



Volume 11, Issue 1, 32-42

Research Article

SJIF Impact Factor 7.632

ISSN 2278 - 4357

9

APPLICATIONS OF HYPERBARIC OXYGEN THERAPY IN OSTEOPOROSIS

Khodor Haidar Hassan*, Joelle Azzi, Gian Marco Oppo, Hadi Raef Rida, Roberto Vecchioni, Mohamad Ali Chahrour, Mehdi Raef Rida, Fadel Nahle, Hadi Farhat, Edwin Parra Prada and Ahmad Najib Ballout

MD, PhD, Lebanese University, Hadath, Beirut, Faculty of Public Health.

Article Received on 27 October 2021,

Revised on 17 Nov. 2021, Accepted on 07 Dec. 2021 DOI: 10.20959/wjpps20221-20850

*Corresponding Author Prof. Khodor Haidar Hassan MD, PhD, Lebanese University, Hadath, Beirut, Faculty of Public Health.

ABSTRACT

Introduction: Hyperbaric Oxygen Therapy (HBOT) is a recommended treatment for all hypoxic pathologies. Hyperbaric Oxygen Therapy (HBOT) consists of breathing 100% oxygen or enriched mixtures at pressures higher than atmospheric pressure, in a suitable environment. HBOT has been used for decades as a recommended treatment for hypoxic-ischemic and infectious disorders. The pressures utilized vary from 2.2 to 2.8 ATA. Materials and Methods: 20 Osteoporosis patients treated underwent HBOT at 1.5 ATA and 100% oxygen for a total of 15 sessions. Results: 20 postmenopausal women with osteoporosis aged between 38 -64 with a

median age of 60.5 are treated with HBOT. They are assigned for 15 sessions;5 sessions per week; 1hour per session then repeated after 15 days rest for 15 sessions for 3 sessions per week. The bone mineral density (BMD) findings before and after the sessions show a significant value increase of 18.5% with a 1% margin of errors. 19 out of 20 patients show a high reduction in lumbar spasms. 17 out of 20 patients marked a significant drop from high to moderate and low pain. 15 out of 20 patients manifest a clinical improvement with a range of motion in the lumbar spine. **Conclusions:** Osteoporosis patients treated with HBOT were demonstrated. No side effects were observed in any of the women who were treated.

KEYWORDS: The bone mineral density (BMD) findings before and after the sessions show a significant value increase of 18.5% with a 1% margin of errors.

INTRODUCTION

Osteoporosis is a severe bone disease characterized by low bone mineral density (BMD),

microarchitectural deterioration of bone tissue, compromised bone strength which weakens the bones and considerably increases the fractures, particularly in the hip, spine, and wrist.^[1-11]

Known as the silent epidemic, the risk of fractures increases with age after peak bone mass reaches its maximum in the late third or early fourth decade of life.^[2,7,9,10,12,13] However, fractures happen in one in three women versus one in five men.^[2,3,7,9,14,15] Past the age of 50 years following the onset of menopause, osteoporosis is mainly pronounced in postmenopausal women^[5,10,16] due to an imbalance between bone resorption (osteoclasts) and formation (osteoblasts), responsible for the disruption in the bone remodeling cycle.^[5,9,17,18,19]

This skeletal disease reduces the quality of life and increases morbidity and mortality in persons.^[1,8,20-25] Therefore, all patients with existing fractures at the spine or the hip should be screened for osteoporosis as having a BMD T-score of less than or equal to 2.5 standard deviations (SDs), and it is usually measured via dual-energy x-ray absorptiometry (DXA).^[2,3,8-11,20,21,26-28]

Osteoporosis is acknowledged as a worldwide public health problem, affecting 50 million Americans in the United States.^[2,7,21,29] In addition, more than 90,000 hip fractures per year are reported in patients over 50 years in Italy.^[30,31] Osteoporotic patients are incurable because no treatment can fully restore the reduced BMD caused by the disease.^[2,21] Thus, to maintain and promote bone health, adequate exercise and protein, calcium, and vitamin D supplementation reduce the risk of fracture and the financial burden of osteoporosis in society.^[9,11,20] In addition, several treatments such as alendronate, risedronate, zoledronic acid, or denosumab can be initiated and recommended for osteoporotic patients at high risk of fracture.^[20,30,32-35]

Hyperbaric Oxygen Therapy (HBOT) is a non-invasive method that consists of breathing 100% oxygen or enriched mixtures at pressures higher than atmospheric pressure in a suitable environment (hyperbaric chamber). Thus, the forces utilized vary from 2.2 to 2.8 ATA.^[36-42]

HBOT has been used as an alternative or adjunct treatment that can effectively treat chronic osteomyelitis, rheumatoid arthritis, transient osteoporosis of the hip (TOH), avascular necrosis, orthopedic disorders, femoral head necrosis, osteoradionecrosis, bone grafts and dental implants compromised skin grafts/flaps, hypoxic-ischemic, crush injuries, diabetic foot

problems, infectious disorders, gas gangrene, autism, arterial gas embolism, carbon monoxide poisoning, decompression sickness and other medical areas with strong evidence.^[43-82]

Several studies demonstrated the mechanism of action of HBOT on the skeleton and bone formation in orthopedics.^[18,19,43-82] It:

- 1- Improves oxygen supply in bone tissue
- 2- Induces vasoconstriction and reduce bone marrow pressure
- 3- Stimulates cellular proliferation and collagen synthesis
- 4- Improves bone healing
- 5- Stimulates angiogenesis and osteogenesis 6- Alleviates the pain
- 6- Produces free radical, synthesis of cytokine and modulate the immune response
- 7- Enhances osteoclast and osteoblast function for remodeling and repair

Therefore, the present study evaluated HBOT effectiveness in postmenopausal women with osteoporosis for accelerated recovery, identified the incidence of complications among patient treatments, and assessed HBOT as a recommended treatment.

MATERIALS AND METHODS

Twenty menopausal patients with osteoporosis, aged between 38 to 73 years, underwent HBOT. During the study, all pharmacological treatments for osteoporosis were discontinued. The study protocol was approved by institutional review boards (IRB) of the hospitals before study initiation. In addition, written informed consent was obtained from each participant.

Patients with untreated pneumothorax or severe chronic obstructive pulmonary disease (COPD), epilepsy, hyperthermia, hypoglycemia, panic disorder, drug abuse, or other diseases were excluded.

Oxygen at 100% was administered at a pressure of 2.2 ATA. All patients underwent an entire course of treatment consisting of 15 daily sessions, five times per week followed by 15 days off, then 15 additional sessions every other day and every three weeks. The sessions took place over a total period of 60 minutes.

To evaluate bone mineral density, an ACM GAMMA dual-photon absorptiometry (DPA) with gadolinium 153 was performed before and after the complete sessions of HBOT. Densitometric BMD measurements were taken at the level of 304 between L1 and L4 of lumbar vertebrae unless there is clinical indication for the study of the last dorsal vertebrae.

In addition, patients experiencing certain clinical signs such as active and passive mobility of the spine and limbs, spinal or osteoarticular pain, the feeling of "more relaxation" of the patient, joint and limb pain is relieved.

For further research, it is expected that a third mineralometry dual photon will be performed fourmonths after the second one and that half of the patients will receive daily treatment with calcitonin spray. In contrast, the other half will refrain from a specific medical treatment for osteoporosis.

This is to evaluate the effects of exposure to HBOT on the bone remodeling cycle via osteoclast inhibition and osteoblast activation.

RESULTS

20 post-menopausal women with osteoporosis aged between 38 -64 with a median age of 60.5 were treated with HBOT. They were assigned for 15 sessions; 5 sessions per week; 1hour per session then repeated after 15 days rest for 15 sessions for 3 sessions per week. The BMD findings before and after the sessions showed a significant value increase of 18.5% with a 1% margin of errors. 19 out of 20 patients showed a high reduction in lumbar spasms. 17 out of 20 patients marked a significant drop from high to moderate and low pain. 15 out of 20 patients manifest a clinical improvement with a range of motion in the lumbar spine.

DISCUSSION

As we age, we lose vital bone capacity and impair oxygen utilization. Pressurized oxygen can correct bone pathologies such as chronic osteomyelitis, aseptic necrosis, and poorly consolidated fractures. Our current finding shows that treating osteoporotic patients with HBOT can significantly improve BMD, promote recovery of the lumbar spine reducelumbar spasms and pain. These results are consistent with previous studies. HBOT can significantly increase calcium, collagen deposition, significant components of bone matrix, and essential framework for bone mineralization. HBOT can gradually restore osteoprogenitor cells (osteocytes activity) and bone microcirculation, improve ischemia and hypoxia at the fracture sites through time, and extend the degradation phase, which increases bone hardness and flexibility, thereby preventing and treating osteoporosis.^[18,19,45,47,48,49,51,55,56,60,76,83,84] Assessment of osteoporotic risk factors and measurement of bone mineral density may help identify patients who will benefit from HBOT intervention and potentially reduce the morbidity and mortality associated with osteoporosis-associated fractures in this population.

CONCLUSIONS

Osteoporosis patients treated with HBOT were demonstrated in this study. No side effects were observed in any of the women who were treated. The results of this research suggest that HBOT is an acute treatment with high efficacy, capable of inducing a rapid increase in bone calcium tone. However, the duration of the effect remains to be specified, since hyperbaric O2 does not intervene on the causal factors of osteoporosis, but only on the possible mechanisms that regulate the function of osteocytes. Our study was done over a period of three months. It is planned to examine the duration of BMD improvement over a two-year period along with the possible enhancement of the action of the physical hyperbaric treatment and the pharmacological treatment.

REFERENCES

- Lippuner K. Medical treatment of vertebral osteoporosis. Eur Spine J., 2003 Oct; 12 Suppl 2(Suppl 2): S132-41. doi: 10.1007/s00586-003-0608-x.
- Katharina Kerschan-Schindl. Prevention and rehabilitation of osteoporosis. Wien Med Wochenschr, 2016 Feb; 166(1-2): 22-7. doi: 10.1007/s10354-015-0417-y.
- Jian Wu et al. Association of collagen type I alpha 1 +1245G/T polymorphism and osteoporosis risk in post-menopausal women: a meta-analysis. Int J Rheum Dis., 2017Jul; 20(7): 903-910. doi: 10.1111/1756-185X.13052.
- Golob AL, Laya MB (2015) Osteoporosis: screening, prevention, and management. Med ClinN Am, 99: 587–606.
- P Tu et al. Polymorphisms in genes in the RANKL/RANK/OPG pathway are associated with bone mineral density at different skeletal sites in post-menopausal women. Osteoporos Int., 2015 Jan; 26(1): 179-85. doi: 10.1007/s00198-014-2854-7
- 6. LiWF, Hou SX, Yu B, LiMM, Ferec C, Chen JM(2010) Genetics of osteoporosis: acceleratingpace in gene identification and validation. Hum Genet, 3: 249–285.
- Nancy E. Lane. Epidemiology, etiology, and diagnosis of osteoporosis. American Journal ofObstetrics and Gynecology, 2006; 194: S3–11.
- Armas LA et al. Pathophysiology of Osteoporosis new mechanistic insights. Endocrinol Metab Clin North Am., 2012 Sep; 41(3): 475-86. doi: 10.1016/j.ecl.2012.04.006. Epub 2012 Jun 9.PMID: 22877425
- Coughlan.T, Dockery.F. Osteoporosis and fracture risk in older people. Clin Med (Lond). 2014. Clinical Medicine, 2014; 14(2): 187–91.
- 10. Aspray TJ et al. Osteoporosis and the Ageing Skeleton. Subcell Biochem, 2019; 91:

453-476. doi: 10.1007/978-981-13-3681-2_16. PMID: 30888662 Review.

- 11. Kristine E Ensrud et al. Osteoporosis. Ann Intern Med., 2017 Aug 1; 167(3): ITC17-ITC32. doi: 10.7326/AITC201708010. PMID: 28761958.
- Eastell R, Szulc P. Use of bone turnover markers in postmenopausal osteoporosis. Lancet Diabetes Endocrinol, 2017 Nov; 5(11): 908-923. doi: 10.1016/S2213-8587(17)30184-5.
- Carl Neuerburg et al. Investigation and management of osteoporosis in aged trauma patients: a treatment algorithm adapted to the German guidelines for osteoporosis. J Orthop Surg Res., 2017 Jun 8; 12(1): 86. doi: 10.1186/s13018-017-0585-0. PMID: 28595648. PMCID:PMC5465580.
- Crandall CJ et al. A Comparison of US and Canadian Osteoporosis Screening and Treatment Strategies in Postmenopausal Women. J Bone Miner Res., 2019 Apr; 34(4): 607-615. doi: 10.1002/jbmr.3636. Epub 2019 Jan 15. PMID: 30536628.
- Oden A, McCloskey E, Kanis J, Harvey N, Johansson H (2015) Burden of high fracture probability worldwide: secular increases 2010–2040. Osteoporosis Int., 26: 2243–8.
- 16. Pasco JA et al. The Epidemiology of Incident Fracture from Cradle to Senescence. Calcif Tissue Int., 2015 Dec; 97(6): 568-76. doi: 10.1007/s00223-015-0053-y. Epub 2015 Aug 29. PMID:26319674. DOI: 10.1007/s00223-015-0053-y
- Garnero P et al. Increased bone turnover in late postmenopausal women is a major determinant of osteoporosis. J Bone Miner Res., 1996 Mar; 11(3): 337-49. doi: 10.1002/jbmr.5650110307. PMID: 8852944
- Hadil Al Hadi et al. Hyperbaric oxygen therapy suppresses osteoclast formation and bone resorption. J Orthop Res., 2013 Nov; 31(11): 1839-44. doi: 10.1002/jor.22443. Epub 2013 Jul 22.
- McArdle A et al. The role and regulation of osteoclasts in normal bone homeostasis and in response to injury. Plast Reconstr Surg., 2015 Mar; 135(3): 808-816. doi: 10.1097/PRS.000000000000963. PMID: 25719699.
- 20. Baccaro.L.F et al The epidemiology and management of postmenopausal osteoporosis: a viewpoint from Brazil. Clin Interv Aging, 2015; 10: 583-91. doi: 10.2147/CIA.S54614.
- Vu H. Nguyen. Osteoporosis prevention and osteoporosis exercise in community-based public health programs. Osteoporos Sarcopenia, 2017 Mar; 3(1): 18-31. doi: 10.1016/j.afos.2016.11.004.
- 22. Johnell O et al. Mortality after osteoporosis fractures. Osteoporos Int, 2004; 15: 38e42.
- 23. Leboime A et al. Osteoporosis and mortality. Jt Bone Spine, 2010; 77: S107e12.
- 24. Neuerburg.C et al. Investigation and management of osteoporosis in aged trauma patients:

37

a treatment algorithm adapted to the German guidelines for osteoporosis. Journal of OrthopaedicSurgery and Research, 2017; 12: 86.

- 25. L M Giangregorio et al. Too Fit To Fracture: exercise recommendations for individuals with osteoporosis or osteoporotic vertebral fracture. Osteoporos Int., 2014 Mar; 25(3): 821-35. doi: 10.1007/s00198-013-2523-2.
- 26. Rudäng R, et al. Bone material strength is associated with areal BMD but not with prevalent fractures in older women. Osteoporos Int., 2016. Apr; 27(4): 1585-1592. doi: 10.1007/s00198-015-3419-0. PMID: 26630975.Epub 2015 Dec 2.
- Muschitz C, et al. Prevalence of vertebral fracture in elderly men and women with osteopenia. Wien Klin Wochenschr, 2009; 121(15-16): 528-36. doi: 10.1007/s00508-009-1216-5.PMID: 19787324
- 28. E S Siris et al. The effect of age and bone mineral density on the absolute, excess, and relative risk of fracture in postmenopausal women aged 50-99: results from the National Osteoporosis Risk Assessment (NORA). Osteoporos Int., 2006; 17(4): 565-74. doi: 10.1007/s00198-005-0027-4. Epub 2006 Jan 4. PMID: 16392027.
- 29. Wright NC, et al. The recent prevalence of osteoporosis and low bone mass in the United States based on bone mineral density at the femoral neck or lumbar spine. J Bone Miner Res., 2014 Nov; 29(11): 2520-6. doi: 10.1002/jbmr.2269.PMID: 24771492
- 30. Stefano Gonnelli et al. Prescription of anti-osteoporosis medications after hospitalization for hip fracture: a multicentre Italian survey. Aging Clin Exp Res., 2017 Oct; 29(5): 1031-1037.doi: 10.1007/s40520-016-0681-8. PMID: 27943127. Epub 2016 Dec 9.
- 31. Piscitelli P et al. Incidence and costs of hip fractures vs strokes and acute myocardial infarction in Italy: comparative analysis based on national hospitalization records. Clin Interv Aging., 2012; 7: 575-83. doi: 10.2147/CIA.S36828. Epub 2012 Dec 17. PMID: 23269863
- 32. Rossini M, et al. Guidelines for the diagnosis, prevention and management of osteoporosis.Reumatismo, 2016. Jun 23; 68(1): 1-39. doi: 10.4081/reumatismo.2016.870.
 PMID: 27339372
- Kanis JA et al. European guidance for the diagnosis and management of osteoporosis in postmenopausal women. Osteoporos Int., 2019 Jan; 30(1): 3-44. doi: 10.1007/s00198-018-4704-5.Epub 2018 Oct 15. PMID: 30324412
- 34. Kanis JA et al. Algorithm for the management of patients at low, high and very high risk of osteoporotic fractures. Osteoporos Int., 2020 Jan; 31(1): 1-12. doi: 10.1007/s00198-019-05176-3.Epub 2019 Nov 13. PMID: 31720707

- 35. Qaseem A et al. Treatment of Low Bone Density or Osteoporosis to Prevent Fractures in Men and Women: A Clinical Practice Guideline Update From the American College of Physicians. Ann Intern Med., 2017 Jun 6; 166(11): 818-839. doi: 10.7326/M15-1361. Epub 2017 May 9. PMID: 28492856.
- 36. Learch RM et al. Hyperbaric oxygen therapy. BMJ, 1998; 317(7166): 1140-1143.
- Ossigeno iperbarico. Fisiologia ed applicazioni terapeutiche. Ed: Studio(Chieti), 1984.38-Medicina subacquea iperbarica. Data PG. Ed: Studio (Chieti), 1984.
- Recenti acquisizioni fisiologiche sull'ossigeno in iperbarismo.Sparacia A, Sparacia B. Minerva Anestesiol, 1991; 57: 187-204.
- 39. Ossigenoterapia iperbarica. Applicazioni cliniche. Oriani G, Faglia E. Ed SIO, via Capelcelatro 69, 20129, Milano, 1999.
- 40. Textbook of Hyperbaric Medicine, Jain K.K Editon:5 Upd Exp, April 3, 2009.
- 41. Hyperbaric Oxygen Therapy. Committe Report Editor, N:B: Hampson. Undersea and Hyperbaric Medical Society, Kensington MD, 1999.
- 42. KESİKBURUN.S et al. Transient Osteoporosis of the Hip and Hyperbaric Oxygen Therapy: A Report of Two Cases. Turk J Phys Med Rehab, 2015; 61: 80-3.
- 43. Reis ND et al. Hyperbaric oxygen therapy as a treatment for stage-I avascular necrosis of thefemoral head. J Bone Joint Surg Br, 2003; 85: 371-5.
- 44. Wu D et al.(2007) Effects of hyperbaric oxygen on proliferation and differentiation of osteoblasts from human alveolar bone. Connect Tissue Res., 48: 206–213.
- 45. Kawada S, Ohtani M, Ishii N (2010). Increased oxygen tension attenuates acute ultraviolet-B- induced skin angiogenesis and wrinkle formation. Am J Physiol Regul Integr Comp Physiol, 299: R694–R701.
- 46. Lu C et al. (2013) The role of oxygen during fracture healing. Bone, 52: 220–229.
- 47. Sammarco.MC et al. Hyperbaric Oxygen Promotes Proximal Bone Regeneration and Organized Collagen Composition during Digit Regeneration. PLoS One., 2015 Oct 9; 10(10): e0140156.
- 48. Juan.L et al. Impact of Hyperbaric Oxygen on the Healing of Bone Tissues Around Implants.Implant Dent., 2018 Dec; 27(6): 653-659.
- Liao.J et al. Impact of Hyperbaric Oxygen on Tissue Healing around Dental Implants in Beagles. Med Sci Monit., 2018 Nov 13; 24: 8150-8159.
- 50. Sacco.R et al. A Systematic Review of Oxygen Therapy for the Management of Medication-Related Osteonecrosis of the Jaw (MRONJ). Appl. Sci., 2019; 9(5): 1026. DOI: 10.3390/app9051026

- 51. Vezzani.G et al. Hyperbaric oxygen therapy modulates serum OPG/RANKL in femoral head necrosis patients. JOURNAL OF ENZYME INHIBITION AND MEDICINAL CHEMISTRY, 2017; 32(1): 707–711.
- Sen RK. Management of avascular necrosis of femoral head at pre-collapse stage. Indian J Orthop, 2009; 43: 6–16.
- Camporesi EM et al. Hyperbaric oxygen therapy in femoral head necrosis. J Arthroplasty, 2010; 25(6): 118-123.
- 54. Bennett M. Hyperbaric oxygen therapy improved both pain scores and range of motion in patients with early idiopathic femoral head necrosis (Ficat stage II). Diving Hyperb Med, 2011; 41: 105.
- 55. Al Hadi H, Smerdon GR, Fox SW. Hyperbaric oxygen therapy accelerates osteoblast differentiation and promotes bone formation. J Dent, 2015; 43: 382–8.
- Gretl Lam et al. Hyperbaric Oxygen Therapy: Exploring the Clinical Evidence. Adv Skin Wound Care., 2017 Apr; 30(4): 181-190. doi: 10.1097/01.ASW.0000513089.75457.22.
- 57. Koren.L et al. Hyperbaric Oxygen for Stage I and II Femoral Head Osteonecrosis. Orthopedics, 2015 Mar; 38(3): e200-5.
- 58. Strauss M et al. Femoral head necrosis and hyperbaric oxygen therapy. In: Hyperbaric Medicine Practice. 3rd ed. Vol. 34. North Palm Beach, FL: Best, 2008: 943.
- 59. Arnett TR et al. 2003. Hypoxia is a major stimulator of osteoclast formation and bone resorption. J Cell Physiol, 196: 2–8.
- 60. Fukuoka H et al. 2005. Hypoxic stress enhances osteoclast differentiation via increasing IGF2 production by non-osteoclastic cells. Biochem Biophys Res Commun, 328: 885–894.
- 61. Knowles HJ, Athanasou NA. 2008. Hypoxia-inducible factor is expressed in giant cell tumour of bone and mediates paracrine effects of hypoxia on monocyte-osteoclast differentiationvia induction of VEGF. J Pathol, 215: 56–66.
- 62. Utting JC et al. 2010. Hypoxia stimulates osteoclast formation from human peripheral blood.Cell Biochem Funct, 28: 374–380.
- 63. Kawashima M, Tamura H, Nagayoshi I, et al. Hyperbaric oxygen therapy in orthopedic conditions. Undersea Hyperb Med, 2004; 31: 155-62.
- 64. Zamboni WA. The microcirculation and ischaemia-reperfusion: basic mechanism of hyperbaric oxygen. In Whelan K (ed.) Hyperbaric medicine practice. Flagstaff: Best Publishing, 2004: 779-94.
- 65. Zelinski LM, Ohgami Y, Chung E, Shirachi DY, Quock RM. A prolonged nitric oxide-

dependent, opioid-mediated antinociceptive effect of hyperbaric oxygen in mice. J Pain, 2009; 10(2): 167-72.

- 66. Al-Waili, N.S et al., Hyperbaric oxygen in the treatment of patients with cerebral stroke, brain trauma, and neurologic disease. Advances in therapy, 2005; 22(6): 659-678.
- 67. Williams, S.T. The role of hyperbaric oxygen therapy in trauma. Trauma, 2010; 12(1): 13-20.
- 68. Efrati, S. et al., Hyperbaric oxygen therapy can diminish fibromyalgia syndrome– prospective clinical trial. PloS one, 2015; 10(5): e0127012.
- 69. Gill, A. and C.N. Bell. Hyperbaric oxygen: its uses, mechanisms of action and outcomes. Qjm, 2004; 97(7): 385-395.
- 70. Thackham, J.A. et al. The use of hyperbaric oxygen therapy to treat chronic wounds: a review. Wound Repair and Regeneration, 2008; 16(3): 321-330.
- 71. Sunkari, V.G., et al., Hyperbaric oxygen therapy activates hypoxia-inducible factor 1 (HIF-1), which contributes to improved wound healing in diabetic mice. Wound Repair and Regeneration, 2015; 23(1): 98-103.
- 72. Huang. KC et al. Hyperbaric Oxygen Therapy in Orthopedic Conditions: An Evaluation ofSafety. J Trauma, 2006; 61: 913–917.
- 73. Sugihara A, Watanabe H, Oohashi M, et al. The effect of hyperbaric oxygen therapy on thebout of treatment for soft tissue infections. J Infect, 2004; 48: 330 –333.
- 74. Sahni.T et al. Use of hyperbaric oxygen therapy in management of orthopedic disorders.Apollo Medicine, 2012 December; 9(4): 318e322.
- 75. Flaviana Soares Rocha et al. Influence of hyperbaric oxygen on the initial stages of bone healing. Oral Surg Oral Med Oral Pathol Oral Radiol, 2015 Nov; 120(5): 581-7.
- 76. Fok TC et al. Hyperbaric oxygen results in increased vascular endothelial growth factor (VEGF) protein expression in rabbit calvarial critical-sized defects. Oral Surg Oral Med OralPathol Oral Radiol Endod, 2008; 105: 417-422.
- 77. Lin SS et al. Effects of hyperbaric oxygen on the osteogenic differentiation of mesenchymalstem cells. BMC Musculoskelet Disord, 2014; 15: 56.
- 78. Wilson HD et al. Hyperbaric oxygen treatment decreases inflammation and mechanical hypersensitivity in an animal model of inflammatory pain. Brain Res., 2006; 1098: 126-128.
- 79. Zhang Q et al. Hyperbaric oxygen attenuates apoptosis and decreases inflammation in an ischemic wound model. J Invest Dermatol, 2008; 128: 2102-2112.
- 80. Kang TS et al. Effect of hyperbaric oxygen on the growth factor profile of fibroblasts.

L

ArchFacial Plast Surg., 2004; 6: 31-35.

- 81. Vecchione, R. et al. LOW PRESSURE HYPERBARIC OXYGEN THERAPY IN AUTISM SPECTRUM DISORDERS: A PROSPECTIVE, RANDOMIZED STUDY OF 30 CHILDREN.
- 82. International Journal of Current Research, 8(05): 30587-30598.
- 83. Sims NA et al. Coupling the activities of bone formation and resorption: a multitude of signals within the basic multicellular unit. BoneKEy reports, 2014; 3: 481. doi: 10.1038/bonekey.2013.215 PMID: 24466412; PubMed Central PMCID: PMC3899560.
- Kollet O et al. Osteoclasts degrade endosteal components and promote mobilization of hematopoietic progenitor cells. Nature medicine, 2006; 12(6): 657–64. doi: 10.1038/nm1417PMID: 16715089.

View publication stats

L