Decompression illness in Japanese Ama divers

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Summary

Decompression illness (DCI) following repetitive breath-hold (BH) dives has been disputed and debated in the medical fields. We've experienced some professional Japanese breath-hold divers (Ama) with DCI after dives. Their magnetic resonance images of the brain show multiple cerebral infarcts in border or terminal zone areas, and these lesions are considered to be cerebral gas embolism. A questionnaire survey conducted in a district revealed that twelve of 185 Ama divers had experienced stroke-like symptoms such as sensory numbness [7], motor weakness [6], speech disturbance [3], and visual deficit [1]. All neurological disorders were transient and recovered within a month. However, spinal involvements, which are common in compressed air diving, have not been seen in Ama divers. The mechanisms of DCI following BH diving are not clear. We have hypothesized that venous bubbles formed after repetitive BH dives are trapped in the small pulmonary arteries and they are compressed during descending to the bottom and can pass through the lungs. The arterialized bubbles expand during ascending to the surface and they probably induce cerebral gas embolism. However, there are some problems to confirm the hypothesis.

Preface

Although decompression illness (DCI) is well known in compressed air diving, the development is rare among breath-hold (BH) divers. We have experienced some Japanese professional BH (Ama) divers being afflicted, and their events were mainly neurological disorders caused by cerebral stroke (1-3). When an interview survey was conducted in a village, many Ama divers were found to have experienced neurological accidents during BH diving (3). Moreover, several similar cases have been recently reported in professional and amateur BH divers from some areas (4-6). Based on the reported, clinical symptoms and neuro-radiological findings of DCI in BH divers, cerebral lesions are particularly prominent. We show the clinical characteristics of DCI in Japanese Ama divers and hypothesize the mechanisms for DCI following repetitive BH dives.

Japanese Ama divers in Abu-Hagi District

Our interview survey in the village on Mishima Island showed many Ama divers had experienced stroke-like neurological accidents during repetitive BH dives (3). These diving accidents happened during or after more than three hours of repetitive dives. All symptoms were neurological disorders, and motor weakness and sensory numbness in one side were the most common. Others were dysarthria, visual deficit, convulsive seizure, unconsciousness, and so on. In addition to obvious neurological events, many Ama divers frequently felt dizziness, nausea and/or euphoria after long-term deep and repetitive dives. However, there haven't been any informations concerning diving accidents in Ama divers on a large scale.

To estimate diving risks for Ama divers, we've performed a preliminary questionnaire study in Hagi-Abu district, Yamaguchi Prefecture, Japan. This area includes 14 diving branches. The association for fishermen and Ama divers controls their working schedules as days and periods of time, and has 381 Ama divers. In 2008 a standard questionnaire was carried out by the support of the association, and we received answers from 185 Ama divers; 28 partially assisted divers and 157 unassisted divers. They were questioned about their diving

patterns including depth, length of diving shift, diving time and surfacing interval. We obtained a history of diving-related physical and neurological events such as skin rash, joint pain, dizziness, vertigo, nausea, euphoria, motor and sensory involvement, unconsciousness and other clinical conditions. A separate for the divers included questions on the presence of hypertension, cardiac arrhythmia, diabetic mellitus and cerebrovascular diseases.

Twelve of 185 Ama divers (56 yr: 42 to 71) had experienced stroke-like neurological disorders during dives; eleven cases were partially assisted divers and one was unassisted (Fig. 1). Partially assisted Ama divers with DCI descended passively with a 15-20-kg bar weight to the bottom, and then they swam to the surface without assistance. Their diving depths measured with fish finders were 10-20 msw, and diving time and surface interval were 30-75 and 10-40 sec, respectively. A non-assisted Ama diver had motor weakness and paresthesia in one side, and his diving depth was 12 msw. The most common symptoms were paresthesia [7] and motor weakness [6] in one side, and dizziness was accompanied with or without neurological disorders. Moreover, limb pain appeared in a partially assisted Ama diver who had also motor weakness, sensory numbness and speech disturbance. Characteristically, these neurological disorders were transient and resolved completely within a month.

Case Presentation

Case 1: A 33year-old male Ama diver was referred to our hospital due to visual disturbance. He was a partially assisted BH diver who descended to 15-25 msw using a 15-kg weight and then ascended without assistance. He repeatedly made 1-1.5 min dives with 1-min surface interval between dives. He began to dive at 0920 and continued diving until noon, and he started to work after a 20-min lunch break. He noticed dizziness and blurred vision at around 1400. He consulted to a doctor on fourth day, and his magnetic resonance image (MRI) showed two cerebral lesions in the left occipital lobe and the right basal ganglia (Fig.2). The right-to-left shunt could not be detected using the Doppler echocardiography. The size of the lesions regressed for several days, but he had a partial visual deficit five years later.

Case 2: A 43-year-old male Ama diver had a history of diving accidents at the age of 34 years. His diving patters were similar to those of Case 1. After deep and repetitive BH dives, he noticed motor weakness and sensory numbness in his left side. However, he had a convulsive seizure with unconsciousness three hours after improving of hemiparesis. When he was evaluated nine years after the event, he had no neurological deficits despite multiple cerebral lesions in the frontal lobes and periventricular white matter (Fig. 3).

Case 3: A 39-year-old male Ama diver is an acute case of DCI. His diving patterns were similar or same as those in above cases. Previously, he had three times of diving accident. The first event happened at 17 years old, and the second was 25 years of age. Previous diving accident was the left motor weakness, but at this time he had motor weakness and sensory numbness in his right side. His brain MRI on third day showed multiple infractions of acute and old stages (Fig. 4).

Diving accidents in Ama divers

- **Neurological diving accidents: 12/185 Ama**
  assisted : 11/28 (39.3%)
  non-assisted: 1/157 (0.6%)

Neurological symptoms (12)	***Accompanied symptoms (10)***
• paresthesia: 7	• dizziness: 8
• motor weakness: 6	• vertigo: 2
• speech: 3	• nausea: 2
• visual: 1	• limb pain: 1

Fig.1 Results of a questionnaire study for Ama divers

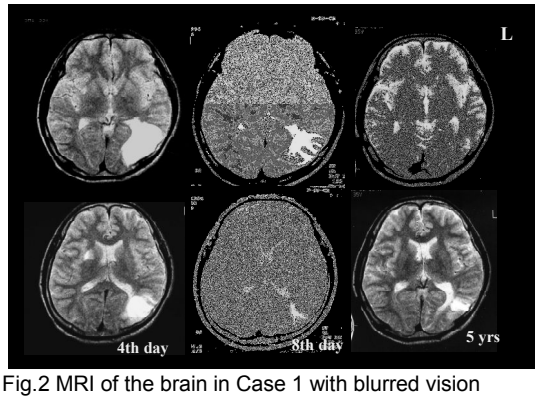

Fig.2 MRI of the brain in Case 1 with blurred vision

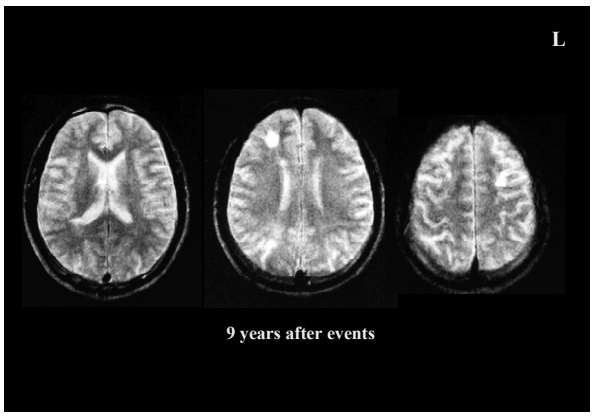

Fig.3 MRI of the brain in Case 2 with a history of motor and sensory involvement and convulsive seizure 9 years earlier

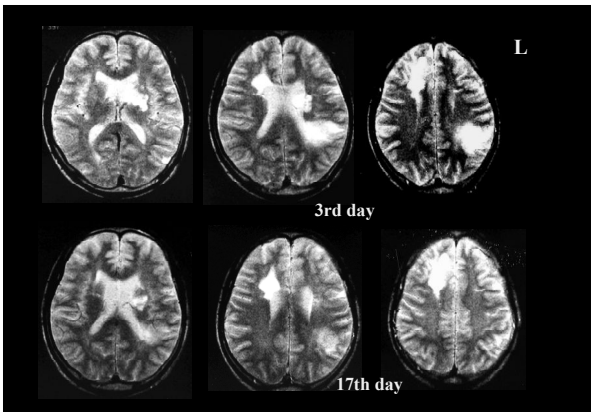

Fig.4 MRI of the brain in Case 3 with acute and chronic diving accidents

Development Mechanisms

Mechanisms for DCI in BH divers have not been elucidated (Fig.5). Nitrogen (N_2) accumulation in fatty tissues increases consecutively throughout repetitive BH dives despite quickly reaching a steady state in the brain (7, 8). From the point of N_2 kinetics in BH diving, it is easy to consider that N_2 gas bubbles are formed in the venous side of tissues rather than the brain. Although the bubble formation following repetitive BH dives is controversial, venous bubbles were recorded in Japanese Ama divers (9, 10). In addition, some investigators suggest that the bubble formation in venous side is a possible mechanism for cerebral DCI (11-13). After deep, repetitive BH dives, bubbles may be formed in venous side and reach to the lungs. Microbubbles passing through the pulmonary capillaries, are generally harmless to tissues including the brain. Since an experimental study showed that microbubbles impair the blood brain barrier transiently (14), we

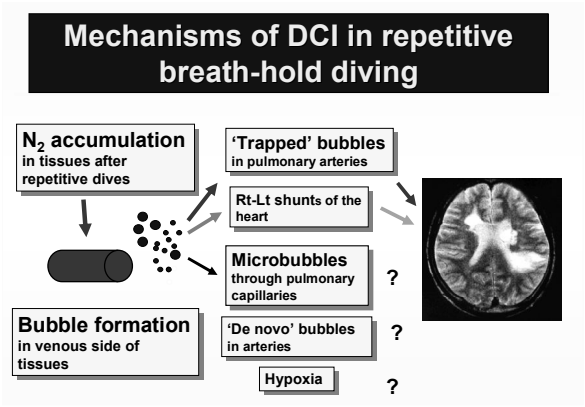

Fig.5 Mechanisms of decompression illness in repetitive breath-hold dives

once thought the major mechanism of DCI in BH divers was such microbubbles (2, 3, 11). However, we do not think microbubbles are the major mechanism of DCI from the MRI findings of Ama divers with DCI. Because multiple infarcts are located in border and terminal zones of the cerebral arteries, we should consider another factor as a major mechanism of DCI in BH divers. Venous bubbles larger than 21 μm are retained or trapped at small pulmonary arteries of mammalian lungs (15). When Ama divers descend to the bottom, such "trapped" bubbles are compressed and easily pass through pulmonary capillaries. Such bubbles passing through the lungs enlarge during ascending to the surface and the bubbles induce arterial gas embolism. In addition, there is a possibility that "trapped" bubbles are related to microbubbles. "Trapped" bubbles and microbubbles are mixed, and these bubbles probably induce cerebral infarcts. Moreover, other mechanisms such as cardiac shunt or pulmonary barotrauma should be considered, but we do not think these are major etiological factors of DCI caused by repetitive BH dives (16).

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