse the use of hyperbaric oxygen therapy during a complication post orthognathic surgery with a case discussion.

Previously hyperbaric oxygen therapy has been used in the treatment of gangrenes and stubborn wounds where there is a severe lack of oxygen supply to the tissue. The damaged blood vessels release fluid which gets leaked into the adjacent tissue leading to swelling. The hyperbaric chamber which is at high pressure causes an increase the blood oxygen levels reducing the swelling and solving the problem of tissue starvation. HBOT enhances the healing process and also block the harmful bacteria. It also helps in the collagen formation and formation of new skin cells.

HBOT is known to accelerate healing and cause a drastic decrease in the inflammation. This treatment along with antibiotic therapy can fight infections and repopulates injured tissue with stem cells, causing regeneration and revitalization of the tissues.

HBOT can be used to treat several conditions such as osteoradionecrosis of the jaws, injury to nerves, dental and facial infections, post-operative pain and inflammation, enhance facial flap healing, etc.

MODE OF ACTION

Hyperbaric oxygen therapy has various effects on the body. These effects can be classified as primary and secondary effects. Primary effects include the direct effects such as increase in the oxygen tension and tissue diffusion rate. Whereas secondary or immediate effects include constriction of vessels, formation of new blood vessels, proliferation of fibroblasts and increase in the leukocyte oxidative killing[17]. These effects that are observed can be described on the basis of gaseous laws and the effects of hyperoxygenation on the physiology and the biochemistry of the body.

According to Henry's Law, the partial pressure of the gas is proportional to the amount of gas which gets dissolved in a liquid or tissue[18]. Hyperbaric oxygen therapy increases the amount of oxygen levels in the body which in turn leads to increase in the oxygen tension of the tissues. This explains the effects of hyperoxia on the hypoxic tissues.

When there is decrease in oxygen tension a neutrophil influx is observed. These neutrophils are activated and they use up an excessive of oxygen. This causes oxygen levels to drop in the hypoxic tissues. Reduced oxygen levels can lead to tissue injuries so to reverse the effect, hyperbaric oxygen therapy is used which causes the concentration of oxygen in the tissues to rise, which in turn helps the neutrophils and accelerates the healing process[19-20].

Hyperoxygenation leads to constriction of vessels in the normal tissues. This effect is beneficial in treating tissue oedema seen post trauma. Hence hyperbaric oxygen therapy is used in the treatment of burns, compartment syndrome and crush injuries. It is also known that there is three-times increase in the diffusion distance of oxygen[21]. In a study that was done on rabbit ear chambers it was observed that the oxygen concentration had an influence on the capillary growth. An increase in the oxygen tension formation of capillaries was also observed[22].

Avascular necrosis is a rare complication seen post operatively in orthognathic surgery. The main factors which can increase the risk of avascular necrosis of maxilla can be local and systematic.

Local factors include

- Radiation treatment
- Infection, trauma
- Surgery related such as- sacrifice of descending palatine artery, perforation/stripping of palatal mucosa, adrenaline injected into mucosa, perioperative vascular thrombosis, segmental osteotomies, extensive advancement.
- Anatomy related- craniofacial dysplasia's, orofacial clefts, vascular anomalies
- Previous surgery- cleft palate repair, surgically assisted rapid palatal expansion

Systemic factors include

- Cigarette smoking
- Pregnancy
- Chemotherapy
- Haematological conditions- Sickle cell disease, Leukaemia, Gaucher's disease, Thalassaemia
- Caisson disease
- Systemic lupus erythematosus
- Diabetes mellitus
- Vasculitis
- Inflammatory bowel disease
- Drugs- Vasoconstrictors, High dose steroids

CASE REVIEW

A 17-year-old male patient had reported to the hospital with a chief complaint of forwardly placed lower teeth. All records were taken for diagnostic purposes and analysed and the patient was diagnosed with Skeletal Class III malocclusion with a Class III molar and canine relationship with a reverse overjet of -1mm. Extra-oral examination revealed a reduced malar prominence. Patient was advised for a bijaw orthognathic surgery post decompensation of the arches by orthodontic treatment.

After an orthodontic treatment of 14 months, the arches were decompensated and a reverse over jet of -6mm was achieved. Facebow was used to transfer the jaw relation on a whip mix articulator and a model surgery was done to determine the amount of maxillary advancement and mandibular setback.

The planned surgery was carried out under general anaesthesia through transmylohyoid intubation under sterile aseptic conditions. Lignocaine with 1:2,00,000 adrenaline was injected in to the surgical site and circumvestibular incision was given using monopolar cautery. Le Fort I osteotomy was performed with 4mm advancement of maxilla and Bilateral sagittal split osteotomy and 4 mm setback of mandible. The intraoperative period was uneventful without any excessive bleeding or any other complication such as perforation of the palatal mucosa. Patient was advised to report for a check-up 1-week post-surgery during which wound infection and avascularity of the maxilla was noticed (Fig 1.). On further examination reduced mouth opening was observed. There was sloughing of gingival tissues in the upper arch specifically in the canine region bilaterally as seen in Fig 1. Additionally, tissue necrosis was seen in posterior part of the palate suggestive of avascular necrosis as a rare complication of Le Fort I osteotomy.

On the 13th post-operative day debridement was done of the maxillary arch (Fig 2.). Followed by hyperbaric oxygen therapy which was started for the patient with 2 cycles per day for 20 minutes each (Fig 3.). A total of 15 HBO (Hyperbaric oxygen) cycles were completed for the patient.

Within a gap of 2 weeks 5 additional HBO cycles were done. Simultaneously he was treated with intravenous amoxiclav, gentamicin and metronidazole for 2 weeks. He was started on therapeutic doses of Pentoxifylline, Vitamin E and multivitamins to improve capillary blood flow.

There was improvement seen in the healing of the necrosed tissues with increased blood flow after 15th HBO cycle (Fig 4.). Mild recession was seen at the canine and molar region which was further treated with periodontal therapy.

DISCUSSION

Post-surgically, in maxilla there is a reduction of blood flow by 50% seen during the first
postoperative day on sacrificing the descending palatine arteries[17]. If only one artery is sacrificed it is seen that there is an excellent blood supply collaterally seen. Any injury to the descending palatine arteries leads to a brief ischaemic period, further which is compensated by proliferation of vessels and permit in healing of tissues as shown by many studies.

This collateral supply from other blood vessels includes facial artery and the ascending pharyngeal artery, helps in maintaining the viability of the palatine pedicle. This pedicle can tolerate 10mm and larger amount of anterior positioning of the maxilla during surgery.

Avascular necrosis following Le Fort I osteotomies has been accredited for a weakly vascular palatal pedicle. According to the hypothesis by Bell, repositioning dento-osseous structures significantly, design of the soft tissue flap, widening of the vascular palatal pedicle, division of maxilla into multiple sections, hypotension or transection of the descending palatine vessels, might help lead to this complication[23-24].

It is suggested by previous literature that segmental osteotomy shows a higher risk of ischaemic
Complications[23-24]. Maxillary avascular necrosis can be curtailed in the subsequent ways:

– Small segments of maxilla should be avoided anteriorly, and division should be reduced to few segments only.

– Integrity of the palatal mucosa should be maintained.

– Sagittal segmentation should be performed at paramedian sites because of the mucosa being
thicker and bone thinner in that region compared to the midline.

Even though hypotensive anaesthesia is not used on purpose for any procedure, it is a method most used to reduce blood loss and keep a bloodless surgical field[25]. A mean arterial blood pressure (MAP) is 30% below a patient's usual MAP, with a minimum MAP of 50mmHg in American society of anaesthesiologists Class I patients and a MAP not <80mmHg in the elderly, is advised to be clinically acceptable[26]. Its suggested that with respect to hypotensive anaesthesia:

– Adjustment must be done with respect to patient's preoperative blood pressure rather than
specific target pressure.

– The level must be restricted to the extent of reducing bleeding in the surgical field.

– The part of surgical procedure to which anaesthesia is given should be confined deemed to benefit by it[27].

CONCLUSION

Although HBOT has wide-ranging uses in medical field. But its definitive use in orthognathic surgical cases requires established evidences. Studies and reviews must begin in the field of dentistry, to provide advanced treatment possibilities and benefits of hyperbaric oxygen therapy.

REFERENCES

1. Devaraj D, Srisakthi D. Hyperbaric oxygen therapy–can it be the new era in dentistry? Journal of clinical and diagnostic research: JCDR. 2014 Feb;8(2):263.
2. Bouachour G, Cronier P, Gouello JL et al: Hyperbaric oxygen therapy in the management of crush injuries: a randomized double-blind placebo-controlled clinical trial. J Trauma, 41(2):333-39 (1996).
3. Sukoff MH, Ragatz RE: Hyperbaric oxygenation for the treatment of acute cerebral edema. Neurosurgery. 10: 29-38 (1982).
4. Nemoto EM, Bettermann K: Basic physiology of hyperbaric oxygen in brain. Neurol Res. 29:116-126 (2007).
5. Rockswold SB, Rockswold GL, Defillo A: Hyperbaric oxygen in traumatic brain injury. Neurol Res. 29: 162-72 (2007).
6. Rusyniac DE, Kirk MA, May JD et al: Hyperbaric oxygen therapy in acute ischemic stroke: results of the hyperbaric oxygen in acute ischemic stroke trial pilot study. Stroke 34: 571-74 (2003).
7. Singhal AB: Oxygen therapy in stroke: past, present and future. Int J. Stroke 4: 191-200 (2006).
8. Bell WH, You ZH, Finn RA, Fields T. Wound healing after multisegmental Le Fort I osteotomy and transection of the descending palatine vessels. J Oral Maxillofac Surg 1995; 53:1425–33.
9. Nathan C: Oxygen and the inflammatory cell. Nature. 17: 675-676 (2003).
10. Lefer AM, Lefer DJ: Pharmacology of the endothelium in ischemia-perfusion and circulatory shock. Ann Rev Pharmacol. Toxicol 33: 71-90 (1993).
11. Dietch EA: Gut failure:its role in the multiple organ failure: Pathophysiology and BasicConcepts of Therapy. New York: Thieme Medical Publisher; 4-59 (1990).
12. Buras J: Basic mechanism of hyperbaric oxygen in the treatment of ischemia-reperfusion injury. Int. J. Anesthesiol. Clin. 38: 91-109 (2003).
13. Hirota K: Involvement of hypoxia-inducible factors in the dysregulation of oxygen homeostasis in sepsis. Cardiovasc Hematol Disord Targets 15(1): 29-40 (2015).
14. Hance Clarke. The role of hyperbaric oxygen therapy within oral and maxillofacial surgery. Toronto: J Oral Hyg Health 2016, 4:3 (Suppl)
15. Danesh-Sani S, Shariati-Sarabi Z, Feiz M. Comprehensive Review of Hyperbaric Oxygen Therapy. Journal of Craniofacial Surgery. 2012;23(5): e483-e491.
16. Davis JC. Hyperbaric oxygen therapy. Journal of Intensive Care Medicine. 1989; 4:55-57.
17. Vandana Mehta, Abhishek De, Balachandran C. Hyperbaric oxygen therapy. Journal of Pakistan Association of Dermatologists. 2009;19: 164-67.
18. AL Gill, CNA Bell. Hyperbaric oxygen therapy: its uses, mechanism of action and outcomes. Q J Med. 2004; 97: 385-95.
19. David R Knighton, Vance D Fiegel, Tim Halverson, Susan Schneider, Tony Brown, Carol L Wells. Oxygen as an antibiotic-The effect of inspired oxygen on bacterial clearance. Arch Surg. 1990; 125:97-100.

20. Knighton DR, Halliday B, Hunt TK. Oxygen as an antibiotic-the effect of inspired oxygen on infection. Arch Surgery. 1984; 119:199-204.
21. Wattel F, Mathieu D, Neviere R, Bocquillon N. Hyperbaric therapy: acute peripheral ischaemia and compartment syndrome: A role for hyperbaric oxygenation. Anaesthesia. 1998; 53:63-65.
22. Knighton DR, Silver IA, Hunt TK. Regulation of wound healing angiogenesis- effect of oxygen gradients and inspired oxygen concentration. Surgery. 1981; 90:262- 70.
23. Ho MW, Boyle MA, Cooper JC, Dodd MD, Richardson D. Surgical complications of segmental Le Fort I osteotomy. Br J Oral Maxillofac Surg 2011; 49:562–6.
24. Lanigan D, Hey JH, West RA. Aseptic necrosis following maxillary osteotomies. J Oral Maxillofac Surg 1990; 48:142–56.
25. Pineiro-Aguilar A, Somoza-Martin M, Gandara-Rey JM, Garcia-Garcia A. Blood loss in orthognathic surgery: a systemic review. J Oral Maxillofac Surg 2011; 69:885–92.
26. Rodrigo C. Induced hypotension during anesthesia, with special reference to orthognathic surgery. Anesth Prog 1995;42: 41–58.
27. Choi BK, Yang EJ, Oh KS, Lo LJ. Assessment of blood loss and need for transfusion during bimaxillary surgery with or without maxillary setback. J Oral Maxillofaci Surg 2013;71: 358–65.

FIGURES

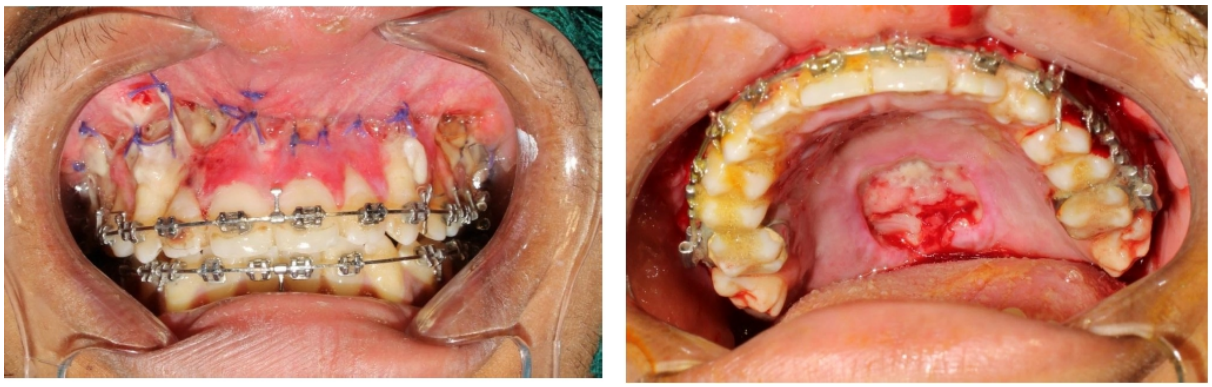


FIG 1. Avascular necrosis on maxilla as seen 1-week post-surgery.

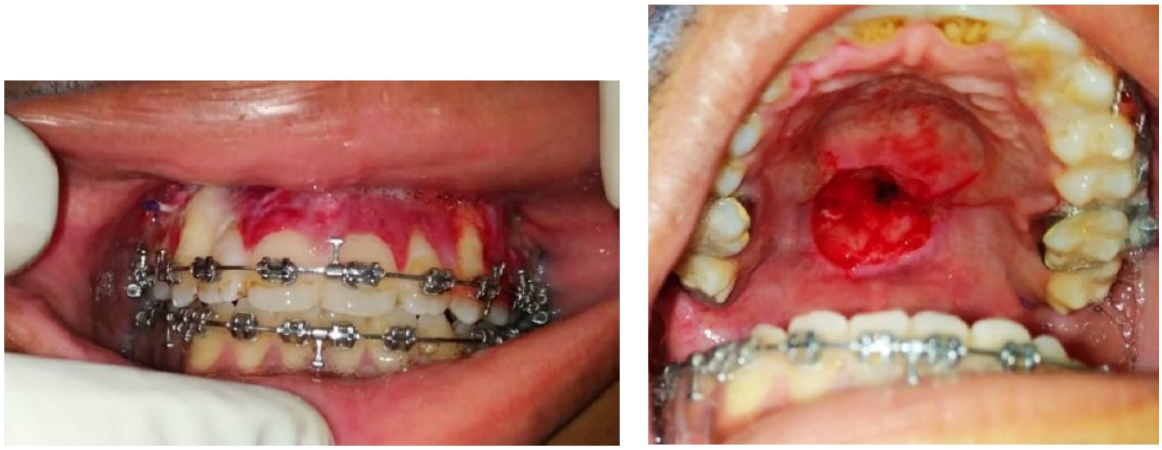


FIG 2. Post Debridemen

FIG 3. 10th HBO cycle given. Improved vascularity can be seen in the maxilla with healing of tissues.

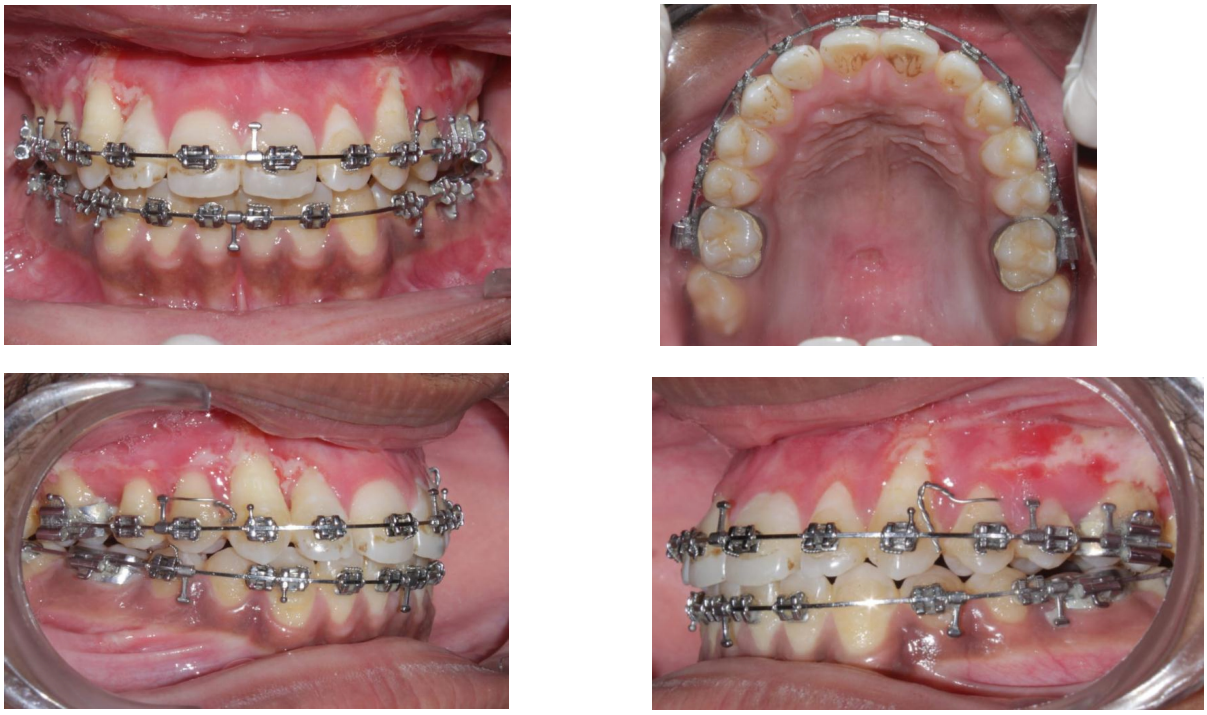
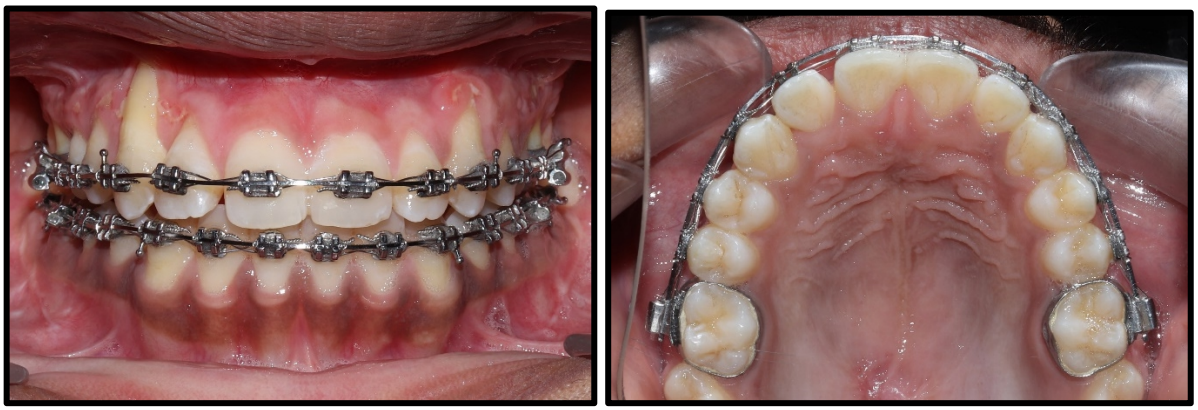


FIG 4. Improved healing of tissues seen after the 15th HBO cycle. Mild recession is seen in the canine and molar region.



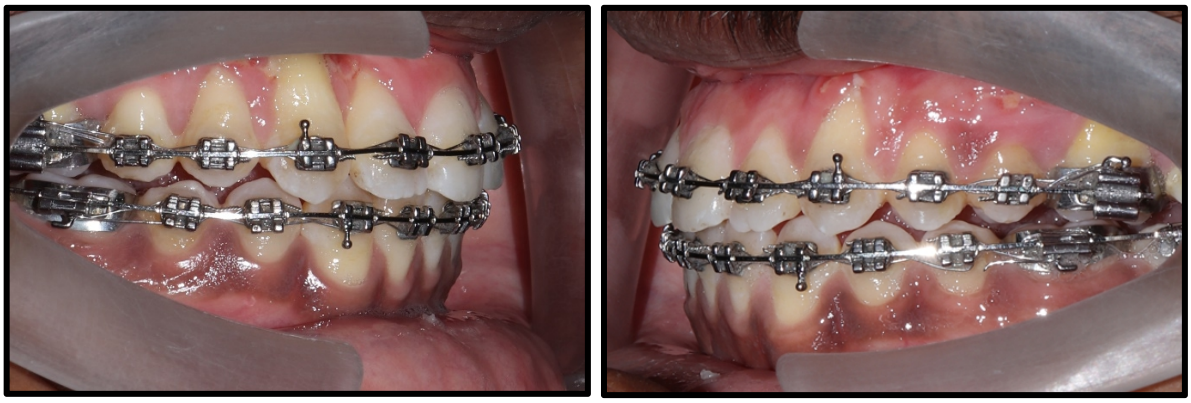


FIG 5. 6 months in treatment after complete hyperbaric oxygen therapy. Free gingival graft treatment is planned in respect to upper right canine.