

## Case reports

### MRI features of spinal cord decompression sickness presenting as a Brown-Sequard syndrome

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#### Abstract

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Decompression sickness often manifests as central nervous system impairment. We report a 49-year-old woman who developed an unusual case of spinal cord decompression sickness presenting as complete Brown-Sequard syndrome. Initial MRI revealed increased signal intensity in the left side of the cervical cord at the level of C2–C3. A second MRI at 10 days post-injury showed signal abnormalities corresponding to an infarction in the posterior spinal artery territory. After two weeks of intensive treatment with various HBOT regimens, the clinical outcome was still poor, but at six months after the injury her neurological condition was greatly improved, with only slight impairment of proprioception on the left when walking remaining.

#### Key words

Decompression sickness, decompression illness, central nervous system, radiological imaging, case reports

#### Introduction

Decompression sickness (DCS) is an acute disorder caused by the development, during decompression, of bubbles formed by inert gas (usually nitrogen) previously dissolved in the tissues. The spinal cord is frequently involved and the clinical presentation varies according to the affected site.<sup>1–3</sup> Neurological symptoms may vary considerably from subjective sensory to complete motor deficiency with sphincter dysfunction. The pathophysiological mechanisms of spinal cord DCS have not yet been fully identified; several hypotheses have been raised, such as venous infarction generated by bubbles, development of autochthonous bubbles within nervous tissue of the spinal cord or embolisation of arterial bubbles.<sup>4</sup> The first hypothesis is the best documented, since the alteration of spinal cord venous drainage that results from the obstruction of the epidural venous system has been shown experimentally.<sup>5</sup> Secondary immuno-inflammatory processes on endothelial activation, as well as the start of blood platelet aggregation and coagulation, would contribute to worsening of the phenomenon.<sup>4</sup> In the present report, we describe a severe case of spinal cord DCS presenting as a Brown-Sequard syndrome with MRI findings suggestive of an occlusion of the posterior spinal artery.

#### Case report

A 49-year-old female, experienced recreational diver was referred to our hyperbaric facility with motor weakness affecting the left lower limb that developed after an uneventful scuba dive to a maximum depth of 55 metres' sea water (msw) for 20 minutes, without violation of the

decompression procedure given by her dive computer. The day before, she had performed two repetitive scuba dives to a maximum depth of 50 msw. The patient was on medication for hypertension and was a non-smoker.

Close questioning revealed that, after the dive in question, she felt faint and had difficulty climbing into the boat. This was followed by an episode of vomiting. One hour later, she complained of transient cervical pain with tingling sensations in her left arm accompanied by muscle weakness gradually developing in the ipsilateral lower limb. She was placed on normobaric oxygen and transferred by helicopter for clinical evaluation and initiation of recompression therapy.

At presentation, her level of consciousness and cognition and cranial nerves were normal. Neurological examination revealed incomplete paralysis of the left lower limb for L2–L5 myotomes (scored 3/5 according to the American Spinal Injury Association) with impaired proprioception at the same level. Paraesthesiae in her left arm persisted without objective sensory signs. Pin-prick and temperature sensations were altered on the right side below the T5 level. The left Babinski reflex was positive while knee and ankle deep tendons reflexes were increased but symmetrical. The remainder of her physical examination was normal.

Four hours after surfacing, she underwent a COMEX 30 hyperbaric treatment (total 300 minutes). She received adjunctive therapy consisting of methylprednisolone (80 mg intravenously), aspirin (250 mg orally) and lignocaine (1 mg kg<sup>-1</sup> intravenously). However, the abnormalities persisted, and during the night her condition deteriorated. Repeat examination revealed increased weakness of the left arm

**Figure 1**

Sagittal T2-weighted magnetic resonance image at 24 hr post-injury showing a hyperintense intramedullary lesion at the level of C2–C3 in a diver with decompression sickness



and leg (2/5), with hypoesthesia to light touch below the T3 dermatome and anaesthesia to pain and temperature on the right side at the C7 level. Anal sphincter tone and micturition were also disturbed. These findings were consistent with a typical Brown-Sequard syndrome on the left. Laboratory data were unremarkable. A transcranial ultrasonography Doppler with agitated saline was performed and did not detect a right-to-left shunt.

According to our protocol for the treatment of serious DCS, hyperbaric therapy was continued the following day with a US Navy Treatment Table 6 (extended). On the third day she underwent another COMEX 30 treatment table, followed by 10 daily sessions of hyperbaric oxygen therapy at 254 kPa for 90 min in combination with intensive physiotherapy.

An initial MRI examination (3.0 T system, GE Medical system, HD TX) of the whole spinal cord was undertaken within 24 hours of the injury. This showed an area of hyperintense signal in the left lateral cervical spinal cord on sagittal and axial T2 imaging at level C2–C3 consistent with an ischaemic lesion (Figures 1 and 2). A repeat MRI performed one week later, revealed bilateral signal abnormalities in the posterior spinal horn, corresponding to a spinal cord infarction in the posterior vertebral artery territory (Figures 3 and 4). After two weeks, the clinical outcome remained poor and she was transferred to a functional rehabilitation centre. However, when reviewed six months after the accident, her neurological condition was greatly improved with complete motor recovery, resolution of sphincter dysfunction and persistence of pain sensation and with only a slight impairment of proprioception on the left when walking. The follow-up MRI did not show any residual abnormalities.

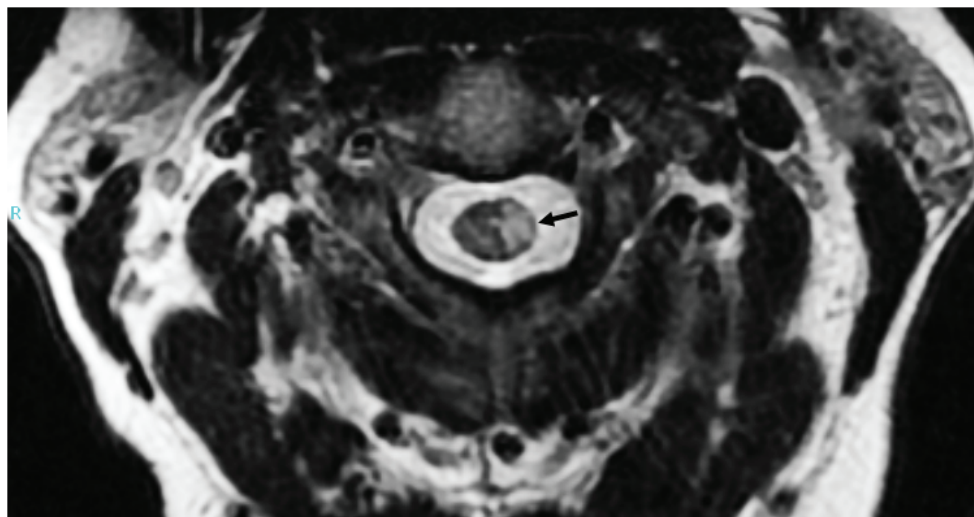
#### Discussion

Spinal cord DCS is not unexpected, but our case is unusual for the following reasons:

- the clinical picture of Brown-Sequard syndrome has rarely been described after scuba diving;
- the repeated MRI scans demonstrated imaging features of diving-related spinal myelopathy that correspond to a variety of pathophysiological possibilities, and

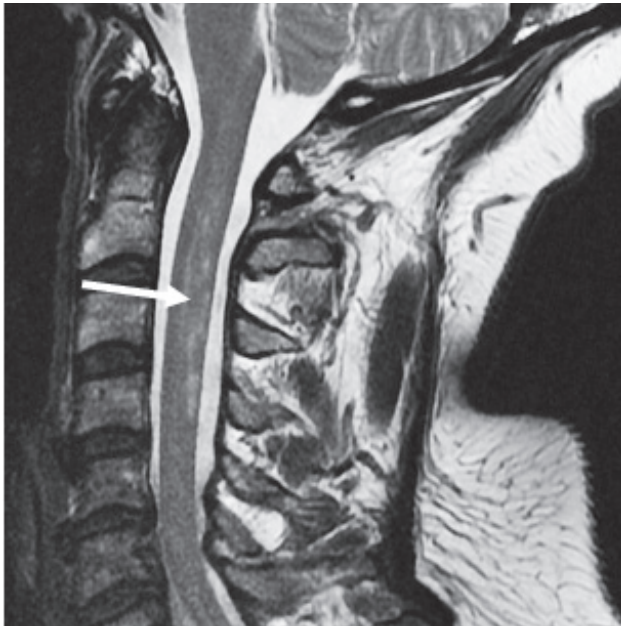
**Figure 2**

Axial T2-weighted magnetic resonance image at 24 hr post-injury showing a hyperintense intramedullary lesion at the level of C2–C3 in a diver with decompression sickness



**Figure 3**

Sagittal T2-weighted MRI in the same diver a week later showing high intensity oedema of the gray matter of the C3 spinal cord with minimal enhancement at the level of C2–C3



- the clinical outcome was ultimately favourable despite severe initial presentation and apparent poor response to HBOT.

To our knowledge, there are only three previously reported cases of Brown-Sequard syndrome attributed to DCS.<sup>6–8</sup> Although the distribution of symptoms was quite different with partial presentation in these cases, MRI findings were consistent with the level of neurological deficit observed

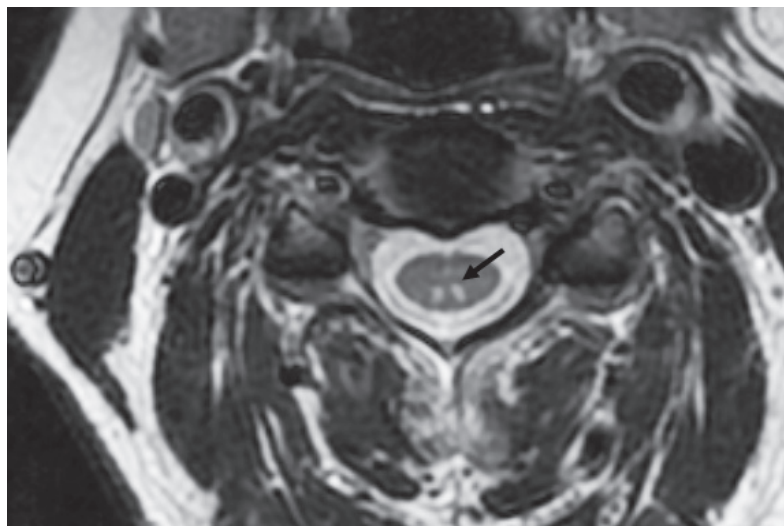
by the authors. In these previous cases, spinal cord lesions were described as focal hyperintense signals on T2-weighted images localised in the lateral and posterior white matter columns of thoracic cord segment as generally observed in past spinal cord DCS imaging studies.<sup>6–12</sup>

In the present report, the first MRI examination showed a diffuse enlargement and increased water content in the left lateral cervical white matter, suggesting the predominant role of oedema and inflammation in the initial part of the pathophysiological process. The most likely mechanism responsible for this myelopathy appears to be spinal cord injury resulting from congestion of the epidural vertebral venous system by nitrogen gas bubbles.<sup>5,13</sup> On the other hand, the repeat MRI depicted a predominance of gray matter involvement, a radiological appearance suggesting infarction in the posterior spinal artery territory.<sup>14,15</sup> This localisation has been described rarely in spinal cord DCS presentation and may be associated with a poor prognosis.<sup>12,16</sup>

Thus, we might assume that the arterial nature and extent of ischaemic damage have two possible origins: either an initial appearance of oedema reflective of ischaemia-reperfusion and cellular dysfunction following arterial occlusion, or a secondary arterial impairment caused by the vasogenic oedema initiated by venous infarction. The severe initial presentation of our patient reflected a likely worse prognosis than the general trend in spinal cord DCS, as evidenced in previous reports.<sup>2,17</sup> However, clinical functional at six months was surprisingly good after treatment encompassing several hyperbaric sessions using heliox mixture, intravenous lignocaine and early intensive physiotherapy and rehabilitation. In practice, the identification of the most important therapeutic factors in preventing severe disability in divers with severe DCS

**Figure 4**

Axial T2-weighted MRI in the same diver a week later demonstrating high intensity oedema of the “H” gray matter of the C3 spinal cord



remains difficult. Nevertheless, this report does suggest that an intensive multidisciplinary treatment programme in the management of such divers holds some promise in the treatment of spinal cord DCS.

### Acknowledgement

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### References

- 1 Francis TJR, Pearson RR, Robertson GA, Hodgson M, Dutka AJ, Flynn ET. Central nervous system decompression sickness: latency of 1070 human cases. *Undersea Biomedical Research*. 1989;15:403-12.
- 2 Blatteau JE, Gempp E, Simon O, Coulange M, Delafosse B, Souday V, et al. Prognostic factors of spinal cord decompression sickness in recreational diving: retrospective and multicentric analysis of 279 cases. *Neurocrit Care*. 2011;15:120-7.
- 3 Francis TJR, Mitchell SJ. Manifestations of decompression disorders. In: Brubakk AO, Neuman TS, editors. *Bennett and Elliot's physiology and medicine of diving*, 5th ed. London: WB Saunders; 2003. p. 578-99.
- 4 Francis TJR, Mitchell SJ. Pathophysiology of decompression sickness. In: Brubakk AO, Neuman TS, editors. *Bennett and Elliot's physiology and medicine of diving*. 5th ed. London: WB Saunders; 2003. p. 530-56.
- 5 Hallenbeck JM, Bove AA, Elliott DH. Mechanisms underlying spinal cord damage in decompression sickness. *Neurology*. 1975;25:308-16.
- 6 Moreh E, Meiner Z, Neeb M, Hiller N, Schwartz I. Spinal decompression sickness presenting as partial Brown-Sequard syndrome and treated with robotic-assisted body-weight support treadmill training. *J Rehabil*. 2009;41:88-9.
- 7 Kimbro T, Tom T, Neuman T. A case of spinal cord decompression sickness presenting as partial Brown-sequard syndrome. *Neurology*. 1997;48:1454-6.
- 8 Hierholzer J, Tempka A, Stroszczyński C, Amodio F, Hosten N, Haas J. MRI in decompression sickness. *Neuroradiology*. 2000;42:368-70.
- 9 Warren LP Jr, Djang WT, Moon RE, Camporesi EM, Sallee DS, Anthony DC, et al. Neuroimaging of scuba diving injuries to the CNS. *AJR Am J Roentgenol*. 1988;151:1003-8.
- 10 Gempp E, Blatteau JE, Stephan E, Pontier JM, Constantin P, Peny C. MRI findings and clinical outcome in 45 divers with spinal cord decompression sickness. *Aviat Space Environ Med*. 2008;79:1-5.
- 11 Sparacia G, Banco A, Sparacia B, Midiri M, Brancatelli G, Accardi M, et al. Magnetic resonance findings in scuba diving-related spinal cord decompression sickness. *MAGMA*. 1997;5:111-5.
- 12 Kei PL, Choong CT, Young T, Lee SH, Lim CCT. Decompression sickness: MRI of the spinal cord. *J Neuroimaging*. 2007;17:1-6.
- 13 Manabe Y, Sakai K, Kashihara K, Shohmori T. Presumed venous infarction in spinal cord decompression sickness. *Am J Neuroradiol*. 1998;19:1578-80.
- 14 Spinal cord infarction. In: Ross JS, Brant-Zawadzki M, Moore KR, Crim J, Chen MZ, Katzman GL, editors. *Diagnostic imaging. Spine*, 1st ed. Salt Lake City: Amirsys; 2007. p. 26-7.
- 15 Masson C, Pruvo JP, Meder JF, Cordonnier C, Touzé E, de la Sayette V, et al. Spinal cord infarction: clinical and magnetic resonance imaging findings and short court term outcome. *J Neurol Neurosurg Psychiatry*. 2004;75:1431-5.
- 16 McCormac J, Mirvis SE, Cotta-Cumba C, Shanmuganathan K. Spinal myelopathy resulting from decompression sickness: MR findings in a case and review of literature. *Emerg Radiol*. 2002;9:240-2.
- 17 Gempp E, Blatteau JE. Risk factors and treatment outcome in scuba divers with spinal cord decompression. *J Crit Care*. 2010;25:236-42.

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