ORIGINAL ARTICLE

Hyperbaric oxygen therapy in patients with thoracic injuries: Is it safe?

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ABSTRACT

BACKGROUND: This retrospective cohort study aimed to evaluate the safety of hyperbaric oxygen therapy (HBOT) in patients with blunt thoracic trauma, with particular focus on crush injuries sustained during the 2023 Kahramanmaraş earthquakes.

METHODS: Twenty-five patients with documented thoracic trauma who underwent HBOT at a tertiary care center were included. HBOT was delivered at 2.4 atmospheres absolute (ATA) for two hours per session in a multiplace chamber. Data on demographics, clinical findings, treatment outcomes, and adverse events were analyzed.

RESULTS: The median age was 23 years (range: 10-57), and 64% were female. The median number of HBOT sessions was 11 (range: 2-37). Three patients (12%) died during follow-up due to severe crush injuries, unrelated to HBOT. Five patients (20%) developed respiratory or cardiac symptoms during treatment, including dyspnea (n=2), chest pain (n=1), dyspnea with chest pain (n=1), and arrhythmia with convulsions (n=1). Pneumomediastinum was incidentally detected in one intubated patient post-session and was managed conservatively, allowing HBOT to continue without further complications. One patient experienced a generalized seizure attributed to central nervous system oxygen toxicity; HBOT was discontinued, neurological evaluation was performed, and no permanent sequelae occurred.

CONCLUSION: Although HBOT is generally considered safe, it may cause cardiopulmonary complications in patients with thoracic trauma, especially those with poor clinical reserve or requiring mechanical ventilation. Most complications observed in this cohort were minor and manageable. HBOT can be safely administered in carefully selected thoracic trauma patients when individualized risk assessment and multidisciplinary monitoring are ensured. Future prospective studies with larger cohorts are needed to further clarify safety profiles and risk stratification strategies.

Keywords: Hyperbaric oxygen therapy; thoracic trauma; blunt chest injury; pulmonary complications.

INTRODUCTION

Thoracic trauma refers to injury of the chest wall, pleural space, or intrathoracic organs caused by blunt or penetrating mechanisms and accounts for a significant proportion of trauma-related deaths.[1] Most thoracic traumas present as blunt injuries, commonly resulting from traffic accidents, falls from height, assaults, or crush injuries.[1] Between 45% and 65% of patients admitted to the emergency department with multiple trauma have an associated thoracic injury.[2]

Hyperbaric oxygen treatment (HBOT) is a therapeutic method in which 100% oxygen is administered, either intermittently or continuously, at pressures higher than 1 atmosphere

Cite this article as: Canarslan Demir K, Avcı AU, Özgök Kangal MK, Sarıyerli Dursun GB, Aydın G, Zaman T, et al. Hyperbaric oxygen therapy in patients with thoracic injuries: Is it safe? Ulus Travma Acil Cerrahi Derg 2025;31:854-859.
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Ulus Travma Acil Cerrahi Derg 2025;31(9):854-859 DOI: 10.14744/tjtes.2025.24187
Submitted: 24.06.2025 Revised: 23.07.2025 Accepted: 28.07.2025 Published: 05.09.2025

absolute (ATA) within a closed chamber.[3] It enhances oxygen diffusion into hypoxic tissue regions and is used for various clinical indications, such as early administration in crush injuries, which has been shown to reduce hypoxia-related tissue damage, necrosis, and the risk of infection.[4,5] Although HBOT is generally considered a safe treatment modality, it carries certain risks of complications. Patients at increased risk for developing pulmonary barotrauma (PBT) during HBOT include those with air-trapping lesions in the lungs, a history of spontaneous pneumothorax, underlying pulmonary disease, mechanical ventilation, or a history of thoracic trauma.[6] Existing lung injuries resulting from thoracic trauma, such as pulmonary contusion, laceration, hematoma, pseudocyst, hemothorax, and tracheobronchial injury, are particularly sensitive to pressure changes during HBOT and may increase the risk of pulmonary complications due to the vulnerability of damaged pulmonary structures.[6-8]

Although the incidence of PBT during HBOT has been reported to be very low in the literature, at approximately 4.5 cases per one million treatments,[8] complications may still occur in high-risk patient groups.[7,9-11] Published case reports on these complications have mostly involved patients treated for carbon monoxide poisoning or chronic wounds, whereas systematic data specific to the thoracic trauma population remain limited. There is currently no consensus in the literature regarding the degree of pulmonary complication risk or their optimal management strategies.

The small sample sizes, reliance on retrospective designs, and variability in patient characteristics across current studies limit the ability to draw definitive conclusions about the safety of HBOT in patients with thoracic trauma. Therefore, conducting studies to fill this gap is essential to guide the safer and more effective use of HBOT in this patient group. The aim of this study is to evaluate the safety profile of HBOT in patients with thoracic trauma and to support clinical decision-making.

MATERIALS AND METHODS

Ethical Approval

The research was carried out in alignment with the ethical guidelines of the Declaration of Helsinki and was approved by the Gulhane Ethics Board of the University of Health Sciences (05/25: 2025-50).

Design and Participants

This retrospective cohort study evaluated patients with accompanying thoracic trauma related to the 2023 Kahramanmaraş earthquake who underwent HBOT at the Department of Underwater and Hyperbaric Medicine, Gülhane Training and Research Hospital.

The study population consisted of both male and female patients, with no age restrictions applied. Patients with missing or incomplete data were excluded from the study. Data collected included age, sex, demographic characteristics, clinical indications for HBOT, and follow-up information regarding morbidity and mortality. In addition, the number of HBOT sessions administered and any complications that occurred during the treatment process were recorded.

HBOT was administered to all patients at 2.4 ATA for a duration of two hours per session in a multi-place chamber (Hipertech ZYRON 12, 2008, Istanbul, Türkiye).

Statistical Analysis

Statistical analyses were performed using Jamovi software (version 2.4.7; The Jamovi Project, Sydney, Australia). The normality of distribution for continuous variables was assessed using the Shapiro-Wilk test. Descriptive statistics were presented as mean ± standard deviation, median (minimum–maximum), or number and percentage [n (%)], as appropriate.

RESULTS

A total of 25 patients were included in the study. The median age was 23 years (range: 10-57). Of these, 16 patients (64%) were female and nine patients (36%) were male. All patients included in the study had sustained thoracic trauma during the earthquake and underwent HBOT due to indications such as crush injury and/or compartment syndrome. The median number of HBOT sessions administered was 11 (range: 2-37). At the end of the treatment process, nine patients (36%) were discharged with complete recovery, while three patients (12%) died during follow-up after the HBOT schedule ended; these deaths were not attributed to HBOT. The remaining 13 patients (52%) could not complete the full course of HBOT as initially scheduled due to poor tolerance, voluntary discontinuation, or transfer to another facility.

Of the 25 patients, two (8%) had asthma, while the remaining 23 patients (92%) had no known chronic pulmonary disease. All patients (100%) had sustained blunt thoracic trauma. The radiological findings observed in these patients are presented in Figure 1.

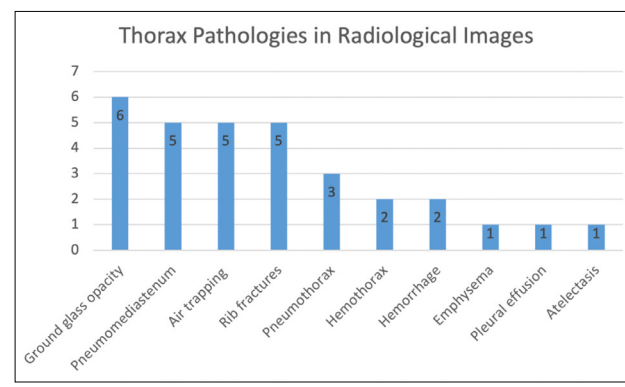

Figure 1. Thoracic pathologies identified on radiological imaging.

Pre-HBOT: Chest tube insertion was required in two patients (8%) prior to HBOT due to pneumothorax secondary to blunt thoracic trauma.

During HBOT: Adverse events were observed in a small number of patients during HBOT sessions. Dyspnea occurred in two patients (8%), chest pain in one patient (4%), both chest pain and dyspnea in one patient (4%), and one patient (4%) developed arrhythmia and convulsions. The remaining 20 patients (80%) completed the treatment without any complaints.

Post-HBOT: Pulmonary barotrauma in the form of pneumomediastinum occurred in only one patient (4%). The patient did not report any symptoms such as chest pain or dyspnea during or after HBOT sessions. Pneumomediastinum was incidentally detected on a routine thoracic computed tomography (CT) scan performed by the primary clinical team for follow-up evaluation. No additional intervention was required, and conservative management was deemed appropriate by the thoracic surgery team. Following multidisciplinary consultation, HBOT was resumed the next day under close clinical monitoring. No additional complications were observed during subsequent HBOT sessions in this patient.

DISCUSSION

In this retrospective study, the safety of HBOT was evaluated in 25 patients with blunt thoracic trauma. While some patients (n=5, 20%) experienced transient symptoms such as dyspnea, chest pain, arrhythmia, and convulsions during treatment, only one case (4%) of pneumomediastinum was recorded after HBOT. These findings indicate that strict and close monitoring is critical in patients with thoracic trauma during and after HBOT. Physicians, attending staff, and primary caregivers should remain vigilant for signs of pulmonary complications both during and after HBOT sessions.

The potential complications of HBOT arise from the physiological effects of pressure changes and high oxygen concentrations.[12] Barotrauma can develop during the pressurization and decompression phases of treatment due to significant pressure-volume fluctuations. PBT results from pulmonary overexpansion in response to a reduction in ambient pressure.[13] PBT of ascent can present in four clinical forms: local pulmonary injury, pneumomediastinum, pneumothorax, and arterial gas embolism (AGE).[14] Pulmonary pathologies that may increase the risk of barotrauma during decompression include reduced pulmonary compliance, a history of spontaneous pneumothorax, acute lower respiratory tract infection, atelectasis, bullous emphysema, pulmonary cysts, blunt chest trauma, subpleural blebs, pulmonary fibrosis, and chronic obstructive pulmonary disease with air trapping.[13] Clinicians should carefully evaluate patients for the risk of PBT prior to initiating HBOT in those with thoracic trauma. To our knowledge, there is only one case report in the literature describing the development of PBT in patients with existing thoracic trauma.[15] In our study, pneumomediastinum was observed in only one patient. Pneumomediastinum is defined as the presence of air within the mediastinal cavity and may occur spontaneously or as a result of iatrogenic factors such as trauma, mechanical ventilation, or endoscopic procedures.[16,17] Intubation and positive-pressure ventilation—especially when high pressures are applied—can lead to alveolar rupture and subsequent leakage of air into the mediastinum.[18] During HBOT, pressure fluctuations, particularly during the decompression phase, may exacerbate pre-existing air leaks or reveal subclinical pulmonary injuries.[19] The possibility of an occult or subclinical pneumothorax should always be considered, and clinicians must remain vigilant for potential complications such as tension pneumothorax.[19] Nevertheless, the literature suggests that HBOT may still be safely administered in carefully selected, asymptomatic, and hemodynamically stable cases of pneumomediastinum under close clinical observation.[20] In our case, the patient was intubated and receiving mechanical ventilation due to critical illness. The patient did not report any complaints at any stage of HBOT. However, pneumomediastinum was incidentally detected on a thoracic CT scan performed for routine monitoring by the primary clinical team. Therefore, it is difficult to definitively attribute the pneumomediastinum to either intubation or HBOT. In this clinically stable patient, HBOT was continued under close monitoring without any complications. This underscores the importance of multidisciplinary evaluation and individualized clinical decision-making in patients undergoing HBOT, particularly those with thoracic trauma or prior airway interventions.

Patients who developed dyspnea, chest pain, convulsions, or arrhythmia during HBOT were evaluated on a case-by-case basis. In our study, dyspnea was observed in two patients. In one of these, the pre-HBOT posteroanterior chest radiograph was interpreted as normal. However, dyspnea persisted throughout the entire fourth session of HBOT. A post-session thoracic CT scan revealed pericardial effusion, bilateral pleural effusions with adjacent passive atelectasis, and complete collapse of the right middle and lower lobes. This patient had undergone two surgical procedures under general anesthesia with endotracheal intubation prior to HBOT and was concurrently receiving dialysis treatment for renal failure. In the literature, atelectasis is described as one of the most frequently observed respiratory complications, particularly after upper abdominal and thoracic surgeries.[21] This clinical picture may be explained by atelectasis secondary to repeated surgical interventions and fluid accumulation in the pleural and pericardial spaces due to postoperative fluid overload and inadequate renal function. It is also possible that HBOT contributed to the aggravation of this condition, as the increased pressure within the hyperbaric environment may elevate the respiratory workload in patients with limited cardiopulmonary reserve, thereby exacerbating clinical dyspnea.[22]

In our study, another patient who developed dyspnea was evaluated in the intensive care unit with a posteroanterior chest radiograph and arterial blood gas analysis after the session. The patient had a history of multiple blood transfusions

and was diagnosed with transfusion-related acute lung injury (TRALI) based on clinical assessment. Due to progressive clinical deterioration and an increasing need for respiratory and systemic support, the patient was unable to continue HBOT sessions.

Pulmonary injuries commonly associated with blunt chest trauma include pneumothorax, hemothorax, pulmonary contusion, and tracheobronchial disruption.[23] Among these, untreated pneumothorax is considered an absolute contraindication for HBOT.[12] However, pneumothorax may not constitute a contraindication to HBOT if it is properly treated. In our study, two patients with thoracic trauma had chest tubes. One of these patients experienced chest pain during the compression phase of HBOT, while the other reported both chest pain and dyspnea during the compression phase. In the first patient, chest pain began during the compression phase of the HBOT session. Evaluation by the thoracic surgery team confirmed that the chest tube was correctly positioned and that oscillation within the tube appeared normal. However, due to recurrence of similar pain during the descent phase in subsequent sessions, it was considered that the increasing ambient pressure during HBOT may have led to a reduction in intrathoracic gas volumes in accordance with Boyle's law. Consequently, the resulting increase in negative intrapleural pressure may have caused the chest tube to come into contact with the pleura, potentially triggering the pain. Based on this clinical assessment, the patient's primary care team decided to discontinue HBOT. The other patient with a chest tube reported experiencing chest pain and dyspnea during the compression phase of all four HBOT sessions. As these symptoms persisted in subsequent sessions, they were, similar to the previous case, attributed to the presence of the chest tube. Once the patient's pneumothorax had resolved, the chest tube was removed. However, following chest tube removal, the thoracic surgery team considered further HBOT sessions to pose a high risk for barotrauma. Consequently, the patient did not continue HBOT.

In our study, another patient developed an arrhythmic episode during HBOT. Blunt thoracic trauma can affect not only pulmonary structures but also cardiovascular components, potentially influencing clinical outcomes and the safety of adjunctive therapies such as HBOT. Although often less evident, cardiac injury remains a relevant concern in such cases.[24] It may result from direct chest impact—particularly during end-diastole—compression of the heart between the sternum and vertebrae, sudden deceleration, or increased intracardiac pressure due to elevated venous return. Other contributing mechanisms include blast injuries, rib or sternal fractures, and concussive forces capable of inducing arrhythmias or even myocardial rupture.[25] In our case, the patient's thorax was compressed under debris; however, no radiological signs of cardiac injury or skeletal fracture were identified.

HBOT itself has been associated with certain cardiovascular effects. It may lead to left ventricular (LV) dysfunction or bradycardia by increasing afterload and filling pressures.[26] Therefore, optimizing cardiac function prior to HBOT is recommended, particularly in high-risk patients.[27] Furthermore, HBOT may influence cardiac conduction, likely through enhanced parasympathetic activity.[28] Although rare, arrhythmias such as extrasystoles and premature ventricular contractions have been reported without major clinical consequences.[28,29] Retrospective evaluation of this patient revealed a history of arrhythmia prior to hospitalization. It is plausible that HBOT either triggered or unmasked an underlying arrhythmogenic tendency. This case highlights the importance of comprehensive cardiovascular assessment and continuous electrocardiographic monitoring in patients with a history of blunt thoracic trauma undergoing HBOT.

One patient experienced a generalized tonic-clonic seizure during an HBOT session. Central nervous system (CNS) oxygen toxicity is a known but rare complication of HBOT.[30] This condition is thought to result from the accumulation of oxygen-derived free radicals in neural tissue, leading to neuronal hyperexcitability, which in some cases can trigger generalized tonic-clonic seizures.[31]

To mitigate this risk, air breaks were incorporated into the treatment protocol, alternating periods of oxygen breathing with short intervals of breathing ambient air. This approach aims to reduce the risk of CNS oxygen toxicity and associated convulsions. However, despite these precautions, such adverse events may still occasionally occur.

In our case, HBOT was discontinued following the seizure, and the patient was evaluated by neurology specialists. The patient had no prior history of epilepsy or seizure susceptibility, and no metabolic or structural abnormalities were identified. In the absence of alternative etiologies, the seizure was considered possibly related to central nervous system oxygen toxicity induced by HBOT. It should be noted, however, that this complication is not specific to thoracic trauma and may potentially occur in any patient receiving HBOT. This case underscores the importance of individualized risk assessment and strict adherence to treatment protocols to minimize the likelihood of such adverse events.

Furthermore, in our study, three patients died during the follow-up period after completion of the planned HBOT sessions. All three patients had sustained severe crush injuries and exhibited poor clinical status at baseline. Importantly, none of these patients experienced any HBOT-related adverse effects or clinical deterioration during the treatment sessions. Therefore, the observed mortalities were not temporally or causally linked to HBOT but were instead attributed to the underlying severity of trauma and systemic complications.

This study has several limitations. Its retrospective nature limits causal inference, and the small sample size reduces generalizability. Additionally, not all patients underwent standardized imaging or pulmonary function testing before and after HBOT, which may have led to underrecognition of subclinical complications. The presence of multiple comorbidi-

ties also made it difficult to isolate the effects of HBOT from other clinical factors.

CONCLUSION

In this study, various respiratory and cardiac symptoms occurred during HBOT in patients (n=5, 20%) with blunt thoracic trauma, and one patient (4%) developed pneumomediastinum. These findings emphasize the importance of thorough clinical evaluation and close monitoring in thoracic trauma patients during and after HBOT. Therefore, HBOT should be administered only in carefully selected post-trauma cases, guided by a multidisciplinary approach and individualized risk assessment, with patients being closely monitored throughout the treatment process.

Ethics Committee Approval: This study was approved by the Gulhane University of Health Sciences Ethics Committee (Date: 08.05.2025, Decision No: 2025/50).

Peer-review: Externally peer-reviewed.

Authorship Contributions: Concept: K.C.D, A.U.A., M.K.Ö.K., G.B.S.D., G.A., T.Z., Ş.K.; Design: K.C.D, A.U.A., M.K.Ö.K., G.B.S.D., G.A., T.Z., Ş.K.; Supervision: K.C.D, A.U.A., M.K.Ö.K., G.B.S.D., G.A., T.Z.; Resource: G.B.S., K.C.D., G.A.; Materials: G.B.S.D., K.C.D.; Data collection and/or processing: M.K.Ö.K.; Analysis and/or interpretation: K.C.D, A.U.A., G.A.; Literature review: K.C.D, A.U.A., M.K.Ö.K., Ş.K.; Writing: K.C.D, A.U.A., M.K.Ö.K., Ş.K.; Critical review: K.C.D, A.U.A., M.K.Ö.K., G.B.S.D., G.A., T.Z., Ş.K.

Conflict of Interest: None declared.

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

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ORİJİNAL ÇALIŞMA - *ÖZ*

Toraks yaralanması olan hastalarda hiperbarik oksijen tedavisi: Güvenli mi?

AMAÇ: Bu retrospektif kohort çalışmada, 2023 Kahramanmaraş depremleri sonrasında künt toraks travması geçiren hastalarda hiperbarik oksijen tedavisinin (HBOT) güvenliğini değerlendirmek amaçlanmıştır.

GEREÇ VE YÖNTEM: Çalışmaya, toraks travması tanısı alarak Gülhane Eğitim ve Araştırma Hastanesi Hiperbarik Tıp Kliniği'nde HBOT uygulanan 25 hasta alındı. HBOT, çok kişilik bir odada 2.4 ATA basınçta, günde bir kez, iki saat süreyle uygulandı. Hastaların demografik verileri, klinik endikasyonları, seans sayıları ve tedavi sırasında gelişen komplikasyonlar retrospektif olarak analiz edildi.

BULGULAR: Hastaların medyan yaşı 23 (dağılım: 10–57) olup, %64'ü kadındı. Medyan HBOT seans sayısı 11 (2–37) idi. Üç hasta (%12), HBOT tamamlandıktan sonraki takip sürecinde, altta yatan ağır crush yaralanmalarına bağlı olarak hayatını kaybetmiştir; bu ölümler HBOT ile ilişkili bulunmamıştır. Tedavi sırasında beş hastada (%20) solunum ve kardiyak semptomlar gözlenmiştir: Dispne (n=2), göğüs ağrısı (n=1), hem dispne hem ağrı (n=1), aritmi ve konvülsiyon (n=1). Bir entübe hastada seans sonrası rutin çekilen toraks BT'de tesadüfen pnömomediastinum saptanmış, hasta stabil seyretmiş ve tedaviye komplikasyonsuz şekilde devam edilmiştir. Ayrıca bir hastada seans sırasında jeneralize tonik-klonik nöbet gelişmiş, santral sinir sistemi oksijen toksisitesine bağlı olabileceği düşünülerek HBOT sonlandırılmış ve hasta nörolojik açıdan değerlendirilmiştir.

SONUÇ: HBOT, toraks travması olan hastalarda potansiyel faydalar sağlayabilecek destekleyici bir tedavi seçeneğidir. Ancak özellikle solunum rezervi kısıtlı, entübe edilen ya da ciddi eşlik eden yaralanmaları bulunan hastalarda komplikasyon riski artabilir. Bu nedenle, tedavi kararı hasta bazında dikkatle değerlendirilmelidir. HBOT'nin bu özel hasta grubunda uygulanabilirliğini artırmak için multidisipliner yaklaşım, bireyselleştirilmiş risk analizi ve yakın klinik izlem gereklidir.

Anahtar sözcükler: Hiperbarik oksijen tedavisi; toraks travmaları; künt toraks yaralanmaları; pulmoner komplikasyonlar.

Ulus Travma Acil Cerrahi Derg 2025;31(9):854-859 DOI: 10.14744/tjtes.2025.24187