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Case Report

Persistent lower paraplegia due to decompression illness in a fisherman using a compressor for diving

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ABSTRACT

Decompression illness (DCI) is a term that encompasses injuries resulting from arterial gas embolism (AGE) and decompression sickness (DCS). It produces symptoms that range from mild musculoskeletal pain to severe neurological involvement. We report the case of a 32-year-old man who developed progressive lower limb weakness, sensory loss below T10, and bowel–bladder dysfunction shortly after a 20-minute compressor-assisted dive. Neurological examination revealed inferior paraplegia with hypoesthesia below the T10 level. MRI showed an intermedullary lesion with spinal cord edema between T8 and T11, consistent with spinal DCI. The patient underwent three sessions of recompression therapy at a secondary hospital without motor improvement and was subsequently referred to a tertiary center. After nine days of hospitalization, partial improvement was observed, though paraplegia persisted. This case highlights the challenges of managing DCI in settings with limited hyperbaric access and emphasizes the importance of early recognition, immediate recompression, and timely referral to improve neurological outcomes and reduce long-term disability in divers.

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INTRODUCTION

Decompression illness (DCI) is a term that encompasses injuries resulting from arterial gas embolism (AGE) and decompression sickness (DCS). AGE is generally caused by damage to the alveolar walls due to pulmonary barotrauma, allowing gas to enter the systemic arterial circulation. Meanwhile, DCS results from the formation of inert gas bubbles in tissue and circulation due to rapidly changing environmental pressure (Tawar & Gokulakrishnan, 2019). Clinically, DCS is divided into two types. Type I is generally characterized by musculoskeletal pain, itching, or skin lesions in the form of cutis marmorata. Type II is more serious because it involves three main organs: (1) neurological manifestations of numbness, paresthesia, or tingling, muscle weakness, impaired gait, physical coordination impairment, or bladder control dysfunction, paralysis or changes in mental status; (2) inner ear manifestations of hearing loss, vertigo, nausea, vomiting, and balance disorders; (3) cardiopulmonary involvement, also known as chokes, characterized by dry cough, retrosternal pain, dyspnea, and sometimes pink foamy sputum due to massive obstruction of the pulmonary blood vessels by gas bubbles (Mitchell, 2024).

To date, there is no specific diagnostic test that can confirm decompression sickness. However, several investigations may support the diagnosis. Chest radiography is important to exclude pneumothorax and pneumomediastinum (Cooper & Hanson, 2023) and remains crucial in the evaluation of injured divers. In cases with neurological manifestations, MRI can help to identify cerebral and spinal cord lesions (Kouki et al., 2025). The definitive therapy for DCS is hyperbaric oxygen therapy, which can reduce gas bubble size, reduce edema, and restore tissue integrity (Simon et al., 2022). When initiated promptly, hyperbaric therapy is associated with better clinical outcomes (Moon & Mitchell, 2021). This report aims to describe a case of spinal DCS with persistent paraplegia due to delayed recompression in a fisherman.

CASE REPORT

A 32-year-old man was referred with tingling in both feet, which began with an inability to move them, and loss of sensation from the tips of the feet to the navel, which had developed shortly after a single compressed-air diving session 11 days prior to admission. Afterward, the patient was unable to defecate or urinate for 11 days and was hospitalized for 6 days at a type C facility. The patient reported diving to an estimated depth of approximately 30 meters for around 30 minutes using a surface-supplied piston air compressor. Ascent to the surface was rapid and uncontrolled, and no decompression stops were performed. The patient denied repetitive dives on the same day. The patient was finally admitted to a type B facility for 4 days, where he underwent three chamber decompression procedures. However, there was no improvement in motor or sensory function. He was eventually referred to a type A hospital.

On physical and neurological examination, the patient was fully conscious with a GCS score of E4V5M6, isochoric pupils (3 mm/3 mm), as well as positive direct and consensual light reflexes. No cranial nerve deficit was found. Motor examination revealed complete paralysis of both lower extremities with a Medical Research Council (MRC) muscle strength score of 0/5 bilaterally. Sensory examination demonstrated hypoesthesia and hypoalgesia below the T10 dermatome. According to the standardized neurological assessment, the patient was classified as American Spinal Injury Association (ASIA) Impairment Scale grade A, indicating complete motor and sensory loss below the level of the lesion. Abdominal examination revealed distension,

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hypertympanic, and decreased bowel sounds.

Lumbosacral X-ray revealed paralumbar muscle spasm. Magnetic resonance imaging (MRI) of the spine revealed an intermedullary lesion at the T8 to T11 level associated with spinal cord edema, as well as minimal intervertebral disc bulging between L4 and L5. The patient was subsequently diagnosed with DCI, specifically Type II decompression sickness, otherwise known as spinal DCS. The patient's condition was also complicated by inferior paraplegia and paralytic ileus.

The patient received a total of three recompression therapy sessions during his hospitalized days at the Type B facility. The first session was conducted using the US Navy Table 6, followed by two additional sessions using US Navy Table 5. Recompression was performed at a chamber pressure equivalent to 2.8 atmospheres absolute (ATA), corresponding to a depth of 18 meters. The interval between symptom onset and the first recompression session was approximately 8 days. Despite the completion of three sessions, no significant motor improvement was observed. Recompression therapy was discontinued due to persistent paralytic ileus with inability to defecate, prompting referral to a Type A tertiary care hospital for further management.

Upon arrival at the emergency department of the referral hospital, the patient was treated with intravenous NaCl 0.9% at 20 drops/min, mecobalamin 2x500 mcg, citicoline 2x500 mg, omeprazole 2x40 mg, paracetamol 3x1 g, and ceftriaxone 2x1 g. A nasogastric tube and urinary catheter were also inserted. After 9 days of hospitalization, autonomic function showed partial recovery, with the return of bowel movements and flatus. Motor function in the lower extremities demonstrated minimal improvement, with MRC muscle strength increasing to 1/5 bilaterally. Sensory deficits and paraplegia persisted.

The patient was discharged with recommendations for regular follow-up in the neurology outpatient clinic, continued physiotherapy, and additional hyperbaric oxygen therapy sessions. After discharge, the patient did not return for follow-up visits, likely due to limited access and distance from the hospital and the island. Therefore, patient progress cannot be assessed.

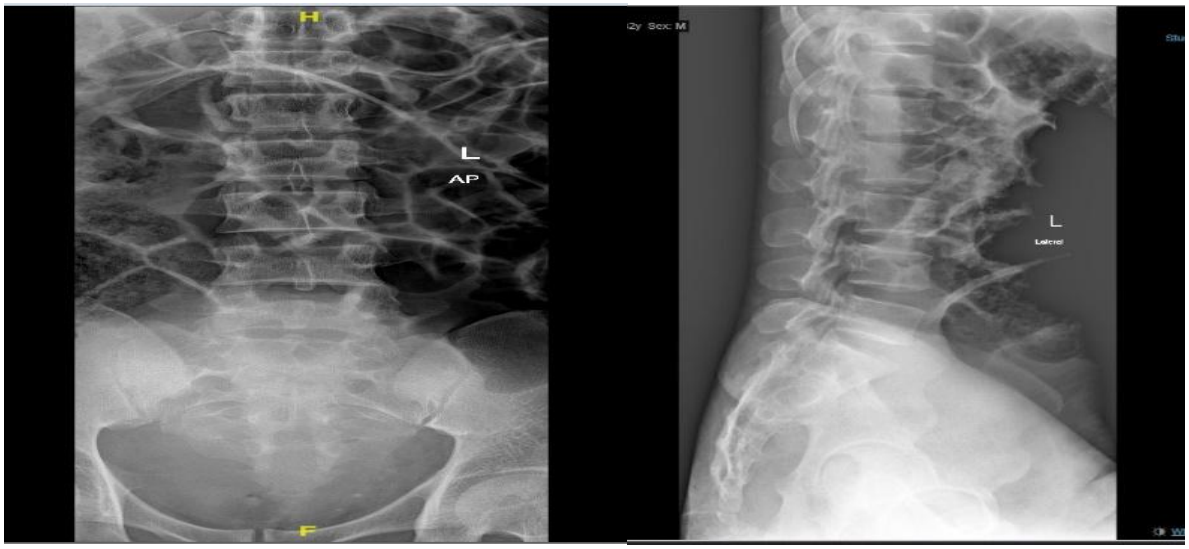

Figure 1. The patient's lumbosacral and BOF 3 position rontgen showed prominent fecal material accompanied by meteorismus and paralumbar muscle spasm.

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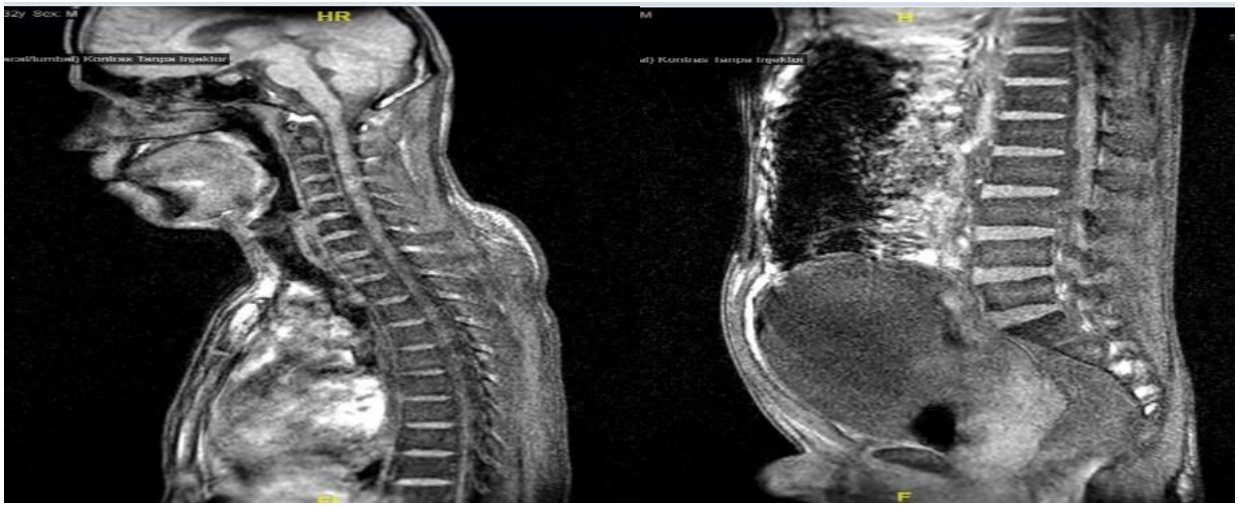

Figure 2. The patient's spine MRI showed an intramedullary lesion at T8 until T11, suggestive of spinal cord edema and minimal disc bulging at lumbar L4 and L5, without central canal stenosis or neural foraminal stenosis.

DISCUSSION

Decompression illness (DCI) is caused by the formation of bubbles primarily from inert gases, particularly nitrogen, dissolved in tissues during exposure to pressurized environments while diving. As the diver ascends to the surface, nitrogen dissolution takes time, and if the partial pressure of the gas in the tissues exceeds the ambient pressure (supersaturation), bubbles may form in the blood and tissues (Edge & Wilmshurst, 2021). In general, the incidence of DCI is relatively uncommon due to advances in diving technology and the implementation of established safety protocols. The incidence estimated among recreational divers is 3 cases per 10,000 dives, whereas commercial divers face a higher incidence, ranging from 1.5 to 10 per 10,000 dives (Cooper & Hanson, 2023). The depth, duration, and rate of ascent are closely related to the risk of DCS (Elsye et al., 2024).

Both increased deep and prolonged bottom time are strongly associated with a higher risk of DCS. Repetitive diving further amplifies this risk: divers performing 7 or more dives per day have been reported to develop DCS symptoms regardless of depth or duration. Among those diving to depths of ≥30 meters with a frequency of five or more dives per day, as many as 94.4% experienced symptoms of DCS (Cha et al., 2019). A study conducted among Korean fishermen using surface-supplied diving (SSD) reported that 93.3% of divers experienced rapid ascent to the surface. The most common cause was the sudden interruption of the air supply, accounting for 82.7% of cases. Among these divers, 71.7% developed Type II DCS (Cha et al., 2019). Gender has also been considered a risk factor for DCS. A report showed that men have a 2.5-fold higher risk compared to women (Cooper & Hanson, 2023). However, other studies have demonstrated contrasting findings, suggesting that the incidence of DCS is higher among female divers (Cha et al., 2019). Given

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these inconsistencies, further research with larger female populations is required to clarify the role of gender in DCS risk.

The mechanism of DIC involves inert gas coming out of solution and forming bubbles in tissues and blood during rapid pressure reduction. These bubbles cause primary mechanical disruption and secondary inflammatory responses, including vascular obstruction, platelet activation, and endothelial damage, resulting in tissue ischemia. Under high pressure, such as diving, nitrogen dissolves into tissues (Cooper & Hanson, 2023). If pressure decreases too quickly, the blood cannot transport the gas to the lungs fast enough, leading to bubble formation in tissues and venous blood. Bubbles act as foreign materials and mechanical obstructions, obstructing blood flow, damaging vascular endothelium, and causing extravascular tissue distortion. Bubbles trigger an inflammatory response, activating platelets, leukocytes, and the complement system. This causes endothelial dysfunction, capillary leakage, and further tissue damage (Mitchell, 2024).

The clinical manifestations of DCI are diverse and are often categorized into two types. Type I is generally considered mild, characterized by musculoskeletal pain and cutaneous symptoms, whereas Type II is more severe, involving neurological and pulmonary complications. Neurological symptoms arise due to the formation of intravascular gas bubbles, which can obstruct venous outflow and subsequently lead to ischemia (Tetzlaff & Eichhorn, 2025). In this case, the patient developed lower-limb weakness immediately after diving, which persisted until presentation to the healthcare facility on day seven. The clinical features included paraparesis progressing to paraplegia, as well as bowel and bladder dysfunction, all of which are consistent with Type II DCS, particularly the spinal form (Edge & Wilmshurst, 2021). Literature reports indicate that paraplegia or quadriplegia occurs in 20 to 25% of cases, paresthesia in 20 30%, bladder dysfunction in 1 to 5%, and abdominal pain in 1 to 5% (Mitchell, 2024).

Diagnosis of DCI relies on thorough clinical examination and detailed medical history, including dive profiles and the temporal relationship between decompression and symptom onset (Tetzlaff & Eichhorn, 2025). Post-recompression, evaluation would include a complete blood count to rule out haemoconcentration and chest radiography to detect pneumothorax (Mitchell et al., 2022). Among the imaging modalities available to study neurologic DCI, MRI appears to be the most accurate for detecting pathological changes in the brain and spinal cord (Kouki et al., 2025). Immediate recompression therapy is most effective when initiated within minutes of symptom onset (Edge & Wilmshurst, 2021). The cornerstone of DCI management is hyperbaric therapy, which mitigates symptoms by reducing bubble size and increasing tissue oxygenation (Moon & Mitchell, 2021). The Divers Alert Network (DAN) recommends continuing treatment for persistent neurological deficits until no meaningful improvement is observed after two consecutive sessions (Pollock & Buteau, 2017).

In Indonesia, hyperbaric oxygen therapy (HBOT) facilities are available in several referral hospitals; however, the distribution remains concentrated in major cities or in regions with specific demands, such as the military and the oil and gas industry. Most hyperbaric chambers are in Java, Bali, and other large urban centers outside Java, while availability in the eastern archipelago, including Maluku and Nusa Tenggara, is still very limited. This disparity often results in significant delays for traditional coastal divers in accessing timely hyperbaric treatment.

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Delays in recompression significantly reduce the likelihood of complete recovery (Edge & Wilmshurst, 2021). Delayed recompression is associated with residual symptoms. One study reported that patients who received recompression more than 48 hours after symptom onset had residual neurologic deficits at day 30 of follow-up (Wilson et al., 2023). When recompression is delayed, the obstructing bubble triggers ischemia and an ischemia/reperfusion-related inflammatory cascade. This is characterized by the migration of neutrophils into the ischemic tissue, which release vasoactive compounds, proteases, and free radicals, further exacerbating tissue damage (Sokolowski et al., 2022). Persistent paraplegia despite repetitive recompression sessions indicates that poor neurological recovery is a consequence of delayed treatment in this patient.

Adjunctive therapies, such as nonsteroidal anti-inflammatory drugs or corticosteroids, remain a subject of debate; however, early diagnosis and prompt recompression continue to be the cornerstones of successful management (Saadi et al., 2019). The limitations of this study include delayed presentation to healthcare and a short follow-up duration. These limitations may affect diagnostic precision and clinical interpretation. Written informed consent for publication of this case report and accompanying clinical data was obtained from the patient.

CONCLUSION

This case illustrates Type II decompression sickness (spinal DCS) with severe neurological complications that persisted despite three sessions of recompression therapy due to the patient's delayed arrival at a health facility. This case demonstrates the importance of educating the public, especially fishermen, about safe diving practices and access to hyperbaric facilities. Prompt diagnosis and treatment can reduce disability rates.

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