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A Case of spinal cord decompression sickness: Diagnostic dilemmas and barriers to hyperbaric therapy in Indonesia

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Case Report

ABSTRACT

The majority of dives go off without any problems, but there are physiological changes brought on by pressure fluctuations in the underwater environment that might cause serious harm. Decompression sickness (DCS) is a condition resulting from the formation of nitrogen bubbles in body tissues due to rapid decompression after a dive, and one of the severe manifestations of DCS is spinal cord DCS. Although it is uncommon, it is a neurological diving emergency that can cause permanent impairment. The gold standard treatment for all DCS, including spinal cord DCS, is hyperbaric oxygen (HBO) therapy. In this article, we present a case report of a 43-year-old male with spinal cord DCS with a high MEDSUBHYP score, but who refused HBO therapy, which led to long-term morbidity and residual neurological deficits. The patient had received non-steroidal anti-inflammatory drugs (NSAIDs) and methylprednisolone as adjunctive therapy, but they provided no benefit. This article discusses the clinical presentation of spinal cord DCS, its challenging diagnosis, and several factors that predicted poor prognosis in patients with spinal cord DCS. We also highlight the logistical barrier to getting HBO therapy in Indonesia, which may be one of the main reasons the patient refused to be referred to another facility.

INTRODUCTION

Over the past few decades, diving has gained its popularity globally, not only for recreational diving but also for commercial and scientific diving, which have increased annually, including in Indonesia. Indonesia is one of the world's largest archipelagic countries and is known for its rich marine biodiversity. Indonesia has also been named 'The World's Best Scuba Dive Destination' by the UK's DIVE Magazine for three consecutive years from 2017 to 2019.[1,2] The vast majority of dives are completed without incident. However, there are physiological changes and several injuries related to pressure changes in underwater environments. These conditions may occur on descent, at depth, or on ascent. One of the most commonly known is Decompression Sickness (DCS).[2–5]

Decompression sickness is a condition resulting from the formation of nitrogen bubbles in body tissues due to rapid decompression after a dive. These bubbles come from dissolved inert gas when the diver rapidly ascends from depth, and then the bubbles formed cause both mechanical and ischemic damage to tissues. DCS is categorized into 'Type I DCS,' which is also called 'pain-only' and 'mild' and involves the skin, joints, and lymphatic system only, and 'Type II DCS,' which is more 'severe' and 'serious' DCS involving the central nervous system (brain and spinal cord), vestibular (or 'staggers'), and cardiopulmonary symptoms ('chokes').[2,4,6] The other condition like Arterial Gas Embolism (AGE) is a condition where bubbles introduced into arterial

circulation lead into multifocal ischemia; this condition is sometimes called 'Type III DCS', but because it is almost impossible to differentiate all of these terms and the treatment protocols for all disorders are the same, Decompression Illness (DCI) is used as a collective term for these diving disorders.[7]

The prevalence of DCS is quite rare due to technological improvement and the establishment of diving safety protocols, but since diving activity is increasing, the number of DCS cases is increasing, and in severe cases, it may lead to permanent disability. The overall incidence of DCS is around 0.015% for scientific divers, 0.01-0.19% for recreational divers, 0.03% for US Navy divers, and for commercial divers, around 0.095% or up to 10 cases per 10,000 dives.[2,5,6]

Among all DCS cases, the incidence of spinal cord DCS is rare, and it can occur even in experienced divers. It is a neurological emergency with potential for long-term disability (around 26.9% suffered permanent residual disability). Several factors are well known to be related to spinal cord DCS prognosis, with early treatment with hyperbaric oxygen (HBO) being associated with a better prognosis, making prompt recognition critical. The challenging part is 'diagnosis of spinal cord DCS is a pure clinical diagnosis', as neither laboratory nor imaging studies are helpful.[8]

In Indonesia, despite the advancements in diving safety and the well-known impact of HBO therapy in spinal cord DCS, access to HBO facilities in Indonesia remains a significant challenge, contributing to poorer outcomes. In this article, we present a case report of a 43-year-old male with spinal cord DCS and then highlight several factors that may affect its management and prognosis, including the impact of delayed or refused HBO therapy on long-term neurological function.

CASE DESCRIPTION

A 43-year-old male, a commercial crab and lobster diver, presented to our emergency department (ED) with bilateral leg weakness, inability to urinate, and a constant, dull pain encircling his upper abdomen, all of which had persisted for the past 30 hours. On the day of the symptom onset, he performed multiple Self-Contained Underwater Breathing Apparatus (SCUBA) dives to a depth of 30 meters of seawater (msw) for approximately one hour – nearly twice as long as his usual duration – while following the recommended decompression stops. The symptoms began approximately 5 to 10 minutes after he resurfaced on the boat. He did not seek immediate medical attention, assuming the symptoms would resolve with rest, as he had never experienced any diving-related symptoms in nearly 15 years of diving experience. When the condition failed to improve, he presented to our ED 30 hours after symptom onset. He had no prior medical history and denied any trauma, shortness of breath, tinnitus, fever, cough, diarrhea, or other systemic symptoms.

On admission to our ED, which was almost 30 hours after the onset of symptoms occurred in exact, his vital signs were all normal, but physical examination showed upper motor neuron (UMN) type paraparesis with motor strength 1 out of 5 and para-hypesthesia at the umbilical level. No abnormality was found in cranial nerve examination. Urinary retention was confirmed as almost 1.5 L of urine came out after Foley catheter placement, and further testing revealed decreased tone of the anal sphincter as well. We suspected a diagnosis of type II Spinal Cord DCS and gave high-flow oxygen with 15 liters of oxygen per minute using a non-rebreather mask. Fluids were given to achieve a urine output of at least 1 mL/kg/min. Laboratory studies revealed hemoglobin level was 15.6 g/dL with hematocrit 46.6%, a slight increase in white blood cells to 11.77 x 103/L, and normal electrolyte levels with no abnormalities found in liver and kidney function. Several studies to exclude differential diagnoses were performed, and all (chest and spine X-ray) returned normal.

The patient's MEDSUBHYP score was calculated: he received 1 point each for age and back pain, 3 points for a stable clinical condition, 4 points each for paraparesis and sensory deficits, and 6 points for bladder dysfunction, giving a total score of 19. A score greater than 7 is associated with increased risk of poor outcomes.

Thoraco-lumbar Magnetic Resonance Imaging (MRI) with contrast was scheduled for the next day to exclude any other diagnosis, and we explained all the possible options to the patient,

including the suspected diagnosis of spinal cord DCS, the prognosis, and the necessity of recompression therapy. Unfortunately, there is no hyperbaric oxygen (HBO) chamber in our hospital, so we educated him and his family to refer to a nearby hospital with an HBO facility. The patient rejected the option and preferred to do an MRI. Conservative management was initiated with intravenous ketorolac (30 mg three times daily) for pain control, along with high-dose intravenous methylprednisolone (250 mg four times daily) to reduce potential spinal cord inflammation, while awaiting the MRI results.

The next day, MRI revealed signs of spinal cord ischemia with hyperintense lesions (T1/T2 WI) of the posterior horn of the thoracic cord at the level of T1-T3 (figures 1 and 2), further increasing our suspicion of a spinal cord DCS diagnosis. We re-educated the patient and his family for hyperbaric therapy, but again, the patient denied the option due to the nearby hospital with the HBO facility being 92 km away from our hospital and 166 km away from the patient's home.

Management was continued with symptomatic treatment using intravenous ketorolac (30 mg as needed) for pain control. Intravenous methylprednisolone was discontinued after MRI findings revealed no signs of spinal cord inflammation. A physiotherapy consultation was initiated on the second day of admission. Physiotherapy sessions were conducted twice daily and included icing to relieve the pain, log-rolling and turning exercises to prevent pressure ulcers, pelvic exercises, and bladder training for pelvic strengthening and urinary control, as well as transcutaneous electrical nerve stimulation (TENS) combined with neuromuscular electrical stimulation (NMES).

The patient was discharged after five days of hospitalization with minimal motor improvement, achieving a muscle strength of 2 out of 5 in the lower limbs. During the first three weeks of follow-up, he continued his rehabilitation twice weekly and began to regain strength in both legs, although urinary catheterization was still required. The Foley catheter was successfully removed at the fourth week. At the latest follow-up, two months after symptom onset, he remained moderately disabled and was still unable to walk independently.

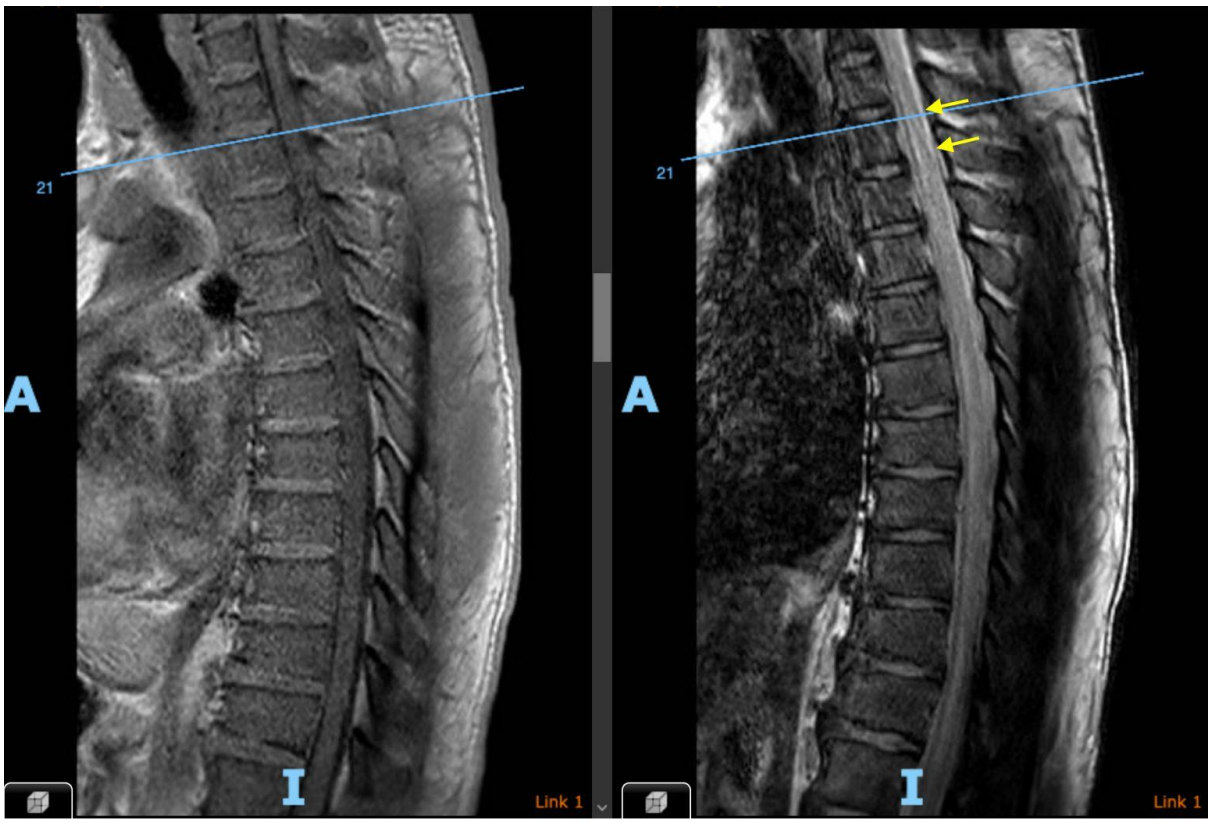

Figure 1. Sagittal view - MRI revealed signs of spinal cord ischemia in the thoracic cord T1-T3 with hyperintense lesions of the posterior horn of the thoracic cord at the level of T1-T3 (yellow arrow)

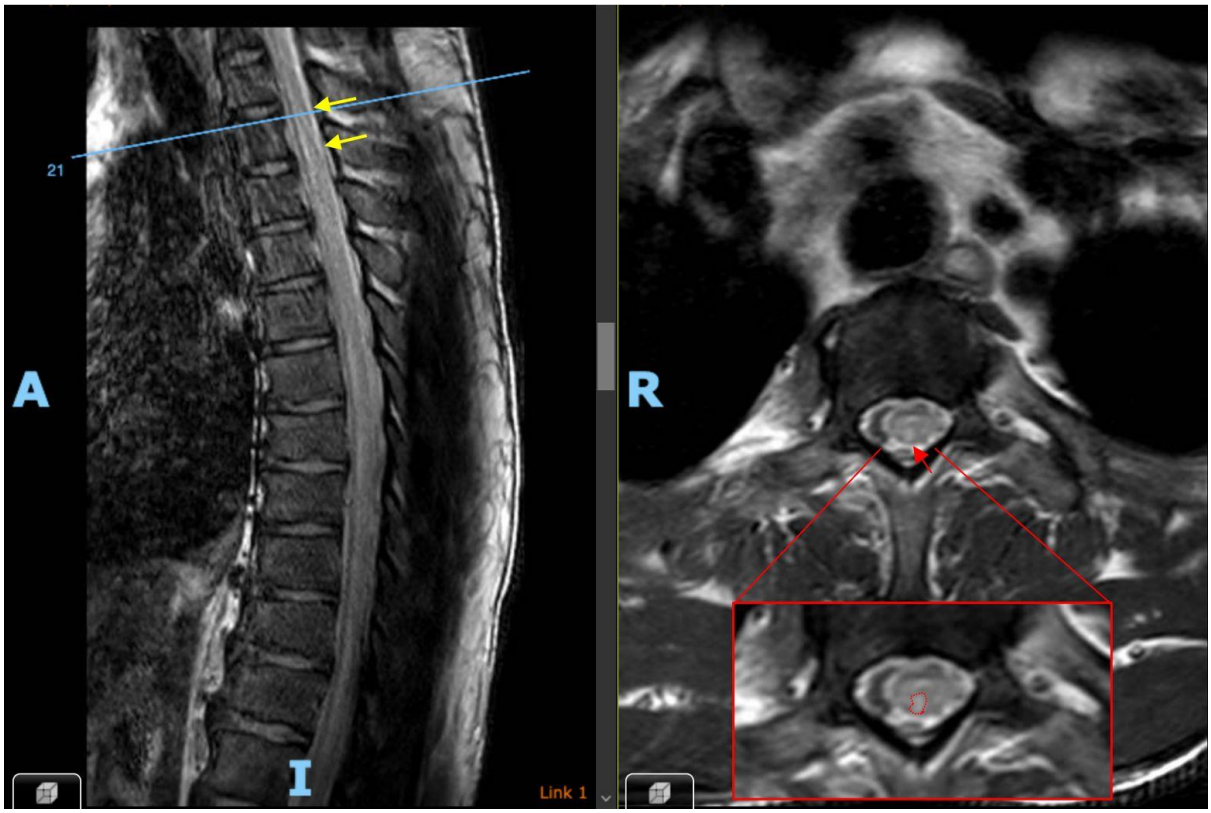

Figure 2. MRI T2WI showed hyper-intense lesion in posterior horn thoracic cord; left image: sagittal view (yellow arrow), right image: axial view (red arrow, dashed red area)

DISCUSSION

Decompression Sickness (DCS) is a condition caused by the formation of nitrogen gas bubbles in body tissues due to rapid decompression. These bubbles can cause both mechanical and ischemic damage, with the spinal cord being one of the most commonly affected areas in Type II DCS.[2,4,9] This case report describes a diver who developed spinal cord DCS, highlighting the diagnostic challenges, management strategies, and several factors associated with a poor prognosis. The specific vulnerability of the thoracic spinal cord to DCS is attributed to its unique vascular supply, particularly the presence of watershed areas with limited collateral circulation.[10,11] The blood supply in this region primarily relies on the anterior spinal artery (ASA) and radiculomedullary arteries, which have limited anastomotic connections. This makes the area less capable of compensating for ischemic events caused by vascular occlusion from gas bubbles.[12] Consequently, ischemic lesions frequently manifest in the posterior horn of the spinal cord, a region that is particularly susceptible due to its distal vascular positioning and reduce blood flow during episodes of hypoperfusion.[12] These mechanical effects, combined with the inflammatory response to bubble-induced tissue damage, result in both vascular occlusion and neural injury.[6–8]

The patient's neurological symptoms, which appeared just 5-10 minutes after resurfacing, align with the typical presentation of severe spinal cord DCS. This short latency period is often correlated with a higher degree of symptom severity.[7,8] Clinical manifestations such as UMN-type paraparesis, a circumferential band of paresthesia around the chest and abdomen, and urinary retention are all characteristic of spinal cord involvement in DCS.[4,6,7,13] While acute neurological conditions like transverse myelitis and non-traumatic spinal cord infarction can present similarly, the patient's clear history of a recent SCUBA dive makes DCS the most probable diagnosis.[14–16] Diagnosis of spinal cord DCS is fundamentally a **clinical one**, relying heavily on a recent diving history and neurological examination.[7,17] Imaging, including MRI, is primarily used to rule out alternative diagnoses. Although MRI findings in DCS can be non-specific or even normal, the presence of hyperintense lesions on T2-weighted images (T2-WI) in the posterior

horn of the thoracic spinal cord (T1-T3), as seen in our patient, strongly supported the diagnosis. These findings are consistent with ischemic injury and may be associated with a poorer outcome.[6,18]

Regarding management, the gold standard treatment for DCS is hyperbaric oxygen therapy (HBO), which works by reducing the size of nitrogen bubbles, improving oxygen delivery to ischemic tissues, and promoting nitrogen washout.[4–7] Unfortunately, the patient in this case is unable to access HBO therapy immediately, highlighting a significant challenge in the management of DCS. Furthermore, while HBO remains the cornerstone of treatment, several drugs have been proposed as adjunctive therapies, but none are definitively evidence-based and usually only on the basis of theoretical attraction or the results of *in vivo* experiments.[6,19] The use of high-dose methylprednisolone is considered in this case, based on its theoretical neuroprotective effects against inflammation and ischemia, similar to its application in acute spinal cord injuries.[20,21] It is important to emphasize that such adjunctive therapies should never be a substitute for timely HBO therapy.

The patient's inability to receive HBO therapy immediately due to the distance to the nearest facility underscores a major systemic issue in Indonesia. Access to HBO facilities remains a significant challenge for divers, particularly those in remote locations. The limited number of operational HBO facilities in the country and their scattered distribution, often concentrated in major urban centers and high-traffic diving areas like Bali, Komodo, and Raja Ampat, create a substantial geographical disparity.[5,22–24] This insufficiency is a critical concern for diver safety, as delays in receiving HBO therapy are directly linked to poorer neurological outcomes and increased morbidity.[25,26] Research suggests that while recreational diving tourism is expanding rapidly, the healthcare infrastructure, specifically the availability of HBO facilities, has not kept pace with this growth.[24] This case serves as a poignant example of how these logistical barriers can severely impact the management and final outcome of a spinal cord DCS case, highlighting the urgent need for a more comprehensive and strategically planned healthcare network to support the growing diving community in Indonesia.

In conclusion, the prognosis for spinal cord DCS is dependent on several key factors: a short latency period, a delay in diagnosis and management, the severity of the initial injury (assessed using the MEDSUBHYP scoring scale), the presence of a pathological ischemic lesion on MRI, and the availability of HBO therapy.[6,7,27–29] The severity of the initial injury in this case was assessed using the MEDSUBHYP scoring scale, as detailed in Table 1, with the specific calculation of the patient's score presented in Table 2 which resulted in a high score of 19, indicating a high probability of morbidity. Our patient exhibited almost all of the factors associated with a poor prognosis: a very short latency period (5-10 minutes), a significant delay in seeking medical attention (nearly 30 hours), and the presence of a pathological ischemic lesion on MRI. These combined factors, along with the inability to undergo immediate HBO therapy due to logistical barriers, are likely responsible for the moderate degree of disability, including persistent difficulty walking, that the patient experienced even after 2 months of recovery and rehabilitation.[6,7]

Table 1. The MEDSUBHYP Scoring Scale

Variable	Option	Score
Age >42 y.o	*No*	0
	Yes	1
Back pain	*No*	0
	Yes	1
Clinical course before recompression	*Better*	0
	Stable	2
	Worse	5
Objective sensory deficit	*No*	0
	Yes	4
Motor Impairment	*No*	0

Variable	Option	Score
	Paresis	4
	Paraplegia	5
Bladder dysfunction	*No*	0
	Yes	6

Table 2. Calculation of the MEDSUBHYP Score for the Patient

Variable	Patient Condition	Assigned Score
Age >42 y.o	43 years old	1
Back pain	Yes	1
Clinical course before recompression	Stable for 30 hours	2
Objective sensory deficit	Yes (Parahypesthesia)	4
Motor Impairment	Paresis	4
Bladder dysfunction	Yes	6
Total Score		19

CONCLUSION

This case emphasizes the importance of early diagnosis and timely treatment of spinal cord DCS. Potential delays from initial onset of symptoms until diagnosis and any potential problems in between have been discussed, including the availability of hyperbaric oxygen therapy, which remains the gold standard for treatment in spinal cord DCS. Timely access to HBO therapy remains crucial for improving recovery and minimizing the long-term impact of spinal cord DCS. In Indonesia, the low number of HBO therapy facilities and logistical barriers can also delay initiation of HBO therapy and lead to suboptimal outcomes. Conservative management with NSAIDs and corticosteroids provides some symptomatic relief, but the lack of HBO therapy likely contributes to the persistent neurological deficits.

CONFLICT OF INTEREST

The authors declare that they have no competing interest. This case report was conducted without any external funding or support. The authors have no financial or personal relationships that could influence the outcome of this report.

PATIENT CONSENT STATEMENT

Written informed consent was obtained from the patient for the publication of this case report, including all accompanying images and clinical information. The patient understood that personal identifiers would not be disclosed and that all efforts would be made to maintain confidentiality. Proof of consent has been provided to the editorial office.

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DATA AVAILABILITY STATEMENT

All data supporting the findings of this study are available within the article. Further details

can be obtained from the corresponding author upon reasonable request.

SUPPLEMENTARY MATERIAL(S)

No supplementary materials were included in this manuscript.

AUTHORS CONTRIBUTIONS

SEA contributed to the conception and design of the study, as well as the clinical management and manuscript preparation. DT contributed to the clinical assessment, literature review, and revision of the manuscript. CIA was responsible for coordinating the case documentation, manuscript drafting, and served as the corresponding author. RWP contributed to data collection, imaging analysis, and formatting of the manuscript. All authors have read and approved the final version of the manuscript.

DECLARATION OF USING AI IN THE WRITING PROCESS

The authors used artificial intelligence (AI) tools to assist in the literature search and reference organization related to this case report. However, the interpretation, critical analysis, and writing of the manuscript were entirely performed by the authors. No AI tool was used to generate or draft any part of the final text

LIST OF ABBREVIATIONS

DCS: Decompression Sickness, MRI: Magnetic Resonance Imaging, ICU: Intensive Care Unit, CT: Computed Tomography, T2WI: T2-Weighted Imaging, T1WI: T1-Weighted Imaging, GCS: Glasgow Coma Scale, HBOT: Hyperbaric Oxygen Therapy

REFERENCES

1. Kusubandio W. Cleanliness, health, safety, and environmental sustainability guidance for indonesia's dive industry. 2020.
2. Fitriasari E, Komang N, Dewi Untari S, Fitra NA. Risk factors for decompression sickness. JMI. 2024;3(2):3806–18. DOI: 10.58344/jmi.v3i2.1095.
3. Walls RM, Hockberger R, Gausche-Hill M, Erickson TB, Wilcox SR. Rosen's emergency medicine: concepts and clinical practice. In: Ron M. Walls, Marianne Gausche-Hill, Timothy Bruce Erickson, Susan Renee Wilcox, editors. 10th ed. Elsevier; 2022. p. 1773–8.
4. Sadler CA, Snyder BK. Diving disorders. In: Tintinalli JE, Ma OJ, Yealy DM, Meckler GD, Stapczynski JS, Cline DM, et al., editors. Tintinalli's Emergency Medicine: A Comprehensive Study Guide, 9e. New York, NY: McGraw-Hill Education; 2020. p. 1368.
5. Dewi NW, Sutanegara KDP, Praramdana MN, Al Kasiron R, Zulkarnaen DA. Decompression sickness type 1 in traditional fishermen: A literature review. Biocity J Pharm Biosci Clin Community. 2023;1(2):81–8. DOI:10.30.812/biocity.v1i2.2523.
6. Mitchell SJ. Decompression illness: a comprehensive overview. Diving and Hyperbaric Medicine Journal. 2024;54(1(Suppl)):1–53. DOI:10.28920/dhm54.1.suppl.1-53.
7. Mitchell SJ, Bennet MH, Moon RE. Decompression sickness and arterial gas embolism. N Engl J Med. 2022;386(13):1254–64. DOI:10.1056/nejmra2116554.
8. Denoble PJ, Marroni A. Differential diagnosis of decompression illness workshop proceedings. In: Denoble PJ, Marroni A, editors. Durham, NC: Divers Alert Network; 2019.
9. Cooper JS, Hanson KC. Decompression sickness. In: Kenneth C. Hanson, Jeffrey S. Cooper, editors. StatPearls - NCBI Bookshelf. Treasure Island, Florida: StatPearls Publishing; 2023.
10. Izumi R, Hayashi K, Nakaya Y, Suzuki A, Takaku N, Sato M, et al. A case of spinal cord infarction with pansensory deficit: Discussing the possible etiology. Cureus. 2024;16:71880. DOI:10.7759/cureus.71880.
11. Ullah MW, Qaseem A, Amray AN. Post vaccination guillain barre syndrome: A case report. Cureus. 2018;10:e2511. DOI:10.7759/cureus.2511.
12. Maženský D, Flešárova S, Šulla I. Arterial blood supply to the spinal cord in animal models of spinal cord injury. a review. The Anatomical Record. 2017;300(12):2091–106.

DOI:10.1002/ar.23694.
13. Edge CJ, Wilmshurst PT. The pathophysiologies of diving diseases. BJA education. 2021;21(9):343-8. DOI:10.1016/j.bjae.2021.05.003.
14. Noh MSFM, Bahari N, Rashid AMA. Tuberculous myelopathy associated with longitudinally extensive lesion: A clinicoradiological review of reported cases. J Clin Neurol. 2020;16(3):369. DOI:10.3988/jcn.2020.16.3.369.
15. Ali L, Muhammad A, Ahmed I, Haroon KH. Acute spinal cord ischemic stroke; a rare but potentially devastating stroke emergency in young healthy adults. J Clin Images Med Case Rep. 2021;2(5). doi:10.52768/2766-7820/1375.
16. Ayub-Ferreira SM, Fonseca A, Correia F, Cunha J, Taveira M. Anterior spinal cord infarction: A rare diagnosis with an uncommon presentation. Cureus. 2024;16:e64083. DOI:10.7759/cureus.64083.
17. Saadi A, Ferenczi E, Reda H. Spinal decompression sickness in an experienced scuba diver: A case report and review of literature. The Neurohospitalist. 2019;9(4):235–8. DOI:10.1177/1941874419828895.
18. Bilgin YÖÜ, Köşkderelioğlu A, Bilgin RR. Decompression sickness with simultaneous cranial and spinal involvement: a case report. Turk Noroloji Dergisi. 2021;27(3):330–3. DOI:10.4274/TND.2021.05914.
19. Kot J, Lenkiewicz E, Lizak E, Goralczyk P, Chreptowicz U. Spinal cord decompression sickness in an inside attendant after a standard hyperbaric oxygen treatment session. Diving and Hyperbaric Medicine. 2021;51(1):103–6. DOI:10.28920/dhm51.1.103-106.
20. Tan VH, Chin K, Kumar A, Chng J, Soh CR. Treatment preferences for decompression illness amongst singapore dive physicians. Diving Hyperb Med. 2017;47(2):118–22. DOI:10.28920/dhm47.2.118-122.
21. Prakash M, Hariharan R, Chandy BR. Spinal cord injury in decompression sickness: a case report. Indian J Phys Med Rehabil. 2014;25(1):18–21. DOI:10.5005/ijopmr-25-1-18.
22. Hisnindarsyah H, Usemahu SN, Mainase J. Respon pasien dengan decompression sickness tipe i terhadap pemberian terapi oksigen hiperbarik di RSAL dr.F.X Suhardjo tahun 2016. Molucca Medica. 2018;28–34. DOI:10.30598/molmed.2018.v11.i2.28.
23. Maharja R, Ikhsan N. Faktor yang berhubungan dengan gejala penyakit dekompresi pada nelayan penyelam ikan factors associated with symptoms of decompressed disease among fish diving fisherman. Journal of Industrial Hygiene and Occupational Health. 2023;7(2). DOI: 10.21111/jihoh.v7i2.
24. Mitchell SJ, Bennett MH, Bryson P, Butler FK, Doolette DJ, Holm JR, et al. Pre hospital management of decompression illness: Expert review of key principles and controversies. Diving Hyperb Med. 2018;48(1):45–55. DOI:10.28920/dhm48.1.45-55.
25. Ahn J, Kim H. Recompression of spinal cord decompression sickness. Journal of the Neurological Sciences. 2021;429. DOI:10.1016/j.jns.2021.118243.
26. Howle LE, Weber PW, Hada EA, Vann RD, Denoble PJ. The probability and severity of decompression sickness. PLoS ONE. 2017;12(3). DOI:10.1371/journal.pone.0172665.
27. Simonnet B, Roffi R, Lehot H, Morin J, Druelle A, Daubresse L, et al. Therapeutic management of severe spinal cord decompression sickness in a hyperbaric center. Frontiers in Medicine. 2023;10. DOI:10.3389/fmed.2023.1172646.
28. Druelle A, Blatteau JE, Daubresse Duchadeuil L, Morin J, Roffi R, Dufresne PL, et al. Case report: reassessing guidelines for safe resumption of diving after spinal decompression sickness: Insights from a challenging case. Frontiers in Medicine. 2024;11. DOI:10.3389/fmed.2024.1347465.
29. Blatteau JE, Lambrechts K, Ruffez J. Factors influencing the severity of long-term sequelae in fisherman-divers with neurological decompression sickness. Diving and Hyperbaric Medicine. 2020;50:9–16. DOI:10.28920/dhm50.1.9-16.