Archives of Anesthesiology and Critical Care (Summer 2025); 11(3): 425-428.

Available online at http://aacc.tums.ac.ir

Case Report

Complete Resolution of Severe Decompression Sickness in a Diver Following Oxygen Therapy: A Case Report

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ARTICLE INFO

Article history:
Received 17 September 2024
Revised 08 October 2024
Accepted 22 October 2024

Keywords:
Decompression sickness;
Oxygen therapy;
Corticosteroid;
Diver

ABSTRACT

Decompression sickness (DCS) is a rare but serious risk for divers, characterized by the formation of inert gas bubbles in the bloodstream and tissues due to rapid decompression. This case report highlights a 25-year-old male recreational diver who developed type 2 DCS, presenting with neurological and pulmonary symptoms following a dive. Upon admission to the emergency department, the patient presented with severe shortness of breath, accompanied by nausea, dizziness, numbness and tingling sensations throughout the body, and joint and muscle pain. Physical examination revealed signs of respiratory distress, bilateral upper and lower limb spasticity resembling decorticate posturing, notable hypertonia, and generalized paresthesia. Despite the absence of a hyperbaric chamber facility, the patient was successfully treated with 100% normobaric oxygen (via a Jackson-Rees circuit) and adjunctive medications, including intravenous fluids, dexamethasone, fentanyl, and midazolam, resulting in rapid symptom resolution. The patient was discharged without residual symptoms and remained symptom-free at the one-month follow-up. This case underscores the importance of recognizing DCS and demonstrates that 100% normobaric oxygen therapy and corticosteroids can be effective in treating DCS when a hyperbaric chamber is unavailable.

Introduction

Decompression sickness (DCS) is one of the potential risks linked to underwater diving. The overall lifetime incidence of DCS among recreational divers is only 1 case per 5,463 dives (and as low as 1 case in 20,291 dives for severe DCS), indicating its rarity [1]. DCS occurs when inert gas bubbles are released into the bloodstream and tissues. It develops when a diver's prolonged exposure to depth leads to the supersaturation of dissolved gases, primarily nitrogen. During ascent, if the rate of decompression exceeds the body's capacity to eliminate these inert gases, bubbles form in extravascular tissues. These bubbles can mechanically compress tissues, obstruct blood vessels, and then precipitate tissue injury and organ dysfunction [2-3].

The manifestation of DCS varies depending on the extent and location of nitrogen bubbles within the body. Based on the clinical presentation and severity, DCS is classified into two types. Type 1 DCS primarily manifests as musculoskeletal, cutaneous, or lymphatic symptoms, whereas type 2 DCS is characterized by more severe systemic effects, including neurological, pulmonary, and vestibulo-auditory symptoms [3-4]. Since no specific diagnostic test exists for DCS, the diagnosis primarily relies on clinical presentation, making it crucial for physicians to recognize the signs and symptoms [5]. Moreover, hyperbaric oxygen therapy (HBOT), the definitive treatment for DCS [6], is unfortunately unavailable in many low-resource settings,

The authors declare no conflicts of interest.
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DOI: 10.18502/aacc.v11i3.18509

including our center in Wakatobi Regency. Despite this limitation, in this case report, we successfully demonstrated the resolution of DCS solely through the administration of 100% normobaric oxygen along with adjunctive medications.

Case Report

A previously healthy 25-year-old male recreational diver presented to the local emergency department (ED) with a chief complaint of shortness of breath after SCUBA diving in the ocean. The patient dove while breathing compressed air (21% oxygen) to a maximum depth of 95 feet, with a bottom time of 35 minutes. He ascended gradually from depth, following the staged decompression suggested by his dive computer, and performed a safety stop at 15 feet below the surface for three minutes. There was no history of breath-holding during ascent. The maximum dive depth, the times spent at specific depths, and the rate of ascent are illustrated in (Figure 1). Within 10 minutes after surfacing, he experienced numbness and tingling sensations in his hands and feet, which progressively spread throughout his body, accompanied by constant nausea and dizziness. Upon moving his extremities, he noted stiff bent arms and wrists, clenched fists, and extended legs with marked joint and muscle pain. He also had difficulty speaking due to facial muscle spasms. Shortly thereafter, he developed severe shortness of breath and hence was taken to a local hospital. The patient had no past history of cardiac, pulmonary, or neurological disease. He also reported diving several times in the past without any issues.

On admission, the patient was conscious and oriented, hemodynamically stable, but very dyspneic with bilateral upper and lower limb spasticity resembling decorticate posturing. His vital signs were as follows: blood pressure 130/80 mmHg, pulse rate 110 beats/minute, respiratory rate 30 breaths/minute, and oxygen saturation 81% on room air. His breath sounds were rapid, equal, and clear bilaterally. There were supraclavicular and intercostal retractions, as well as the use of accessory muscles. His heart rate was regular, and heart sounds were normal. Neurological examination was notable for hypertonia in both upper and lower extremities, along with generalized paresthesia. A complete blood count revealed an elevated white blood cell count of 15 × 10^9/L with 92.7% neutrophils and 5.4% lymphocytes, while the other results were unremarkable.

Based on the clinical manifestations and recent dive history, the patient was diagnosed with neurological and pulmonary type 2 DCS. Due to the unavailability of a hyperbaric chamber facility, he was treated supportively with 100% normobaric oxygen (via a Jackson-Rees circuit), intravenous fluids, dexamethasone, fentanyl (for musculoskeletal pain), and midazolam (for minimal sedation). Immediately afterward, his clinical status improved with the gradual resolution of his symptoms. The patient was discharged the next day with no residual neurological or pulmonary symptoms. At the one-month follow-up, the patient reported no recurring symptoms or notable residual defects.

Discussion

The case of a 25-year-old male diver presenting with severe type 2 DCS highlights several critical aspects of this rare yet potentially serious condition. The low lifetime incidence of DCS may contribute to a lack of familiarity and preparedness with the disease among healthcare professionals [1]. This case underscores the importance of recognizing and promptly treating the condition, even in settings lacking advanced therapeutic options like HBOT.

During a dive, the increased ambient pressure causes nitrogen from the breathing gas to dissolve into the body's tissues and fluids. As the diver ascends and the ambient pressure decreases, the nitrogen in the tissues becomes supersaturated because it is more soluble at higher pressures. If the ascent is too rapid, the nitrogen does not have time to be safely eliminated through the lungs and instead forms bubbles in the tissues and bloodstream. These gas bubbles can cause mechanical damage to tissues and obstruct blood flow. They can also trigger a cascade of inflammatory responses, leading to the release of inflammatory mediators that cause further damage to the affected tissue [2-3].

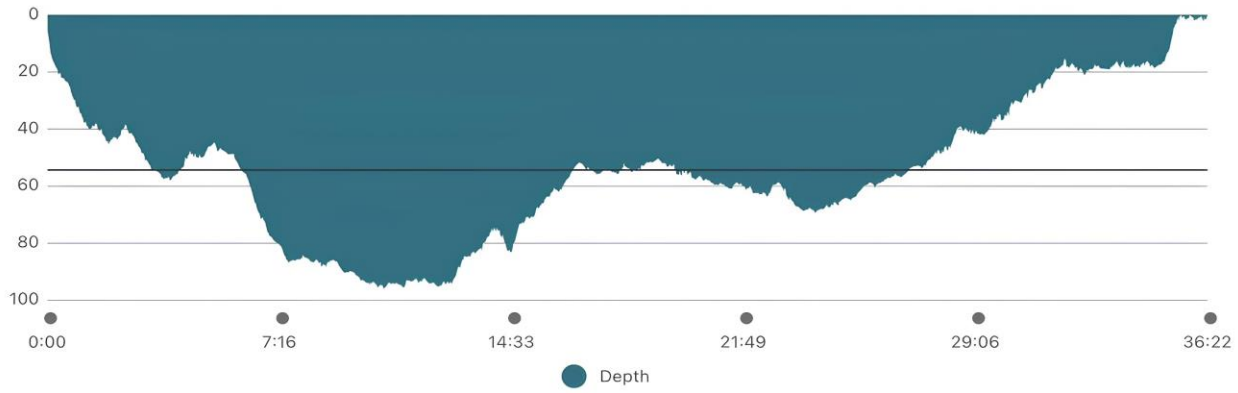

Figure 1- The patient's depth-time profile as recorded by a dive computer

Despite being very conservative with the safety protocol suggested by his dive computer, our patient still developed symptoms of DCS. This indicates that factors other than the exposure profile (depth, bottom time, and ascent rate) may contribute to an individual's susceptibility to bubble formation. These factors include workload during the dive, thermal status, medical and physical fitness, state of hydration, and the type of breathing gas mixture [3]. One such mixture is oxygen-enriched air (i.e., nitrox), which contains a higher percentage of oxygen and a correspondingly lower percentage of nitrogen than compressed air. The use of nitrox reduces the nitrogen load on the body at a given depth, thereby decreasing the risk of bubble formation [7]. Additionally, the presence of a large patent foramen ovale (PFO) may increase the risk of developing DCS, particularly neurological DCS. In individuals with a PFO, nitrogen bubbles can bypass the lungs' filtration system and enter the arterial circulation, potentially leading to embolic events in the brain and other organs. As a result of this mechanism, the risk of DCS remains high even with adherence to proper diving profiles [8]. Given that an echocardiographic evaluation was not performed, we cannot rule out the possibility of concurrent PFO in our patient.

Since there is no specific test for acute DCS, the diagnosis is purely clinical and requires a high level of suspicion, especially in individuals showing signs and symptoms consistent with DCS that appear during or after a dive [5]. DCS may exhibit diverse clinical manifestations, which vary based on the location and degree of bubble formation, and is typically classified into two types. Type 1, the most common and less severe form of DCS, presents with musculoskeletal or cutaneous symptoms, including joint pain ("the bends"), regional swelling, and skin rash. Type 2 involves more serious neurological or pulmonary complications. Neurological DCS, caused by nitrogen bubbles affecting the spinal cord and rarely the brain, often manifests as paresthesias, muscle weakness, difficulty speaking, incontinence, paralysis, spasticity, and altered mental status. When the nerves of the inner ear are affected, symptoms may include tinnitus, hearing loss, dizziness, nausea, and vomiting. Pulmonary DCS, characterized by nitrogen bubbles in the lungs that impede gas exchange, may manifest initially as a dry cough and substernal pain. This can escalate to dyspnea ("the chokes") and, in severe cases, lead to cardiovascular collapse [3-4]. Based on the temporal relation of the symptoms with the patient's recent dive, a diagnosis of type 2 DCS with neurological and pulmonary manifestations was made.

HBOT is widely regarded as the definitive treatment for DCS. By placing the patient in a hyperbaric chamber, HBOT recreates the pressurized environment of a dive, facilitating the rapid elimination of inert gas bubbles by reducing their size in accordance with Boyle's law [6]. However, in settings where a hyperbaric chamber is unavailable, alternative treatments must be considered. In this case, we administered 100% normobaric oxygen and corticosteroids, which ultimately served as the definitive treatment for our patient. A previous report on an Indian diver also demonstrated similar findings, with oxygen therapy and corticosteroids leading to clinical improvement, albeit not complete resolution [9].

High-concentration oxygen should be administered as soon as possible after the onset of symptoms, regardless of oxygen saturation levels, since it not only mitigates hypoxic damage caused by vascular occlusion from gas emboli but also enhances nitrogen elimination. By creating a steep diffusion gradient, oxygen therapy promotes the rapid washout of inert gases from tissues and the bloodstream. This can significantly reduce bubble size and mechanical stress on tissues, which is critical in alleviating the systemic effects of DCS [10]. Dexamethasone, a corticosteroid, was also administered in this case due to its potent anti-inflammatory effects. Given the inflammatory nature of DCS, the use of corticosteroids can be clinically beneficial, although their efficacy remains controversial. While there are anecdotal reports suggesting that corticosteroids may be of benefit in the treatment of DCS, particularly in its severe form [10-11], this has not been established through controlled clinical trials. Nevertheless, until further data indicate otherwise, using corticosteroids as adjunctive therapy for DCS is a reasonable recommendation.

Conclusion

This case emphasizes the importance of early recognition and prompt treatment of DCS. Due to its rarity, especially in regions where diving is uncommon, emergency physicians must maintain a high index of suspicion and include DCS in the differential diagnosis when encountering patients with symptoms that occur during or after SCUBA diving. While HBO therapy remains the mainstay of treatment for DCS, in settings where a hyperbaric chamber is unavailable, administering 100% normobaric oxygen and corticosteroids can be an effective alternative.

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