●Original Article

Spinal decompression sickness with possible partial Brown-Séquard syndrome

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We report a rare case of spinal decompression sickness with possible partial Brown-Séquard syndrome. A 45-year-old male conducted a scuba dive to 100 feet with a total diving time of 23 minutes and a rapid ascent. Five minutes after surfacing, he noted numbness in both lower extremities. Following immediate in-water recompression at 33 feet for15minutes at the site, he received recompression therapy to 165 feet seven hours later. His condition deteriorated and he finally showed what we considered possible partial Brown-Séquard syndrome. To our knowledge, only two cases of partial Brown-Séquard syndrome have been reported in decompression sickness. By analyzing all three cases, we identified four common factors : having unusual findings for typical Brown-Séquard syndrome, performing in-water recompression, being symptomatic in the first 10 minutes after surfacing, and showing refractoriness to recompression therapy.

Keywords :
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INTRODUCTION

Decompression sickness (DCS) is believed to occur when an inert gas with which the tissues are supersaturated changes from the soluble to the gaseous form; this occurs when decompression is too rapid[1)2)]. On the basis of its clinical manifestations, DCS has been divided into type 1 (predominantly joint pain) and type 2 (predominantly spinal cord lesions)[3)]. The frequency of spinal DCS has been reported to be 20 to 60% of the cases of DCS that occurred in air dives[4)5)]. To our knowledge, only two cases of spinal DCS exhibiting partial Brown-Séquard syndrome have been reported[6)7)]. We now present a case report of possible partial Brown-Séquard syndrome due to spinal DCS in which we primarily focus on an analysis of the clinical information available in the three cases and their associations with current concepts in DCS.

CASE REPORT

A 45-year-old male, a civilian diver, peformed a scuba dive with his friends in the offshore waters around Niijima island, which is located approximately 150 km south of Tokyo in Japan. The maximum depth of the dive was 100 feet and total diving time was 23 minutes, according to his diving computer. The speed of his ascent was more than 60 feet/minute because he noted that the residual air was not suffcient to conduct an uneventful ascent.

About five minutes after surfacing, he noticed numbness in his lower extremities. He denied

breath holding during the ascent, and he had neither loss of consciousness nor focal weakness. He again dived to a depth of 33 feet and stayed there for 15 minutes, breathing air to achieve in-water recompression. After surfacing from the in-water recompression, his symptoms were relieved to some extent. About 60 minutes later, his symptoms worsened while he was taking a bath. The area of numbness expanded up to his buttock and a cramp-like sensation appeared first in the right thigh.

Two hours after this deterioration, he visited a local medical dispensary on foot and a diagnosis of spinal DCS was made. Seven hours after developing the initial symptoms, he received US Navy Treatment Table 6A (USN TT6A). During the recompression therapy, his symptoms clearly worsened after an ascent from 165 feet to 30 feet. After the recompression therapy was over, he could not walk by himself and he could not void.

In the evening of the day after the dive, he was referred to our squadron (Japan Maritime Self-Defense Force Undersea Medical Center ; JMSDF UMC). We could find no evidence of a pneumothorax, mediastinal emphysema, or subcutaneous emphysema by chest radiography and physical examination. In a neurological examination, he was well oriented and denied dizziness. He showed sensory dissociation with decreased sensation to vibration and position on the right, and to pain and temperature on the left up to T10. At the T8-10 level on the right, he also noted hyperesthesia with paresthesia. Muscle strength was well preserved in the left lower extremity, while the right lower extremity was generally weakened. The deep tendon reflexes showed hyperreflexia with ankle clonus in both lower extremities. A Babinski's sign was absent on both sides. He could not stand up due to loss of position sense and muscle weakness on the right. A volume of 1550 ml of residual urine was drained after insertion of a urinary catheter. We considered these neurological findings to be consistent with partial Brown-Séquard syndrome of the right side, although the presence of bilateral ankle clonus and urinary retention were unusual findings for the typical form of this syndrome.

After the physical examination and confirmation of the diagnosis of spinal DCS, we perfomed US Navy Treatment Table 6 (USN TT6) with full extension. Although the hyperesthesia with paresthesia at T8-10 on the right side improved, the other symptoms showed no change. Since we judged the current case to be refractory to the recompression therapy involved in TT6, we undertook USN TT7 on the third day after his visit to JMSDF UMC. Subsequently, we continued recompression therapy using USN TT6 with full extension. After the second recompression therapy using USN TT6, he could walk on his own. His urinary catheter was successfully removed after the sixth session of therapy using USN TT6. Although a mild sensory abnormality remained even after a total of ten repetitions of recompression therapy including USN TT7 and USN TT6, his muscle weakness and neurogenic bladder showed a degree of improvement, and he was discharged.

DISCUSSION

Neurological lesions in divers with DCS commonly occur in the spinal cord. However, none of the patients with DCS reported by Aharon-Peretz et al. were described as having partial Brown-Séquard syndrome[5]. **Table 1** gives a summary of the clinical information available on spinal DCS with partial or complete Brown-Séquard syndrome ; this information is derived from two previous reports[6,7] and the present case. By analyzing these three cases, we identified four common factors in the physical findings and the phenomenon of spinal DCS presenting as possible partial Brown-Séquard syndrome : 1) the patients all had unusual symptoms for typical Brown-Séquard syndrome, 2) the patients all performed in-water recompression while breathing air (for various times and at various depths), 3) the initial symptoms appeared

Table 1 Spinal decompression sickness presenting as partial Brown-Sequard syndrome

Case	Authors. yr.	Age Sex	Diving apparatus	Dive profile Max depth/dive time/ascent	Time of onset after surfacing	Initial symptoms	In-water recomp Max depth /time	Delay before O2 treatment
1	Levin et al. 1989	29 M	Diving bell	140ft/13min/rapid	6min	Nausea Dizziness	165 ft/10.25 hr	10 hr.<
2	Kimbro et al. 1997	35 M	ND	Repetitive dive 1st：60ft/15min/uneventful 2nd：64ft/36min/uneventful Interval：10min	Immediately	Stabbing pain in midback	64ft/30min<	4hr.
3	Current case	45 M	Scuba	100ft/23min/rapid	5min	Numbness of lower extremities	33ft/15min	7hr.

(Table1. Continued)

Case	O2 Treatment	Level of BSSx Damaged side	Unusual findings for typical BSSx	Major residual symptoms
1	TT6Ax1,TT5x1, 2.5ATA/95min x16	T5-6 Left	Bilateral Babinski's sign Neurogenic bladder	Weakness of lower extremities Paresthesia of hands and feet Neurogenic bladder
2	TT6x1, 2.4ATA/90min x10	T6-11 ? Right	Bilateral hyperreflexia with ankle clonus Neurogenic bladder Dissociation of the level between left and right	Neurogenic bladder Neuropathic pain in left lower extremity
3	TT6Ax1,TT7x1, TT6x9	T10 Right	Bilateral hyperreflexia with ankle clonus Neurogenic bladder	Mild gait disturbance Mild sensory abnomality of lower extremities

ND：Not Described；O2：oxygen；TT：United States Navy Treatment Tables；recomp.：recompression；BSSx：Brown-Séquard syndrome.
2.5 ATA represents 253 kPa.

within the first 10 minutes after surfacing, and 4) the patients all showed refractoriness to vigorous treatment using a recompression chamber, and all had residual symptoms. We now need to discuss these four common factors in turn.

First, all three patients characteristically had a neurogenic bladder and exhibited a bilateral ankle clonus or Babinski's sign, which may suggest bilaterality of the symptoms, a feature considered to be quite unusual for typical Brown-Séquard syndrome. In contrast to the discrete sensory and motor losses that are usually evident after spinal cord trauma, paresthesia in one or more limbs or sensory loss with a patchy distribution seems to be more common in spinal DCS[3]. Of the previous two cases reported of Brown-Séquard syndrome associated with diving injury, that in the article entitled "A case of spinal cord decompression sickness as partial Brown-Séquard syndrome" by Kimbro T et al.[7] showed hyperreflexia of both lower extremities with an 8- to 10-beat ankle clonus and also mild urinary retention. Further, in the other article, one patient presented a bilateral Babinski's sign and a neurogenic bladder[6]. This case was clearly diagnosed as having Brown-Séquard syndrome. Consequently, we feel that the present case justifies the description "partial Brown-Séquard syndrome". However, we accept that opinions will differ on this point, so in this paper we prefer to the present case as showing "possible partial Brown-Séquard syndrome" or "putative Brown-Séquard syndrome". The unusual manifestations in our case, including a neurogenic bladder and a bilateral ankle clonus, would result from damage occurring below the spinal level associated with partial Brown-Séquard syndrome. We speculate that the typical form of Brown-Séquard syndrome often encountered in spinal trauma may never be encountered in cases of spinal DCS.

Second, all three patients received in-water recompression therapy with air breathing. This procedure may relieve symptoms abruptly; however, it has the great disadvantage of causing further inert gas uptake within the body, which may provoke a more severe DCS after the final ascent. In addition to in-water recompression, Case 1 in Table 1 and the current case received USN TT6A more than ten hours and seven hours, respectively, after their final ascent. USN TT6A was originally developed as a treatment measure for AGE (arterial gas embolism); however, even in cases of AGE, the US Navy modified the diving manual to state clearly that AGE cases showing a marked improvement at 60 feet should be treated by USN TT6[8]. Moreover, the Royal Navy strictly forbids recompression to levels deeper than 60 feet in cases of DCS occurring more than five hours prior to the start of treatment[9]. We feel that the use of USN TT6A in the present case was inappropriate and had harmful effects (like in-water recompression with air breathing). Careful consideration should be given before any decision is taken to use USN TT6A in DCS cases in which a substantial delay has elapsed since surfacing.

Third, the initial symptoms appeared within the first 10 minutes after surfacing. In addition to a delay of more than an hour or so in obtaining treatment[10], rapid onset of symptoms after surfacing is well known to carry a poor prognosis in DCS patients[11]; however, the identity of the factor playing the greatest role in determining the prognosis of DCS patients still remains a subject for discussion. Generally speaking, the symptoms of DCS are believed to be closely associated with large deviations from accepted decompression profiles of the omission of a considerable amount of the decompression procedure. However, the Divers Alert Network (DAN) reported that about 58% of DCS cases occured within the limits shown in US Navy no-decompression tables (i.e.,not requiring decompression stops) [12]. It has also been pointed out that the catastrophic form of DCS involving intolerable girdle pain, loss of bladder function, and a loss of power in the lower extremities has been known to develop after short, deep dives[13]. The present case seems to come into this

category. This may suggest that spinal DCS with partial Brown-Séquard syndrome is one form of the catastrophic type of DCS. In recent years, combined DCS and AGE, also called type 3 DCS, has been reported to result in an unfavorable clinical outcome[2) 14)], and to follow a clinical course similar to those shown by the three cases with putative partial Brown-Séquard syndrome. Case 1 can be classified as type 3 DCS. Although Case 2 followed the decompression profile uneventfully, he complained of light-headedness after surfacing. This case might also have had AGE. The current case showed no obvious AGE symptoms, but he performed a rapid ascent. The catastrophic type of DCS and type 3 DCS may be closely related to each other, cases of these two types occurring following a rapid ascent and showing the presence of AGE.

Finally, the three cases discussed here of DCS patients presenting with features of Brown-Séquard syndrome showed marked refractoriness to subsequent recompression therapy. Although the use of tailing treatment for neurological residual symptoms is recommended by the US Navy[8)] and has become a common practice in the diving community, the management of serious cases of DCS still remains one of our greatest problems. In general, repeated application of USN TT6 with or without extension seems to be a common measure in such cases, even though the results have been far from satisfactory. We conducted USN TT7 in the current case because of his dive profile, the rapid onset of DCS symptoms, and the marked deterioration after USN TT6A. Whether a longer stay at 60 feet in USN TT7 would produce a greater improvement in the patient's condition than repeated treatment with USN TT6 remains to be seen. It is evident that the target for recompression therapy has now shifted from the gas bubbles themselves to the secondary effects induced by gas bubbles, i.e., an accumulation of globules of free fat, activation of the intrinsic clotting system, platelet aggregation, and activation of the complement system[1)]. We need more experience before we can be sure whether use of USN TT7 really is advantageous in cases such as the present one.

Although the nature of the pathophysiological mechanisms contributing to the development of spinal DCS remains controversial, as far as we know three theories have been proposed[1) 7) 15)]: a) the spinal congestion theory, b) the autochthonous bubble theory, and c) arterial bubble embolus hypothesis. It would not be appropriate to enter into a full discussion of these theories in this report; however, the following brief comments may be made. It seems unlikely that random bubbles occurring in the spinal cord would remain seven hours after the diver's final ascent, so the secondary effects of gas bubbles would be more likely to be the significant factor in the current case. In actual fact, autochthonous bubbles may arise as an artifact, since bubbles occurring during decompression are mostly intravascular[16)]. The late onset of spinal DCS and the greater involvement of the spinal cord than the brain are against the presence of a greater blood flow to the latter than the former. On balance, we prefer the first (spinal congestion) theory ; this would involve congestion of the spinal epidural system evolving to spinal cord ischemia, may have made the major contribution to the clinical evolution of the current case into putative partial Brown-Séquard syndrome.

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