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# Hyperbaric Oxygen Therapy for Burn Injuries: A Case Study and Review

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**Abstract:** ***Introduction: Burns are a global health issue, causing 180,000 deaths annually, mostly in low- and middle-income countries. Non-fatal burns lead to significant morbidity, including disfigurement and functional impairment. Burn trauma triggers a systemic stress response with increased inflammation and metabolism. Hyperbaric oxygen therapy (HBOT), which delivers pure oxygen in a pressurized setting, enhances oxygen availability to tissues, aiding wound healing, reducing inflammation, and potentially minimizing hypertrophic scarring by promoting a balance in collagen synthesis. This study aims to present the potential benefits of hyperbaric oxygen therapy in treating burn injuries through a case report and literature review. Case Report: This case report describes a 33yearold male with scald burns treated with hyperbaric oxygen therapy (HBOT). Burns are a common cause of morbidity, and HBOT has shown promise in reducing inflammation, enhancing wound healing, and preventing hypertrophic scarring. The patient experienced significant improvements after five HBOT sessions, with reduced edema, better granulation tissue formation, and normalized lab results. This report also includes a literature review of HBOT use in burn treatments, emphasizing the need for further research into its therapeutic potential. Discussion: Burn injuries induce a systemic inflammatory response that delays healing and increases complications. Neutrophil activation prolongs inflammation, contributing to tissue damage and scarring. We observed improved healing, reduced inflammation, and better tissue viability with HBOT. Since 1965, HBOT has shown benefits in enhancing oxygen delivery, reducing complications, and improving recovery. Further research is needed to explore its full therapeutic potential in burns. Conclusion: This case study demonstrates the benefits of HBOT in burn care, particularly in reducing inflammation and improving healing. While the results are promising, further studies using biomarkers are necessary to explore the full potential of HBOT as a standard treatment for burn injuries.***

**Keywords:** hyperbaric oxygen therapy, burn injuries, wound healing, scald burns, inflammation

## 1. Introduction

Burns represent a significant global public health issue, accounting for an estimated 180 000 deaths annually. The majority of these occur in low- and middle-income countries and almost two thirds occur in the WHO African and South-East Asia Regions. Non-fatal burns are a leading cause of morbidity, including prolonged hospitalization, horrible disfigurement, severe functional impairment and a crippled self-esteem, often with resulting stigma and rejection.[1]

Burn trauma elicits a unique systemic stress response characterized by increased metabolism and inflammation.[2] Irreversible tissue damage can occur if there is a failure in microcirculation, and the surrounding tissue is unable to supply oxygen and nutrients to the ischemic area, causing the static zone to progress into necrosis.[3]

Neutrophil and macrophages represent pivotal early responders to burn-injured tissue.[4,5] Despite their prompt activation in response to burn-induced damage-associated molecular patterns (DAMPs), the functionality of macrophage antigen presentation and neutrophil-mediated pathogen clearance is notably compromised post-burn,[6,7] thereby heightening susceptibility to infection. While immune hyperactivation contributes to tissue injury, excessive immunosuppression exacerbates the risk of infection.

Prolonged neutrophil activation during the inflammatory phase of wound healing can significantly contribute to the formation of hypertrophic scars. In the early stages of wound healing, neutrophils are essential for clearing debris, preventing infection, and initiating the repair process through the release of pro-inflammatory cytokines such as tumor necrosis factor-alpha (TNF-α) and interleukin-1 (IL-1). However, when the inflammatory phase is extended, these cells continue to release high levels of cytokines, growth factors, and proteolytic enzymes, leading to an overproduction of extracellular matrix components like collagen.

Excessive collagen deposition, particularly type III collagen, is a hallmark of hypertrophic scarring. The persistent inflammatory environment not only disrupts the balance between collagen synthesis and degradation but also alters fibroblast activity, contributing to the formation of thick, raised scar tissue. In addition, prolonged inflammation can inhibit the transition to the proliferative and remodeling phases of healing, further exacerbating scar development.

Studies have shown that neutrophil infiltration and sustained inflammation are associated with poor wound healing outcomes and the development of fibrotic tissue, including hypertrophic scars.[9,10] Consequently, the precise modulation of immune responses plays a critical role in shaping clinical outcomes following burn injuries, therapeutic strategies aimed at regulating neutrophil activity and minimizing

prolonged inflammation may, therefore, reduce the risk of hypertrophic scarring and preventing infection.

Oxygen supply is a crucial component in the wound healing process. Oxygen is essential for ATP production through oxidative phosphorylation, making it necessary for oxygen to be adequately delivered to all metabolically active cells in the body. In conditions of hypoxia or hypoxemia, irreversible tissue damage can occur rapidly. Furthermore, oxygen plays a role not only in aerobic respiration but also as a cofactor for enzymatic processes and is a key component in cellular signaling transduction mechanisms.[13]

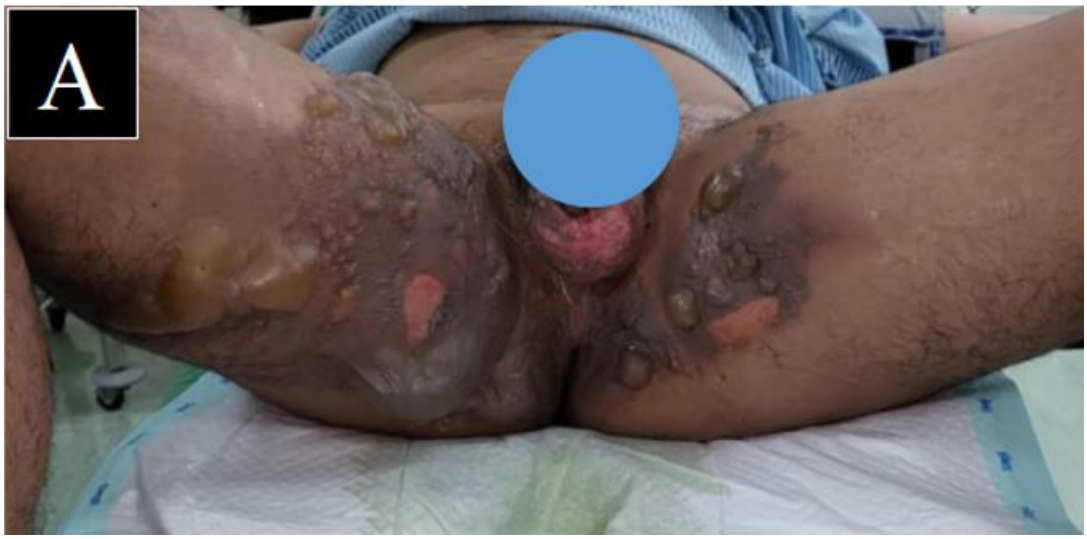
Hyperbaric oxygen therapy involves the administration of pure oxygen in a controlled, pressurized environment, significantly enhancing oxygen availability to tissues. This increase in oxygen tension is critical for various physiological processes essential for wound healing.[11] In burn patients, effective oxygenation is crucial not only for tissue repair but also for modulating the inflammatory response[12,13,14]. Studies suggest that HBOT can modulate collagen synthesis and deposition, potentially reducing the incidence and severity of hypertrophic scarring by promoting a balanced healing environment, HBOT helps to normalize the wound healing process, thereby minimizing the risk of abnormal scar formation.[15,16]

Additionally, HBOT facilitates angiogenesis, the formation of new blood vessels, which is essential for delivering nutrients and oxygen to the healing tissue. Improved angiogenesis not only accelerates healing but also enhances the aesthetic outcomes of burn injuries.

We present a case of scalds burn treated with HBOT as adjuvant therapy.

## 2. Case Report

Male patient, 33 years old, asian race, sustained epidermal to mid-dermal scald burns (hot water) to the genital area and inner thigh after accidentally spilling hot water while preparing food. The water temperature was approximately 85°C. Upon arrival at the emergency department, the patient was fully conscious, alert, and oriented, with a Glasgow Coma Score of 15 and body mass index (BMI) of 23,3. He had erythema and blistering over the affected areas (approximately 15% total body surface area), swelling and exudate noted in the genital area. His blood pressure was 120/80 mmHg), with a heart rate of 110 beats per minute, a respiratory rate of 22 breaths per minute, a body temperature of 37,7 °C, and oxygen saturation ($SpO_2$) 98-99%, with pain level 8 to 9 from 10. He was still able to respond to painful stimuli. Physical examination showed no murmurs with regular rhythm. The abdomen was soft, without tenderness or rebound. No significant past medical history. Routine laboratory examination including complete blood count, electrolytes, renal function test, coagulation profile, blood cultures were obtained. All were within normal limits, excepts for WBC which was 14,700 cells/mm³ and elevated band neutrophils in differential count. The patient underwent standard burn management, which included: primary survey, secondary survey, fluid resuscitation, wound dressing, educational control, debridement, and adjunctive hyperbaric oxygen therapy (HBOT). For HBOT procedure, the patient was evaluated for suitability first before placed in a hyperbaric chamber, which a multi-person chamber, ensuring no contraindications. The chamber was gradually pressurized while the patient breathed nearly 100% oxygen, enhancing oxygen delivery to the tissue, each session last between 60 to 120 minutes, depending on the protocol, throughout the session medical staff monitors vital signs and patient comfort, after decompression, the patient was assessed for any side effects, and follow-up care was planned. The therapy was repeated, with a total of five treatments.



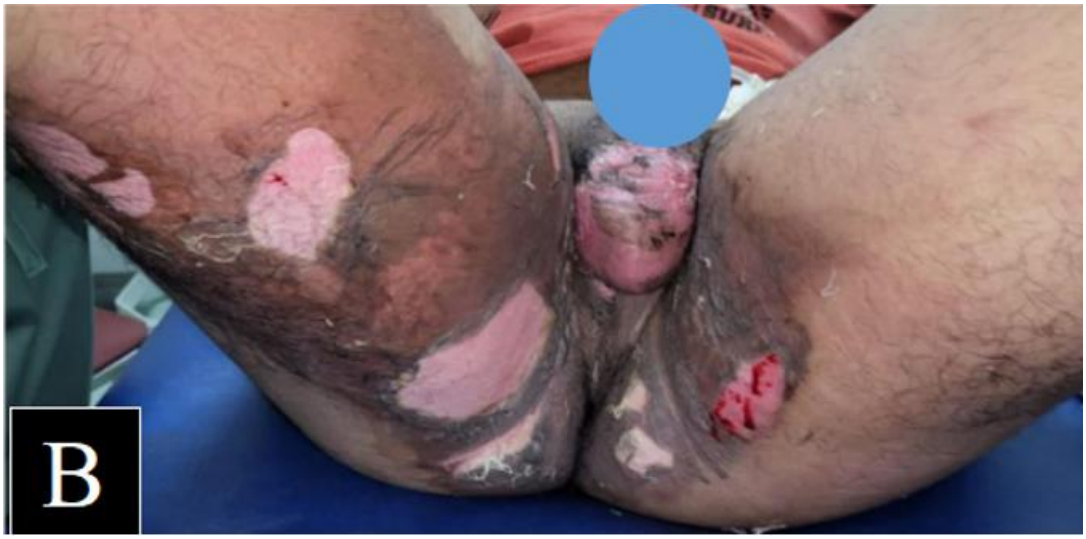
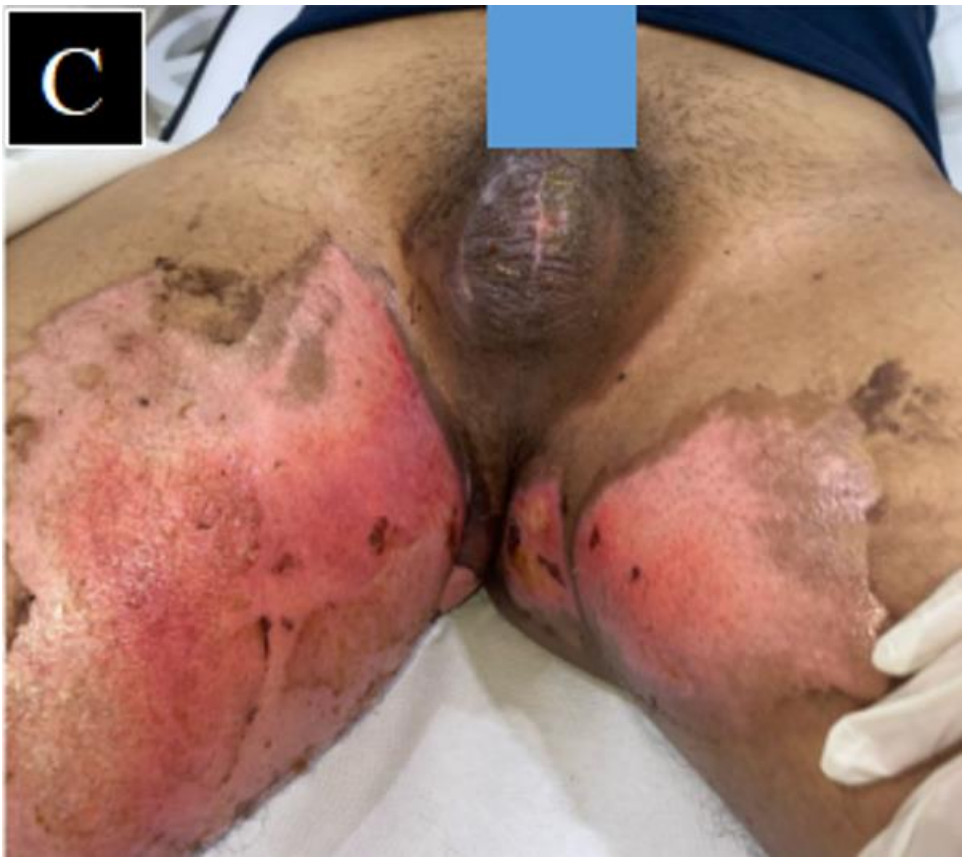



**Figure 1:** (A) The patient's condition upon arrival at the hospital after exposure to hot water. (B) The patient's condition after the first session of HBOT

As we seen on Figure 1., an epidermal to mid-dermal burn wound involves damage extending from the outermost layer of skin (epidermis) into the middle layer (dermis). Epidermal burns, often classified as first-degree burns, typically cause redness, minor swelling, and pain but heal quickly without scarring. When the damage reaches the mid-dermal layer, it becomes a second-degree burn, characterized by blisters, intense pain, and a greater risk of infection. Healing is more complex, often requiring weeks, and may result in scarring or changes in skin pigmentation. Treatment focuses on pain management, preventing infection, and promoting healing through proper wound care.



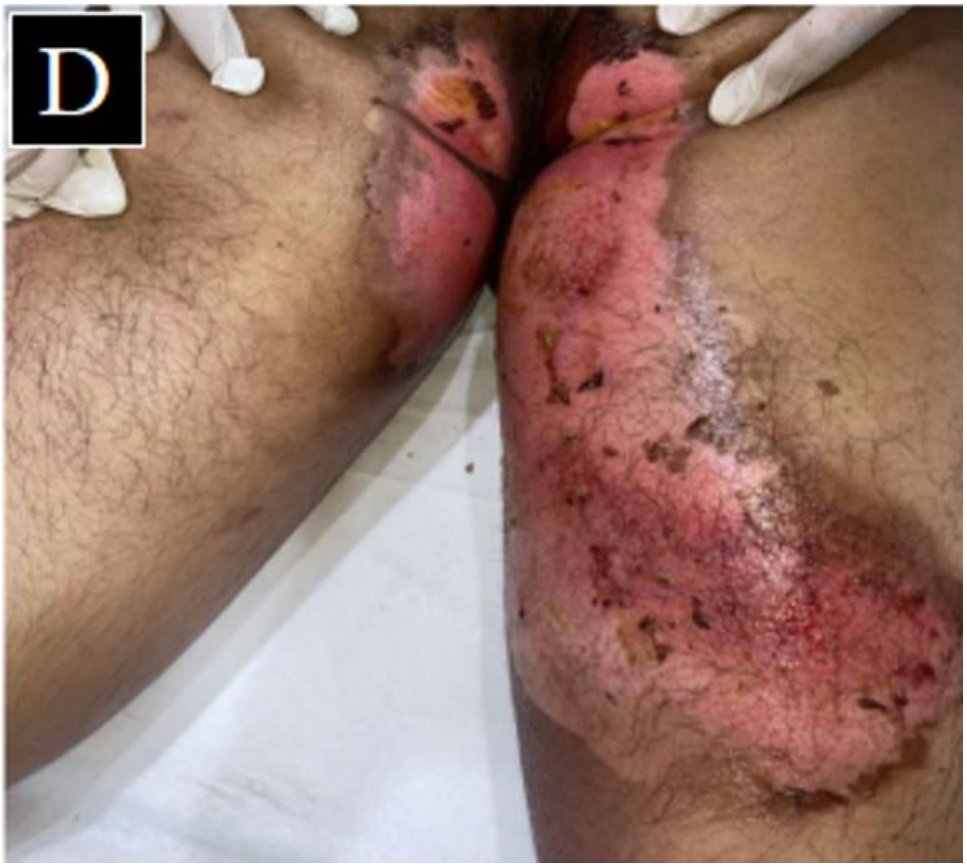
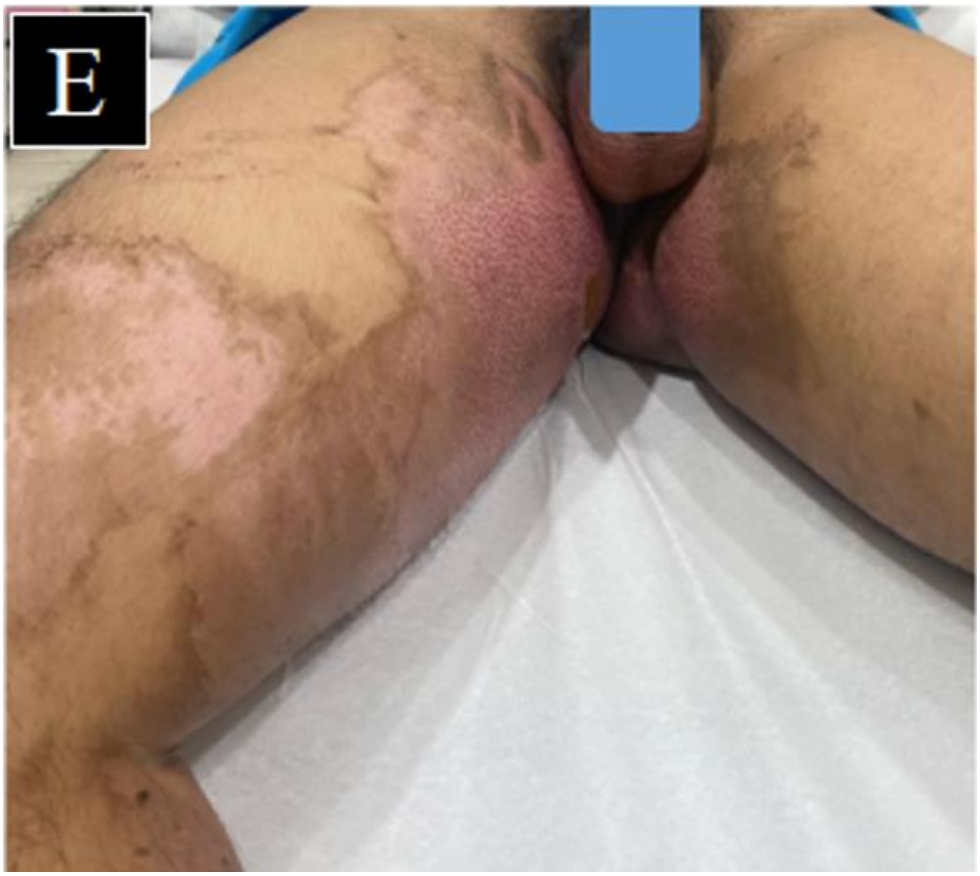



**Figure 2:** (C) The patient's burn area after the third HBOT, 9 days after being admitted to the hospital, front view image. (D) The patient's burn area after the third HBOT, back view image.



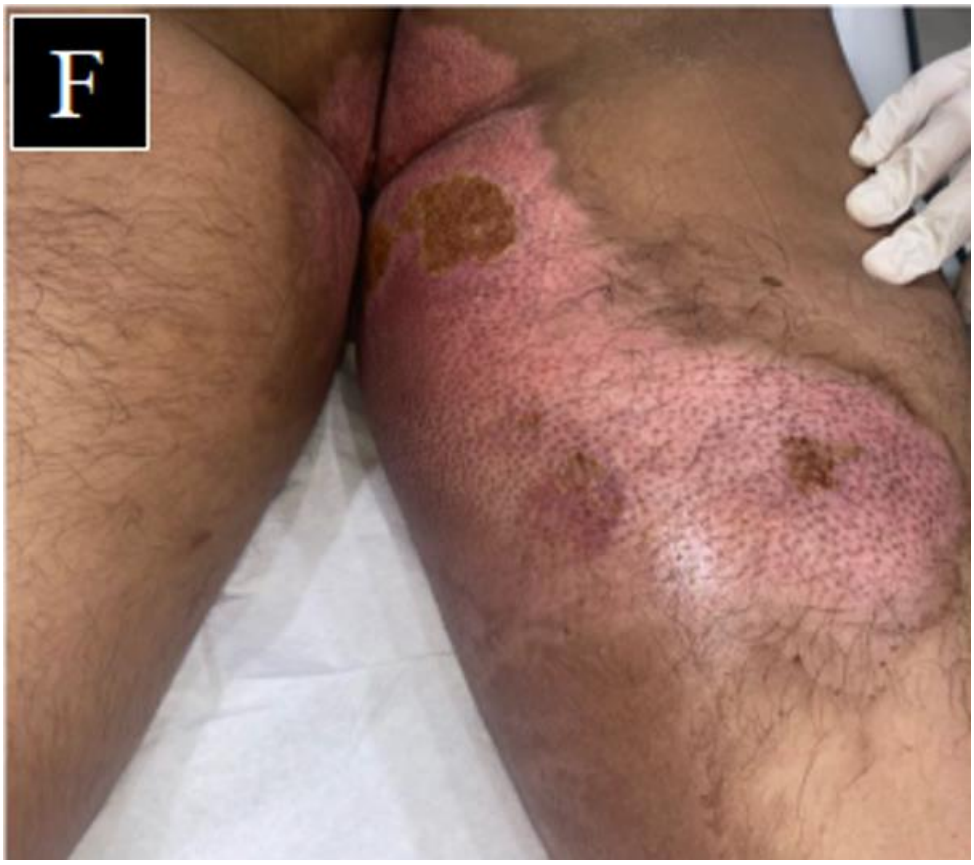



**Figure 3:** (E) The patient's burn area after the fifth session of HBOT, 18 days after being admitted to the hospital, front view image. (F) The patient's burn area after the fifth session of HBOT, back view image.

After undergoing five sessions of HBOT for his mixed epidermal to mid-dermal burn, the patient exhibited significant improvements is wound healing and overall condition. The affected burn sites showed reduced edema, accompanied by an enhanced presence of granulation tissue, indicative of improved angiogenesis and cellular proliferation. This positive change suggests that the elevated oxygen levels delivered during HBOT significantly facilitated the healing processes at the cellular level.

In parallel, laboratory evaluations revealed a substantial decrease in WBC and neutrophil to normal. Scar quality measured by POSAS (Patient and Observer Assessment Scales) indicated a reduction in pigmentation, thickness, surface texture, pliability, and discomfort, aligning with the observed clinical improvements and supporting the hypothesis that hyperbaric oxygen exposure mitigates pain through various mechanisms, including improved tissue oxygenation and reduced ischemia.

Furthermore, the synergistic effect of these physiological and biochemical changes underscores the efficacy of hyperbaric oxygen therapy as a vital adjunct in the management of mid-dermal burns, ultimately leading to more favorable healing outcomes and a lower risk of complications such as infection and hypertrophic scarring. This multifaceted approach highlights the importance of integrating advanced therapies in the treatment protocol for burn injuries, particularly in patients with significant tissue damage.

## 3. Discussion

Burn injuries trigger a distinct systemic stress reaction marked by heightened metabolism and inflammation.[17] Excessive immune activation due to burns can result in tissue damage and organ failure, while immune suppression increases the risk of infections. Neutrophil activation and the release of neutrophil-derived factors further contribute to the intricate immune reactions triggered by burn injuries. Although neutrophils play a crucial role in phagocytosing intracellular pathogens, their persistent presence can prolong the acute inflammatory phase and increase pro-inflammatory mediators (IL-1B, IL-6, TNF-α), resulting in collagen degradation and keratinocyte apoptosis.[18] Neutrophil adhesion to venular endothelium leads to microvascular disturbances and the production of oxygen-derived free radicals, causing damage to the plasma membrane, DNA cross-linking, strand breaks, and peptide fragmentation, which in turn cause ischemia and necrosis in the static zones. Neutrophil released for phagocytosis typically undergo apoptosis, but in burn wounds, neutrophil often fail to reach the injury site due to vessel occlusion and microvascular disturbances result in prolonged healing processes.[19] Experimental and clinical trials have shown delayed neutrophil apoptosis in burn cases. Recent research by Laggner et al. in 2022 found that absolute leukocyte counts and neutrophil levels in burn patients increased several days post-injury, decreased in the first week, and then rose again three weeks post-trauma.[20]

Complications from burn injuries can lead to significant health issues, particularly when they restrict a patient's

ability to perform daily tasks and potentially result in disability. Additionally, burn scars often lead to persistent sensory disturbances, pain, and itching. Tissue damage from thermal burns occurs because surrounding tissues fail to deliver necessary oxygen and nutrients to the cells near the injury.

This case report highlights the potential of HBOT as an adjunct therapy for burn injuries, which can improve healing outcomes and reduce complications such as hypertrophic scarring and infection. Before received any treatment, the patient wound appear red and swollen, with blister, typically an epidermal-mid dermal burn wounds. Laboratory test during the admission presented with elevated WBC (White Blood Cells) and neutrophil counts, alongside a temperature of 37,7$^{o}$C, suggests a potential inflammatory response or possible infection, commonly seen in burn injuries, electrolytes and renal function tests are within normal ranges, indicating stable fluid balance, normal coagulation profile suggests no coagulopathy. But after the fifth HBOT, we observed remarkably signs of reduced inflammation, improved healing, better tissue viability, pain were also reduced, mobility are function properly. We furthermore observed that changes in scar formation and appearance of the wound as it heals. The laboratory results also showed a significant improvement, with previously elevated levels returning to normal ranges.

In 1965, the renowned thoracic surgeon Juro Wada first reported the successful use of hyperbaric oxygen therapy (HBOT) in burn victims. He noted enhanced healing in burn wounds after using HBOT on survivors of a coal mine fire with carbon monoxide poisoning, leading to expectations that HBOT could revolutionize burn wound treatment. Its positive effects, such as promoting wound healing, reducing pathological edema, revascularization, and supporting the immune response, have since been confirmed by numerous experimental and clinical studies.[21,22]

In human studies, as in 1970, Lamy reported fewer infections and improved granulation and healing.[23] By 1974, Hart observed a reduction in the mean healing time[24], while Grossmann in 1978 found that HBOT reduced fluid requirements, healing time for second-degree burns, eschar separation time, donor graft harvesting time, hospital stays, complications, and mortality, along with eliminating paralytic ileus in severe burns and lowering costs.[25] Waisbern in 1982 noted a 75% reduction in the need for grafts with HBOT.[26] Niu in 1987 highlighted reduced fluid loss and earlier re-epithelialization.[27] Cianci's studies in 1989 and 1990 showed shorter hospital stays, fewer surgeries, and reduced costs.[28-30] This was especially crucial for severe burn cases, as extended hospital stays can heighten the risk of infections and other complications. Hammarlund in 1991 reported less exudation, hyperemia, and smaller wound size.[31] However, Brannen in 1997 found no significant difference in surgeries, hospital stays, or mortality between HBOT and non-HBOT groups[32], while Niezgoda the same year confirmed reductions in exudation, hyperemia, and wound size.[33] In 2013, Chong observed significantly lower rates of bacterial cultures like Staphylococcus aureus and Pseudomonas.[34] Rasmussen in 2015 discovered that HBOT attenuates central sensitization caused by thermal injury[35], and Chiang in 2017 noted faster normalization of procalcitonin serum with no limb amputations.[36] Chen in 2018 observed lower post-burn pain scores[37], and Wahl in 2019 reported a long-lasting reduction in pain sensitivity around the injured area, along with an immediate mitigating effect and a preconditioning impact on hyperalgesia.[38] Most recently, in 2020, Hatibie found that HBOT significantly reduced wound complications, hospital stays, and ICAM-1 mRNA gene expression and serum levels.[39]

Clinical management of burn targeting neutrophil function in post-burn injury might serve as an alternative treatment option in severe cases with exacerbated neutrophil activation. This is relevant because neutrophil plays a crucial role in wound healing cascade, particularly during the inflammation phase. Beneficial effects of HBOT have already been demonstrated in various experimental or clinical study and conditions, including wound healing.

## 4. Conclusion

This case study demonstrates the benefits of HBOT, including its ability to reduce burn wound complications. Burn wounds trigger an inflammatory response that involves elevated levels of neutrophil, exacerbating tissue damage if uncontrolled. Hyperbaric oxygen therapy (HBOT) has shown potential in reducing inflammation and promoting wound healing. Overall, HBOT may be an effective intervention to manage burn wounds by both reducing inflammation and improving wound appearance and texture.

More sophisticated studies using biomarkers are necessary to explore the full potential of HBOT as a standard treatment for burn injuries.

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The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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## References

[1] World Health Organization. 2023. Burn fact sheet. Accessed Sept 20, 2024. http://www.who.int/news-room/fact- sheets/detail/burn/.
[2] Guillory AN, Porter C, Suman OE, et al. 2018. Modulation of the hypermetabolic response after burn injury. Total Burn Care. 301–6.
[3] The Education Committee of The Australian and New Zealand Burn Association Limited. 2016. Emergency Management of Severe Burns (EMSB). Albany Creek: ANZBA.

[4] Lateef Z, Stuart G, Jones N, et al. 2019. The cutaneous inflammatory response to thermal burn injury in a Murine model. Int. J. Mol. Sci;20(3):538.
[5] Shallo H, Plackett TP, Heinrich SA, et al. 2003. Monocyte chemoattractant protein-1 (MCP-1) and macrophage infiltration into the skin after burn injury in aged mice. Burns;29(7):641–647.
[6] Hampson P, Dinsdale RJ, Wearn CM, et al. 2017. Neutrophil dysfunction, immature granulocytes, and cell-free DNA are early biomarkers of sepsis in burn-injured patients: A prospective observational cohort study. Ann. Surg;265(6):1241–1249.
[7] Kupper TS, Green DR, Durum SK, et al. 1985. Defective antigen presentation to a cloned T helper cell by macrophages from burned mice can be restored with interleukin-1. Surgery;98(2):199–206.
[8] Moins-Teisserenc H, Cordeiro DJ, Audigier V, et al. 2021. Severe altered immune status after burn injury is associated with bacterial infection and septic shock. Front. Immunol (12) :586195.
[9] Zhang Q, et al. 2015. Neutrophil-mediated inflammation and its role in scar formation. Journal of Wound Care, 24(9), 408–414.
[10] Wang Y, et al. 2018. The role of neutrophils in wound healing and scar formation. Nature Reviews Immunology, 18(5), 365–378.
[11] Thom SR. 2011. Hyperbaric oxygen-Its mechanisms and efficacy. Plastic and reconstructive surgery:127 (Suppl 1),131S.
[12] Harch PG, & Fogarty EF. 2012. Hyperbaric Oxygen Therapy: A Review of Its Use in the Management of Burns. Journal of Burn Care & Research, 33(3), 389-394. doi:10.1097/BCR.0b013e3182513c4e.
[13] Neuman TS, Thom SR, 2008. Physiology and Medicine of Hyperbaric Oxygen Therapy. Philadelphia: Saunders Elsevier.
[14] Shinomiya N, Asai Y. 2020. Hyperbaric Oxygenation Therapy Molecular Mechanisms and Clinical Applications. Singapore: Springer Nature Pte.
[15] Friedman T, Fitzmaurice M, et al. (2006). A randomized, controlled trial of hyperbaric oxygen therapy for the prevention of hypertrophic scars and contractures in patients with burns. Plastic and Reconstructive Surgery, 117(3), 757-765.
[16] Xu J, Sun H, et al. (2013). Hyperbaric oxygen therapy improves collagen deposition and reduces hypertrophic scarring in a rabbit ear model of burn injury.
[17] Guillory AN, Porter C, Suman OE, et al. Modulation of the hypermetabolic response after burn injury. Total Burn Care. 301–6 (2018).
[18] Kumar V, et al. 2018. Inflammation and Wound Healing In: Robbins & Cotran Pathologic Basis of Disease. Elsevier.
[19] Shupp JW, Nasabzadeh TJ, Rosenthal DS, et al. 2010. A Review of the Local Pathophysiologic Bases of Burn Wound Progression. J Burn Care Res:31(6):849-873.
[20] Laggner M, Lingitz MT, Copic D, et al. 2022. Severity of thermal burn injury is associated with systemic neutrophil activation. Sci Rep; 12: 1654.
[21] Wada J, Ikeda T, Kamata K, et al. 1965. Oxygen Hyperbaric Treatment for Carbon Monoxide Poisoning and Severe Burn in Coal Mine Gas Explosion (hokutanyubari). Igakunoaymi (Japan); 5:53.
[22] Villanueva E, et al. 2006. Hyperbaric oxygen therapy for thermal burn. In: The Cochrane Library. Oxford
[23] Lamy ML, Hanquet MM. 1970. Application Opportunity for OHP in a General Hospital—A Two Years Experience with a Monoplace Hyperbaric Oxygen Chamber. In Proceedings of the 4th International Congress on Hyperbaric Medicine Sapporo, Japan, 2–4 September 1969; Igaku Shoin, Ltd.: Tokyo, Japan.
[24] Hart GB, O'reilley RR, Broussard ND, et al. 1974. Treatment of Burns with Hyperbaric Oxygen. Surg Gynecol Obstet.; 139:693-696.
[25] Grossmann AR, Grossmann AJ. 1982. Update on hyperbaric oxygen and treatment of burns. Hyperb Oxyg Rev:3;51–59.
[26] Waisbren BA, Schutz D, Collentine G, et al. 1982. Hyperbaric oxygen in severe burns. Burns;8;176–179.
[27] Niu AKC, Yang C, Lee HC. 1987. Burns treated with adjunctive hyperbaric oxygen therapy: A comparative study in humans. J. Hyperb. Med:2;75–85.
[28] Cianci P, Lueders HW, Lee H, et al. 1988. Adjunctive hyperbaric Oxygen Reduces the Need for surgery in 40-80% burns. J Hyperb Med.: 3:97-101.
[29] Cianci P, Lueders HW, Lee H, et al. 1989. Adjunctive Hyperbaric Oxygen Therapy Reduces Length of Hospitalization in Thermal Burns. J. Burn Care Rehab., 10, 432–435.
[30] Cianci P, Williams C, Lueders HW, et al. 1990. Adjunctive Hyperbaric Oxygen in The Treatment of Thermal Burns: An Economic Analysis. J. Burn Care Rehabil.: 11, 140–143.
[31] Hammarlund C, Svedman C, Svedman P. 1991. Hyperbaric oxygen treatment of healthy volunteers with u.v.-irradiated blister wounds. Burns:17;296–301.
[32] Brannen AL, Still J, Haynes M. 1997. A Randomized Prospective trial of hyperbaric oxygen in a referral burn center population. Am. Surg. : 63, 205–208.
[33] Niezgoda JA, Cianci P, Folden BW, et al. 1997. The effect of hyperbaric oxygen therapy on a burn wound model in human volunteers. Plast. Reconstr. Surg, 99, 1620–1625.
[34] Chong SJ, Kan EM, Song C, et al. 2013. Characterization of early thermal burns and the effects of hyperbaric oxygen treatment: A pilot study. Diving Hyperb. Med. J.; 43, 157–161.
[35] Rasmussen VM, Borgen AE, Jansen EC, et al. 2015. Hyperbaric oxygen therapy attenuates central sensitization induced by a thermal injury in humans. Acta Anaesthesiol. Scand;59;749–762.
[36] Chiang IH, Chen SG, Huang KL, et al. 2017. Adjunctive hyperbaric oxygen therapy in severe burns: Experience in Taiwan Formosa Water Park dust explosion disaster. Burns;43(4):852-857.
[37] Chen KL, Wu CJ, Tseng WS. 2018. Improvement of Satisfaction in Burn Patients Receiving Adjuvant Hyperbaric Oxygen Therapy. Formos. J. Surg.: 51, 184–192.
[38] Wahl AM, Bidstrup K, Smidt-Nielsen IG, et al. 2019. A single session of hyperbaric oxygen therapy demonstrates acute and long-lasting neuroplasticity

effects in humans: A replicated, randomized controlled clinical trial. J. Pain Res, 12, 2337–2348.

[39] Hatibie MJ, et al. 2020. Effect of hyperbaric oxygen therapy on the healing of thermal burn and it's relationship with ICAM-1: a case-control study. A Medicine Surg.; 61:104-9.