Cerebrospinal vascular diseases misdiagnosed as decompression illness:
the importance of considering other neurological diagnoses

Kiyotaka Kohshi 1, Yoshitaka Morimatsu 2, Hideki Tamaki 3, Yukio Murata 1, Katsuko Kohshi 1, Tatsuya Ishitake 2, Petar J. Denoble 4

1 Center for Hyperbaric Medicine and Environmental Health, University Hospital of the Ryukyus, Okinawa, JAPAN
2 Department of Environmental Medicine, Kurume University School of Medicine, Kurume, JAPAN
3 Division of Emergency and General Medicine, Tamaki Hospital, Hagi, JAPAN
4 Divers Alert Network, Durham, North Carolina, U.S.

CORRESPONDING AUTHOR: Kiyotaka Kohshi – kohshi@med.u-ryukyu.ac.jp; kohshi33@gmail.com

ABSTRACT

The diagnosis of decompression illness (DCI), which is based on a history of decompression and clinical findings, can sometimes be confounded with other vascular events of the central nervous system. The authors report three cases of divers who were urgently transported to a hyperbaric facility for hyperbaric oxygen treatment of DCI which at admission turned out to be something else.

The first case, a 45-year-old experienced diver with unconsciousness, was clinically diagnosed as having experienced subarachnoid hemorrhage, which was confirmed by CT scan. The second case, a 49-year-old fisherman with a hemiparesis which occurred during diving, was diagnosed as cerebral stroke, resulting in putaminal hemorrhage. The third case, a 54-year-old fisherman with sensory numbness, ataxic gait and urinary retention following sudden post-dive onset of upper back pain, was diagnosed as spinal epidural hematoma; he also showed blood collection in the spinal canal.

Neurological insults following scuba diving can present clinically with confusing features of cerebral and/or spinal DCI. We emphasize the importance of considering cerebral and/or spinal vascular diseases as unusual causes of neurological deficits after or during diving.

Introduction

Decompression illness (DCI) is a term used to describe illness resulting from a reduction in the ambient pressure surrounding the body. DCI encompasses decompression sickness (DCS) and arterial gas embolism (AGE). DCI can present with a great variety of signs and symptoms, including joint pain, cardiopulmonary and neurological problems as well as skin manifestations such as mottling [1]. The diagnosis of DCI is based mostly on clinical manifestations, estimation of the decompression burden and risk factors, and a relationship with the decompression process, which is unusual in modern medicine, which relies greatly on imaging and testing [2,3]. In case of suspected DCI, there is no laboratory test that can confirm or reject the diagnosis. This makes the diagnosis of DCI difficult, particularly when divers present with atypical clinical manifestations [2]. In the absence of standard criteria, clinical diagnosis of DCI is heavily dependent on the physicians’ experience and interpretation of the severity of the preceding dive exposure and clinical conditions of divers [1,3]. We report three misdiagnosed cases of DCI in scuba divers who were evacuated by helicopter to our hospital for hyperbaric oxygen (HBO2) treatment. Correct diagnosis is crucial to the proper management of the events, based on physical and neurological findings.

KEYWORDS: misdiagnosis; differential diagnosis; cerebral stroke; spinal vascular disease; scuba diving
CASE REPORTS
From June 2011 to December 2013, 54 patients were referred to the hyperbaric division or emergency room of our hospital for diving-related injuries. Among these patients 25 (46%) presented with spinal involvement and cerebral complications were urgently transported for HBO treatment after initial diagnosis of neurological DCI. Three of these patients had other vascular disorders of the central nervous system. All the divers were examined by the same hyperbaric physician (neurosurgeon), and diagnosed with neurological DCI or other types of DCI.

Case 1
A 45-year-old woman was referred to the emergency room with suspicion of cerebral AGE after scuba diving using air (maximum depth 20 meters sea water; bottom time 50 minutes). The dive was conducted under optimal conditions with adequate decompression using her dive computer. The divers was guided by and performed with a professional diving instructor; she made no rapid ascent. She was a non-smoker, an office worker with no particular medical history such as hypertension or diabetes mellitus, and had enjoyed a 20-year career of leisure diving.

Approximately half an hour after exiting the water, the diver experienced sudden severe headache, accompanied by nausea and vomiting. She was immediately transferred to a nearby medical clinic. During transfer she became unresponsive to verbal commands and stopped breathing. Basic life support was initiated by the trained diving staff who accompanied her. After five to 10 minutes of resuscitation, spontaneous vital signs were re-established.

In the clinic, she was intubated and ventilated manually. Because of suspicion of cerebral AGE, she was transported by helicopter to our emergency room for HBO treatment. On arrival, the patient was attempting spontaneous respirations and responding to painful stimuli. Clinical examination revealed a blood pressure of 190/130 mmHg and a regular heart rate of 120 beats per minute. Neurological examination showed that she had seriously disturbed consciousness without motor weakness or paralysis. Her clinical course and neurological conditions (severe headache as a first symptom, progression to unconsciousness and absence of motor weakness) suggested cerebrovascular disease, specifically, subarachnoid hemorrhage. A brain CT scan showed massive diffused high density areas in the subarachnoid spaces, with enlargement of ventricles. She was diagnosed as having experienced subarachnoid hemorrhage and treated accordingly. In this diver, non-DCI diagnosis was established based on clinical judgment and confirmed with CT.

Case 2
A 49-year-old right-handed fisherman developed right hemiparesis and speech difficulty during his third scuba dive of the day. He was transported as cerebral AGE to our emergency room. In more than 30 years of his diving career he had never used a diving watch or dive computer. He had a history of several incidents of DCI presenting with joint or muscle pain. He denies ever experiencing motor weakness or sensory numbness. The diver has been taking medication for gout for more than 10 years. Recently he was diagnosed with hypertension but he is taking no medication for it.

On the day of the accident, he made two similar dives to approximately 20 meters in depth, with durations of 50 to 70 minutes. These were followed by a shallow dive to 5 to 15 meters with a similar dive time. Toward the end of his third dive, while on the bottom, he suddenly had blurred vision and lost control of his right hand and leg. He surfaced in a hurry, but he could not board the boat by himself. His diving partner immediately retrieved him and contacted emergency helicopter services.

The diver was presented to our division three hours later. On arrival, he was alert and was able to give a partial history. He had no muscle or skeletal pain. His neurological examination was significant for an expressive aphasia; he answered simple questions appropriately but many words were incomprehensible or inappropriate. Cranial nerves were intact. Strength on the right side was moderately weak, as was sensation to light touch. Based on the fact that his symptoms began before decompression as well as his neurological findings and clinical course, our tentative diagnosis was cerebral hemorrhage or infarction. We ordered a CT scan, which showed the hemorrhage in the left putamen. With this diagnosis, the patient was transferred to a neurological care ward for treatment of the cerebrovascular disease.
Case 3
A 54-year-old fisherman developed numbness in the upper and lower extremities about half an hour after a compressed-air dive to 20-25 meters; duration of the dive was approximately 50 minutes. In a local clinic, he was diagnosed with spinal DCI manifested by a sensory disturbance and urinary retention and transferred to our hospital for hyperbaric treatment.

At admission, detailed neurological examination showed sensory numbness in his lower extremities, the inside of his legs and in the distal portion of his left hand. He had an ataxic gait, but no motor weakness in his extremities. There was no history of trauma, and he denied having hypertension or any other remarkable medical history. He was a non-smoker and took no medications. At that point, the differential diagnosis included vertebral disc hernia or spinal vascular events. However, the diver had a history of several previous instances of DCI treated with recompression therapy, and he wanted to receive emergency hyperbaric treatment because of the ataxic gait and urinary retention.

We administered two HBO2 treatments at 2.5 atmospheres of pressure for 70 minutes, but the diver experienced no relief. On the post-treatment evaluation, the patient disclosed that he had experienced transient serious back pain spreading to the neck and shoulders just after leaving the sea. This history and the neurological findings suggested another spinal disease.

A magnetic resonance imaging (MRI) test was administered seven days after the symptom onset. The MRI demonstrated a homogeneous, isointense lesion with spinal cord compression, finding that were strongly suggestive of a several-days-old epidural hematoma at the Th1-Th7 level. Fortunately the diver's condition improved gradually without surgical decompression for the epidural lesion.

DISCUSSION
Neurological signs and symptoms that present acutely after diving are most likely manifestations of DCI and are emergency conditions requiring HBO2 treatment. However, different neurological diseases and dysbaric events share similarities in their presentations and thus may be mistaken for one another. The three patients presented here were misdiagnosed as DCI because of the proximity in time to their dives, despite the fact that the neurological symptoms they presented are much more frequently caused by cerebral and/or spinal vascular lesions. The misdiagnosis of DCI has been an issue in diving medicine because of the variety in its clinical and neurological symptoms.

In the first case, the diver was diagnosed initially as having experienced cerebral AGE based on serious events – mainly coma after diving. Although coma and/or collapse occurring within seconds or minutes after diving were thought to be characteristic signs of cerebral AGE [4], Newton's series noted there were no divers with coma and only 3% of treated divers had behavioral or consciousness disturbance [5]. In addition, no patients with cerebral AGE suffered from sudden severe headache while they had mild symptoms of cerebral injury.

The clinical findings of the first case would be compatible with those of subarachnoid hemorrhage, as they included neither hemiparesis nor other focal cerebral deficits. We are aware of a single similar case in the literature, a 47-year-old male diver who had blacked out after an emergency ascent. When he regained consciousness he had developed a severe headache [6]. He was initially treated as having experienced cerebral AGE, but the persistent headache led to diagnostic imaging that revealed an aneurysmal subarachnoid hemorrhage.

The second case is a reminder that in cases of post-dive hemiparesis, the distinction between the AGE and the cerebral hemorrhage is not clear. However, the occurrence of symptoms before the start of decompression, the history of untreated hypertension, and the age of the diver, pointed toward possible hemorrhage. Josefsson and Wester, however, reported a case of cerebellar hemorrhage treated by recompression chamber misdiagnosed as DCI [7]. This case, to our knowledge, is the first documented example of cerebral hemorrhage occurring during a scuba dive.

The possibility of intracranial hemorrhagic events should be considered in diving medicine and in unusual cases of neurological DCI. The diagnostic workup should include neuroradiological studies such as CT scan and MRI even though scuba diving is not associated with an increased risk of cerebral hemorrhage.

In divers, spinal DCS often starts acutely within a couple of hours after surfacing, with numbness and weakness in the legs. It is often progressive, with an ascending level of both sensory and motor deficits [1].
These symptoms suggest involvement in the spinal cord with a predominance in the dorsal and lateral columns. A neurological exam shows similar spinal disorders in spinal epidural hematoma and DCS, conditions that are occasionally difficult to differentiate. In addition, spinal disc hernia should be added to the list of differential diagnosis of DCS when divers demonstrate spinal disorders following a dive [8]. Reul, et al. noted in cervical MRI studies that 32 of 50 scuba divers had disc hernia in at least one segment compared with nine of 50 non-divers (P<0.0001) [9]. In contrast, severe back pain preceding spinal disorders is one of the characteristics of spinal epidural hematoma [10] and is uncommon in spinal DCS [1,5].

Although MRI of the brain and/or spinal cord has been used in divers with acute DCI presenting with cerebral and spinal deficits, the technique is relatively insensitive and often fails to detect lesions in patients with obvious signs and symptoms. However, uncommon cases should be examined using neuroradiological studies to exclude hemorrhagic insults in the brain and spinal cord. Note there is no evidence that scuba divers are at risk for these conditions.

HBO₂ is not generally used for treatment of cerebral hemorrhage because of its lack of therapeutic effects. While a few trials to evaluate a surgical approach for this condition did not show serious complications following HBO₂ treatment [11], surgical procedures for cerebral hemorrhage are not common. Subarachnoid or intracerebral hemorrhage is often treated with emergent surgery. In contrast, a few studies have shown that recompression delay of several hours does not affect the prognosis of neurological DCI [12-14]; moreover, a report from Divers Alert Network described early normobaric oxygen inhalation improved or stabilized symptoms of DCI in 95% of 1,045 cases [15]. Based on these studies, the initial clinical assessment of diving-related conditions upon arrival is critical at a hyperbaric center.

CONCLUSION
These reports illustrate that in cases of acute onset of neurological symptoms following decompression from a dive, a rapid neurological diagnostic workup is essential, as therapy for DCI and cerebrovascular and spinal events will differ. In differential diagnoses of DCI, physicians should consider non-diving cerebral conditions as well as and spinal conditions with vascular causes, and conduct careful clinical probes to determine the need for emergency referral to a hyperbaric facility.

Conflict of interest statement
The authors declare that no conflicts of interest exist with this submission.

REFERENCES


