Neurological accidents caused by repetitive breath-hold dives: two case reports

Kiyotaka Kohshi a,*, Takahiko Katoh b, Haruhiko Abe c, Toshio Okudera d

a Department of Neurosurgery and Division of Hyperbaric Medicine, School of Medicine, University of Occupational and Environmental Health, 1-1 Iseigaoka, Yahatanishi-ku, Kitakyushu 807-8555, Japan
b Department of Internal Medicine, School of Medicine, University of Occupational and Environmental Health, 1-1 Iseigaoka, Yahatanishi-ku, Kitakyushu 807-8555, Japan
c Department of Health Information Science, School of Health Sciences, University of Occupational and Environmental Health, 1-1 Iseigaoka, Yahatanishi-ku, Kitakyushu 807-8555, Japan
d Department of Radiology, Akita Research Institute of Brain and Blood Vessels, 6-10 Senshu-Kabota-machi, Akita 010-0874, Japan

Received 17 February 2000; received in revised form 26 May 2000; accepted 26 June 2000

Abstract

We report two Japanese male professional breath-hold divers (33 and 39 years of age) who experienced neurological disorders during repetitive dives to over 20 m of seawater. One patient had right homonymous hemianopsia, and the other presented with right hemiparesis with facial involvement and sensory deficit. In addition, they each had a history of neurological problems following such dives. Magnetic resonance images of their brains disclosed multiple T2-weighted hyperintensities corresponding to their neurological symptoms. Their brain lesions suggest a multiple cerebral infarction caused by occlusion of the cerebral arteries. We conclude that the repetitive deep breath-hold dives induced the brain involvement. © 2000 Elsevier Science B.V. All rights reserved.

Keywords: Dysbaric accident; Decompression illness; Breath-hold diving; Breath-hold diver; Japanese ama diver; Cerebral infarction

1. Introduction

Dysbaric diving accidents, a collective term of all pathological changes related to altered environmental pressure, include two major forms: barotrauma and decompression sickness (DCS). Barotrauma involves tissue damage resulting from a change in the volume of entrapped gas during decompression. Pulmonary barotrauma, caused by alveolar gas expansion, liberates arterialized gas into the systemic circulation, which leads to cerebral arterial gas embolism. DCS occurs when inert gas bubbles form (nitrogen in air breathing) during inadequate de-

*Corresponding author. Tel.: +81-93-603-1611; fax: +81-93-691-4200.
E-mail address: k-kohshi@clnc.uoeh-u.ac.jp (K. Kohshi).
2. Case reports

2.1. Case 1

A 33-year-old man was referred to a local hospital on May 27, 1996 due to visual disturbance in the right field. He was a male ama diver on Mishima Island in Yamaguchi Prefecture who started his profession at the age of 15. On this island, every ama diver wears a full wet suit and carries a weight belt equivalent to neutral buoyancy (7-8 kg). The patient was a partially assisted breath-hold diver who descended to 15-25 m of sea water (msw; measured by a fish finder) with a 15-kg weight and then ascended without assistance. He engaged in daily diving work during the harvesting season. During that time, he repeatedly made 1-1.5-min dives with 1-min surfacing intervals between dives. He dove generally two shifts a day, one in the morning and the other in the afternoon, taking a lunch break in between. He would normally spend 5 to 6 h in the sea. On May 24, he began diving at 9:20 A.M. and continued diving to 22 msw until noon. After a 20-min lunch break, he began diving again. However, a sudden onset of dizziness and blurred vision in the right field occurred at 2:10 P.M. Three years before, he experienced transient salivation from the right corner of his mouth after such dives. He smoked a pack of cigarettes per day for 14 years, but he had no history of stroke or other vascular diseases. An examination of the visual fields showed right homonymous hemianopsia. Other neurological and physical examinations were normal. A full blood count, standard biological examinations, chest radiograph, 12-lead electrocardiography and transesophageal echocardiography were normal. Magnetic resonance images (MRI) of the brain on the 4th day after the onset showed two hyperintense cerebral lesions in the left occipital lobe and right basal ganglia (Fig. 1). The patient was treated with dexamethasone (12 mg i.v. daily for 3 days), followed by drug tapering of 4 mg daily over 3 days. His visual disturbance regressed within 3 weeks but he had residual right lower quadrantanopsia. MR angiography taken 7 days later showed no evidence of a stenotic or obstructive region in the cerebral and cervical arteries.

2.2. Case 2

A 39-year-old male ama diver was admitted to the same hospital on October 4, 1997 with moderate right hemiparesis and a hemisensory disturbance. He lived on Ainoshima Island near Mishima Island. His diving life and patterns were almost the same as Case 1. On October 2, 1997, he began diving around 9:00 A.M. and continued to dive between 20 and 23 msw. He noticed slight dizziness during dives before noon, so he stopped diving and his symptoms disappeared about 30 min later. After a 1-h lunch break, he began diving again until he noticed motor weakness and sensory numbness in his right side at 2:10 P.M. Three years before, he experienced transient salivation from the right corner of his mouth after such dives. He smoked a pack of cigarettes per day for 14 years, but he had no history of stroke or other vascular diseases. An examination of the visual fields showed right homonymous hemianopsia. Other neurological and physical examinations were normal. A full blood count, standard biological examinations, chest radiograph, 12-lead electrocardiography and transesophageal echocardiography were normal. Magnetic resonance images (MRI) of the brain on the 4th day after the onset showed two hyperintense cerebral lesions in the left occipital lobe and right basal ganglia (Fig. 1). The patient was treated with dexamethasone (12 mg i.v. daily for 3 days), followed by drug tapering of 4 mg daily over 3 days. His visual disturbance regressed within 3 weeks but he had residual right lower quadrantanopsia. MR angiography taken 7 days later showed no evidence of a stenotic or obstructive region in the cerebral and cervical arteries.

Fig. 1. MRI of the brain of Case 1. T2-weighted images (TR/TE: 2000/112) obtained on the 4th day after the accident show two increased signal intensities in the left occipital lobe and the right basal ganglia.
P.M. At the ages of 17, 25 and 27 years, he suffered from transient left hemiparesis during repetitive dives. Except for these events, he had no history of stroke or vascular disease. He had smoked half a pack of cigarettes per day since the age of 19 years. The patient was transferred to the hospital because his neurological symptoms progressed. Neurological examinations revealed moderate right hemiparesis with facial involvement and a hemisensory disturbance. An MRI of the brain on the 3rd day after the onset showed 3 hyperintense cerebral lesions (Fig. 2). Other examinations including cerebral MR angiography, as in Case 1, showed no abnormalities. He continued to receive daily hyperbaric oxygen treatments (2.5 atmospheres absolute, 75 min) for 4 days, during which time his neurological functions improved. A follow-up brain MRI obtained 2 weeks later showed a reduction in the size of the hyperintensities in the left parietal and basal ganglia, except for the lesion in the right frontal lobe. He was discharged with slight residual numbness in his right upper limb.

3. Discussion

Because these divers had neither vascular diseases nor risk factors for stroke, except for smoking, and the events happened during repetitive deep dives, there is a great possibility that the breath-hold dives caused the brain damage. The brain lesions demonstrated by MRI suggest a vascular pathogenesis, especially occlusion of cerebral arteries.

Diving accidents were reported in professional breathhold divers of the Tuamotu Archipelago in the South Pacific [2]. The accidents were called “Taravana” diving syndrome [2], which includes vertigo, nausea, partial or complete paralysis, unconsciousness, and even sudden death. Motor paralysis was the most common symptom and was transient. Many diving physiologists suspect that these disorders are compatible with DCS in the brain. In our patients, the findings of neurological disorders were close to the observations in “Taravana” diving syndrome. Characteristically, these diving accidents in breath-hold divers were limited to brain involvement. However, in compressed air divers, the manifestations of neurological DCS are commonly attributed to spinal cord damage [4]. The above symptoms were considered cerebral arterial gas embolisms rather than DCS in the brain [4].

The mechanisms of brain damage following repetitive breath-hold dives are poorly understood. Paulev [5] concluded that disorders caused by repetitive breath-hold dives were DCS because they were immediately relieved by recompression. Because of possible nitrogen accumulation following repetitive deep breath-hold dives [5–7], nitrogen gas bubbles might have formed in the intra- and extravascular spaces of the brain during or after decompress-
Olszowka and Rahn, however, described that nitrogen accumulation in fatty tissues increases consecutively throughout repetitive breath-hold dives despite quickly reaching a steady state in the brain [7]. From the point of view of nitrogen kinetics, it is difficult to explain how de novo bubbles are generated in the brain and cerebral vessels. In contrast, venous gas bubbles are caused by a continuous release of nitrogen from peripheral fatty tissues during and after decompression. By means of an ultrasonic bubble detector, “silent” venous gas bubbles were recorded in a Japanese ama diver after repetitive dives [8]. Since the mammalian lungs usually constitute a competent filter for venous bubbles larger than 21 μm in diameter [9,10], smaller bubbles released by the lungs do not arrest in the cerebral circulation [11]. Hills and James, however, showed that such microbubbles transiently impair the blood–brain barrier [12]. Microbubbles passing through the lungs might have caused the brain involvement in the ama divers, as suggested by Lanphier [6]. Moreover, other possible risk factors or causes for the development of diving accidents are smoking or bubble arterialization through the lungs and/or via a right-to-left intracardiac shunt [13–15]. Both smoking and an intracardiac shunt are known to be risk factors for diving accidents in compressed air divers [14,15]. Although an intracardiac shunt could not be demonstrated in our 2 patients, diving accidents in breath-hold divers might be induced by these risk factors or causes.

Diving accidents in Japanese ama divers have not been previously reported. However, the diving patterns of these ama divers are very similar to those of other places [16]. Why do other Japanese ama divers not develop clinical signs of diving accidents? We may assume that many diving accidents were transient and less serious than “Taravana” diving syndrome. Moreover, a possible explanation is the tendency of keeping diving accidents secret in the Japanese ama communities.

Acknowledgements

We are grateful to Prof. A Yokota for careful review of the manuscript.

References