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CASE REPORT

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Hyperacute brain magnetic resonance imaging of decompression illness in a commercial breath-hold diver

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Abstract

Decompression illness in breath-hold diving is a rare dysbaric disease mainly characterized by stroke-like neurological disorders. The early use of DWI-MRI combined with ADC map in suspected cases can help in the early diagnosis and treatment.

KEYWORDS

arterial gas embolism, breath-hold diving, cerebral stroke, decompression illness, magnetic resonance imaging

1 | INTRODUCTION

Decompression illness (DCI), dysbaric disease including decompression sickness and arterial gas embolism, is common in compressed-air divers or workers, and extremely rare in breath-hold divers. The accidents following breath-hold diving are almost exclusively neurological, stroke-like events involving the brain.[1,2] Magnetic resonance imaging (MRI) is the most sensitive technique available to evaluate cerebral ischemic lesions. There are much reported data on the prevalence of brain MRI abnormalities and their characteristics in compressed-air and breath-hold divers. While the accuracy of diffusion-weighted imaging (DWI) and apparent diffusion coefficient (ADC) map is well established in the diagnosis of all phases of cerebral ischemia,[3] few previous reports have studied hyperacute DCI of the brain in divers.

We report a case of a commercial breath-hold diver (called Ama) who developed neurological disorders after repetitive dives. Conventional MRI, DWI, and ADC map revealed hyperacute and subacute ischemic lesions of the brain. We emphasize the usefulness of MRI study in the diagnosis of the hyperacute phase of cerebral DCI. Besides, we discuss hyperbaric oxygen (HBO) therapy and normobaric oxygen (NBO) breathing for acute cerebral DCI.

2 | CASE DESCRIPTION

A 65-year-old right-handed fisherman, a nonsmoker who was previously in good health, started partially assisted breath-hold diving at the age of 30. In general, he makes his dives dressed in a full wetsuit and carries a weight belt equivalent to neutral buoyancy. He descends passively from his boat to 10-20 m or more using a 23-kg weight and actively ascends without assistance. The union rule limits harvesting from Monday to Thursday and sets the dive time from 8:00 AM

to 3:00 PM. The diver usually works two shifts a day with a break for lunch. He has been diving the same way for more than 30 years and has never before experienced any neurological symptoms or musculoskeletal pain.

On the day of the accident, which was his fourth successive day of diving, the diver began diving at around 8:00 AM with the usual dive depth of 10-20 m. At the end of the morning diving shift, however, he experienced slurred speech and right-hand paresthesia. On the boat, he could rise without any assistance, but he was not able to walk steadily. He had no chest pain, bloody sputum, loss of consciousness, or motor weakness. Within one hour, he was admitted to the emergency department. His vital parameters were normal (blood pressure 118/82 mm Hg and heart rate 65 beats per minute). We obtained a chest X-ray and a 12-lead ECG, all of which did not present pathological findings. A complete blood count, hematocrit, hemostatic tests, and blood chemistry were within the normal range. The neurological assessment revealed dysarthria, sensory numbness in the right-sided upper limb, and unstable gait. Brain MRI, performed within two hours after the diving accident, included fluid-attenuated inversion recovery (FLAIR), DWI, and ADC map. Hyperintense areas on FLAIR and DWI were shown in the pons and right-sided parietal lobe, in which the former FLAIR hyperintensity was weak compared with the latter one, and the pontine lesion showed lower values on ADC map despite slightly increasing in the other one (Figure 1). There were no atheromatous plaques or abnormalities of supra-aortic vessels.

Given these findings, the diagnosis was hyperacute pontine ischemia caused by the arterial gas embolism (AGE) and subacute ischemic lesion in the parietal white matter. We recommended a transfer to a hyperbaric center with a multi-place hyperbaric chamber, which he refused and opted for a treatment in our mono-place hyperbaric chamber. He promptly underwent an emergency session of HBO therapy (2.0 atmospheres absolute; ATA) for 60 minutes together with intravenous rehydration (Ringer's lactate, 1000 mL). His gait disturbance resolved the next day. Daily HBO therapy was continued for 7 days. He was discharged with a residual numbness in his hand, which gradually improved over several months. At the follow-up visit five months later, the diver was asymptomatic and with no apparent neurological disorders. A follow-up brain MRI showed a reduction of the pontine hyperintensity area but no dramatic change in the right parietal lesion.

3 | DISCUSSION

The diagnosis of DCI, in this case, was reasonable regarding the onset of neurological manifestations soon after multiple breath-hold dives and no history of vascular diseases or risk factors for stroke. Brain MRI including DWI and ADC map was useful to evaluate ischemic lesions and helped decision about treatment. Moreover, HBO therapy following early NBO breathing is effective for hyperacute cerebral DCI in breath-hold divers.

The characteristic of DCI in breath-hold divers is that stroke-like brain involvement is common,[1,2] while the spinal cord, unlike in compressed-air divers, is rare. Typically, cerebral DCI manifests with sensory numbness or motor weakness on one side, disturbed speech, and/or visual deficit after repetitive dives for several hours. The post-dive neurological events in breath-hold divers have not been considered serious because symptoms disappeared rapidly after hyperbaric therapy or resolved spontaneously within several hours. However, in rare cases, post-dive neurological symptoms may be fatal. Cross reported that out of 235 observed breath-hold divers

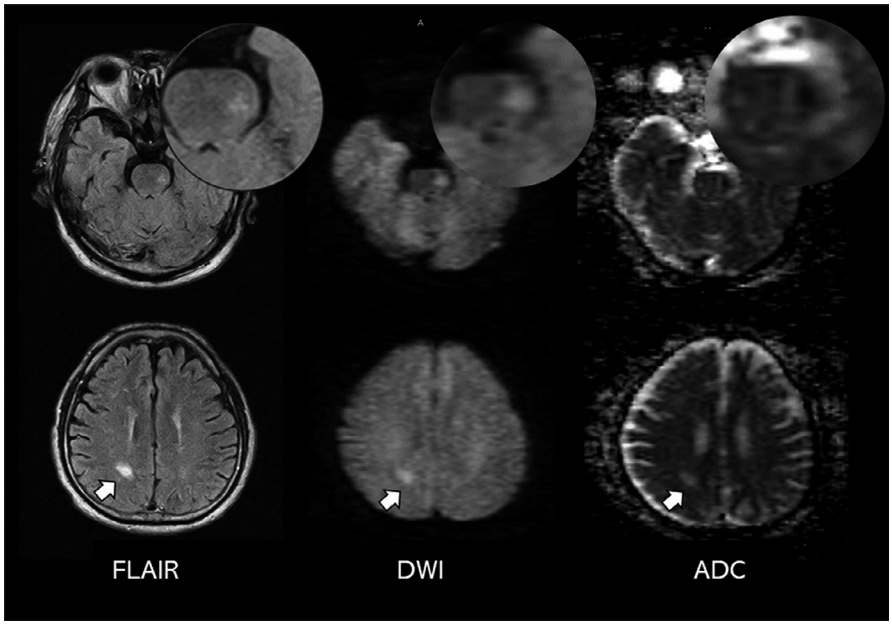

FIGURE 1 FLAIR-MRI, DWI, and ADC map. The upper panels are slices at the brainstem and the lower are at the parietal white matter. Insets are larger images, and arrows show ischemic infarcts

47 (20%) exhibited neurological symptoms known as "taravana" diving syndrome. In three cases, the taravana included unconsciousness, and in two cases mental disturbance and death.[4] Our case series have shown that a few breath-hold divers experienced serious neurological disorders, including unconsciousness, convulsive seizure, and/or crossed sensory numbness.[1] These reports indicate that multiple breath-hold dives in succession are not benign and that neurological accidents following such diving require early diagnosis and treatment.

There are many reports of the ischemic brain lesions in breath-hold divers documented using the MRI technique. The lesions are localized in the brainstem, basal ganglia, and deep and subcortical white matter.[5-7] The lesions in the brainstem or basal ganglia are situated in the terminal zone, and the lesions involving deep or superficial white matter correspond to the border zone or watershed regions.[8,9] They are so-called "low-flow" cerebral circulations as a result of the low perfusion pressure in the terminal supply area. Although conventional MRI with DWI and ADC map is the best imaging modality to find pathological changes of ischemic brain lesions,[3] these techniques were rarely used in suspected cerebral DCI in a hyperacute stage of the ictus. Both DWI and ADC map show changes in ischemic brain tissue within hours after symptom onset.[10] Hyperacute ischemic lesions are characterized by high signals on DWI and low ADC values.[10] In the present case, one of the hyperintensity regions on FLAIR could be diagnosed as hyperacute brain ischemia. These MRI sequences are also useful to estimate the age of brain ischemia when multiple lesions with possible different times of onset are present. In general, diving physicians tend to think that imaging studies are not so helpful for the evaluation or management of neurological DCI. One reason is that conventional MRI is relatively insensitive and often fails to detect lesions in patients with obvious neurological signs and symptoms of DCI.[11,12] However, an MRI study is essential to evaluate stages of brain ischemia following diving and to exclude hemorrhagic insults.[13] It is especially important in the case of suspected DCI after breath-hold diving, which is very rare while the stroke is more common.

Brain lesions in breath-hold divers with DCI are situated in the border or terminal zones of cerebral arteries.[5] In general, the main clinical features of cerebral infarcts in these areas show transient ischemic recovery or mild disability, even though their lesions at the early phase are considerably large on CT or MRI.[8,14] However, neurological symptoms, including unconsciousness, double vision, or crossed sensory numbness, suggest brainstem ischemic attacks, which can cause fatal cardiorespiratory disturbances.[15] HBO therapy as a key role in the treatment of bubble diseases appears to be effective for the hyperacute phase of iatrogenic AGE,[16,17] although the beneficial effect has not been shown in acute ischemic stroke.[18] When fifty-nine divers with neurological DCI were treated with HBO therapy within 6 hours after symptom onset, 22 of them as cerebral involvement did not exhibit neurological and neuropsychological sequelae.[19] Moreover, a survey from Divers Alert Network described early NBO breathing improved or stabilized DCI symptoms in 95% of 1045 cases.[20] DCI in breath-hold divers is mainly cerebral involvement showing stroke-like neurological symptoms and signs.[1,2,7] For the treatment of DCI in breath-hold divers, NBO breathing should start as early as possible, followed by HBO therapy within 6 hours after the onset. Permanent brain changes can remain, despite treatment. Thus, it is of utmost importance for divers to protect themself from the cerebral DCI by NBO breathing after repetitive breath-hold dives for several hours.

4 | CONCLUSION

Repetitive breath-hold dives tend to induce stroke-like neurological disorders which are occasionally serious for divers. Conventional MRI sequence including DWI and ADC map is useful to evaluate the hyperacute ischemic lesions of the brain and to exclude hemorrhagic cerebral insults in divers. HBO therapy following NBO breathing should be applied for injured divers as soon as possible.

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CONFLICT OF INTEREST
None declared.

AUTHOR CONTRIBUTIONS
KK, YM, and TI: wrote the initial draft. HT: obtained patient data (images and clinical history) and contributed to patient care. TI and PJD: critically reviewed the paper. All authors: participated in reviewing the literature, interpretation of clinical findings, critical revision of the manuscript for important intellectual content, and approval of the final version.

INFORMED CONSENT
Informed consent was obtained from the patient.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section.

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