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Spinal Decompression Sickness in an Experienced Scuba Diver: A Case Report and Review of Literature

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Abstract

Decompression sickness from diving is a rare but potentially reversible cause of spinal injury. Early treatment with hyperbaric oxygen is associated with a better neurologic outcome, making prompt recognition and management clinically important. We describe a case of a 65-year-old diver who presented with thoracic back pain and bilateral leg weakness after a 70 feet of sea water (fsw) (21 meters of sea water [msw]) dive, with no acute abnormality on spinal magnetic resonance imaging (MRI). He made a partial recovery after extended hyperbaric oxygen therapy. We discuss the epidemiology and pathophysiology of central nervous system injury in decompression sickness, as well as acute management and prognostic factors for recovery, including the role of adjunctive therapies and the implications of negative MRI. Ultimately, clinicians should make the diagnosis of spinal cord decompression sickness based primarily on clinical evaluation, not on MRI findings.

Keywords

decompression sickness, myelopathy, hyperbaric oxygen, MRI, steroids, NSAIDs

Introduction

Spinal cord injury from decompression sickness is a neurological emergency that should be treated early with hyperbaric oxygen.[1] The exact pathophysiology is not fully understood. Proposed mechanisms include venous occlusion, direct tissue disruption by nitrogen bubbles,[2,3] and subsequent activation of inflammatory cascades.[4,5] This case report highlights 2 controversial themes in the management of spinal decompression sickness: first, the role of adjunctive medications such as steroids and non-steroidal anti-inflammatory agents,[1,6,7] and second, the implications of a negative magnetic resonance imaging (MRI) scan for the ability to predict functional outcome.[8]

Case Report

A 65-year-old experienced scuba diver was diving for scallops. He descended to 70 fsw (21 msw) and began to ascend after 35 minutes when his dive computer signaled that he had 7 minutes of no-decompression time remaining. The diving conditions were described as mild currents. He performed a routine safety stop (usually done at a depth of 15 fsw [4.6 msw]) below the surface for 2 minutes and arrived at the surface feeling normal. Stepping onto the boat, he experienced acute-onset burning paresthesia in the midthoracic region of his back that radiated to both arms, around his chest in a band-like fashion, and subsequently to his anterior thighs and legs. Upon moving his lower extremities, he noted bilateral leg weakness with difficulty walking. He was taken to a local emergency department approximately 2 hours from the onset of symptoms where he was placed on 100% normobaric oxygen therapy. Although his back pain improved, his leg symptoms persisted. He was transferred to our facility for hyperbaric therapy and neurologic evaluation.

The patient's past medical history was notable for hypertension. Neurologic examination revealed flaccid weakness of his legs bilaterally (MRC grade 4/5) and reduced sensation to all modalities in the legs without a sensory level, although he reported a circumferential band of paresthesia in the T4

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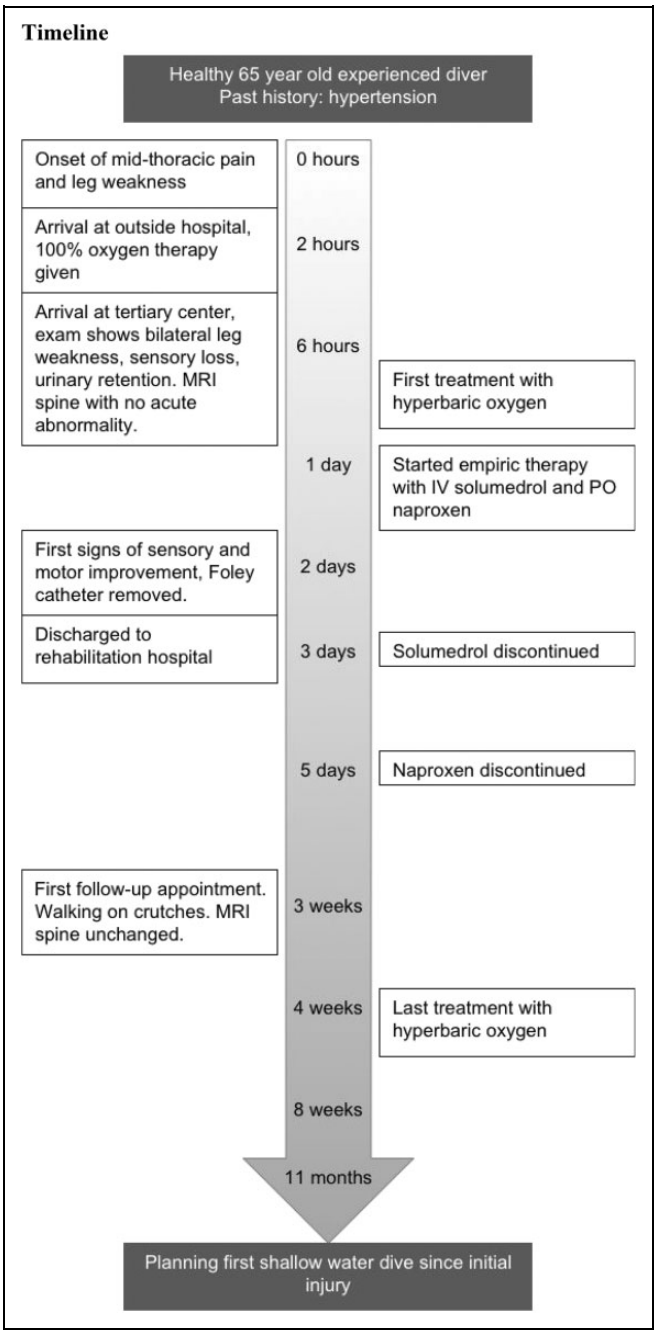
dermatome. His reflexes were normal and plantar responses were mute. He was unable to stand without assistance. A Foley catheter was placed for urinary retention. A 1.5 T MRI scan of the brain and spine (including diffusion-weighted imaging and apparent diffusion coefficient sequences) revealed no acute abnormality or sign of trauma, no intrinsic cord signal abnormalities, and only mild degenerative changes of the spine (Figure 1A). Transthoracic echocardiography excluded an acute cardiac etiology for his thoracic pain and did not demonstrate a patent foramen ovale.

Hyperbaric therapy was started approximately 6 hours after symptom onset, per the protocol of U.S. Navy Treatment Table 6 (U.S. Navy Diving Manual, Revision 6). According to this protocol, he was compressed to 60 fsw within 5 minutes, followed by 3 cycles of 20-minute treatments at 60 fsw with 5-minute air breaks. This was followed by slow (over 30 minutes) decompression to 30 fsw for 2 cycles of 60-minute treatments with 15-minute air breaks, followed by slow return (over 30 minutes) to surface pressure (total treatment time of 4 hours and 50 minutes).

In response to patient preference, the patient received methylprednisolone 250 mg intravenously daily for 3 days as well as naproxen 375 mg orally 3 times daily for 5 days. He underwent a total of 20 hyperbaric chamber treatments over 1 month (2.0 atmospheres absolute for 120 minutes with one 10-minute air break). He noted the first sign of sensory and motor improvement after 3 treatments. The urinary catheter was removed at that time. He was discharged to a rehabilitation hospital.

At his 3-week follow-up, he was walking with crutches. Repeat MRI (3 T) of the cervical and thoracic spine at that time again showed no intrinsic cord signal abnormalities (Figure 1B). At his 2-month follow-up, he was ambulating without a gait aid. He could feel and control micturition. At 11 months, he was planning his first shallow water dive since the injury.



Discussion

Decompression sickness (DCS) is caused when inert nitrogen gas bubbles come out of solution in the bloodstream and tissues after ascent from diving. The increase in environmental pressure during the descent phase of a dive causes more nitrogen to become dissolved in the body tissues, proportional to the environmental pressure, until the tissues become saturated. During the ascent phase, if environmental pressure changes rapidly (ie, if the diver does not ascend slowly enough), the dissolved nitrogen gas cannot be eliminated via diffusion and perfusion processes and the nitrogen starts to come out of solution, forming nitrogen bubbles within the tissues and the bloodstream. The likelihood of nitrogen bubbles forming depends not only on the rate of ascent but also on the dive profile (depth and duration). The "no decompression limit" (NDL) is the amount of time divers can spend underwater without needing to pause during the ascent and is determined by a range of factors including the dive depth, number of dives in a day, and the gas mixture used. Dive tables and dive computers help divers calculate and adjust their NDL for each dive to reduce the risk of DCS. Many divers will make a "safety stop" just below the surface to reduce the risk of DCS; however, remaining within the NDL is the most important way to prevent DCS. The overall incidence of DCS is 0.32 to 35.3/10 000 person-dives.[9]

Symptoms in type I DCS are mild, including constitutional symptoms such as fatigue or malaise, or more specific symptoms involving muscles, skin, and joints. Type II DCS is more severe, affecting the inner ear and central nervous system. The spinal cord is the most commonly affected site in the nervous

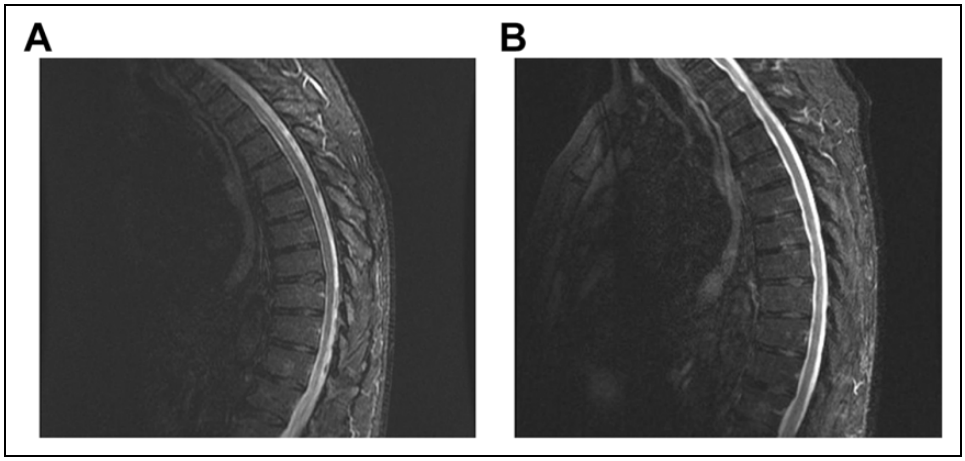


Figure 1. A, 1.5 T magnetic resonance imaging (MRI) thoracic spine short tau inversion recovery (STIR) sequence on admission, (B) 3 T MRI thoracic spine STIR sequence at 3 weeks.

system, with the thoracic spinal cord particularly at risk due to its vascular anatomy and fat content.[10] Patients typically develop symptoms within 1 hour of ascent. Spinal involvement can manifest as a myelopathy, as seen in this case, whereas cerebral involvement can present as encephalopathy.

The exact mechanism by which nitrogen bubbles cause brain and spinal cord damage is not fully determined. Proposed mechanisms include direct mechanical obstruction or compression within tissue parenchyma or blood vessels. In the brain, DCS is thought to predominantly involve the arterial circulation, whereas in the spinal cord, injury is thought to involve obstruction and congestion of venous drainage in addition to direct injury from bubble formation within the cord itself.[3] Oxidative stress as well as excitatory amino acid and cytokine release with activation of inflammatory pathways are also theorized to play a role due to direct nitrogen toxicity to the endothelium.[4,5]

Acute management of DCS involves first aid oxygen therapy followed by hyperbaric (recompression) therapy. Hyperbaric therapy reduces the size and volume of bubbles, allowing easier reabsorption and dissipation of nitrogen, and increases delivery of oxygen to ischemic tissue. Patients with DCS-associated spinal cord injury (SCI) have a more favorable prognosis than those with SCI due to other causes.[11] According to the Divers Alert Network (DAN), treatment with hyperbaric therapy should be continued for persistent neurological symptoms until no significant improvement occurs on two consecutive treatments. Delayed treatment may be inversely related to complete resolution of symptoms.[1]

Given that inflammatory pathophysiology may contribute to spinal injury in decompression sickness, adjunctive therapy with anti-inflammatory agents has been explored, but evidence remains weak. There has only been one randomized trial investigating the role of adjunctive non-steroidal anti-inflammatory agents in DCS.[12] This study demonstrated that a 1-week course of tenoxicam versus placebo produced no significant difference in eventual neurologic outcome but did result in a significant reduction in the number of hyperbaric treatments required, without any adverse complications.[12] The efficacy of steroids in the treatment of DCS is even more controversial, with only anecdotal reports of benefit.[13] In a Cochrane meta-analysis of 8 trials of steroids for acute spinal cord injury (resulting from a range of underlying etiologies), a modest benefit of high-dose methylprednisolone was demonstrated when administered within the first 8 hours of injuy.[6] However, there has been no randomized controlled trial to support the use of steroids specifically for spinal DCS, and their use has been associated with greater risk of adverse events.[1] Steroids are therefore not routinely recommended,[14] but given the absence of clear evidence either in favor or against, patient preference can play a role in the shared decision-making process between providers and patients[7] (as in this case, where the patient was well-versed in the emergency therapeutic options for DCS).

MRI may demonstrate lesions of the brain and spinal cord suggestive of ischemia and edema. When present, spinal cord lesions are frequently dorsolateral, T2-hyperintense, and associated with clinically-apparent neurologic deficits. However, in 40% to 70% of clinical type II spinal DCS, MRI may be normal[8,15-17] and this may predict better clinical recovery.[8,14] There are several potential technical reasons why the MRI may be normal in DCS. These include the use of lower field strength MRI (1.5 T or less), which may have lower sensitivity than 3 T MRI for detecting small spinal cord lesions.[18] Motion artifact (especially if patients are in pain) also poses a challenge for imaging small regions such as the spinal cord. Since DCS is a clinical diagnosis, imaging may be appropriately delayed for delivery of hyperbaric therapy, potentially reducing the window for detection of acute spinal cord signal change. The timely use of specific imaging sequences for acute injury detection, such as diffusion-weighted and diffusion-tensor imaging may help to improve MRI sensitivity

for DCS, but these sequences are not used routinely in clinical practice.[10]

Several additional independent risk factors and clinical features have been associated with poorer recovery from DCS, including older age, diving depth greater than 128 fsw (39 msw), presence of bladder dysfunction at presentation, pre-existing cervical or thoracic vertebral degenerative disease,[19] and persistent or worsening clinical symptoms prior to recompression therapy[11]—all of which were present in this case. Risk of recurrent DCS is greater for experienced divers who continue to dive without adjusting their diving technique and patients with a patent foramen ovale with right-to-left shunt.[20]

Conclusions

Myelopathy from decompression sickness can occur even in experienced divers. Early treatment with hyperbaric therapy helps to improve functional outcome, making early clinical diagnosis crucial. Adjunctive treatment with nonsteroidal anti-inflammatory agents and steroids remains controversial. Magnetic resonance imaging of the spinal cord may be negative, but this does not always portend early or complete neurologic recovery.

Authors' Note

Altaf Saadi and Emily A. Ferenczi authors contributed equally. Patient gave informed consent for this case report.

Declaration of Conflicting Interests

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