The on-site differential diagnosis of decompression sickness from endogenous cerebral ischaemia in an elderly Ama diver using ultrasound

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Abstract

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Commercial or occupational breath-hold (BH) harvest divers along the coast and islands of Japan are collectively called Ama divers. Repetitive BH diving by Ama divers may place them at risk of developing neurological decompression sickness (DCS). We report a 74-year-old Ama diver who demonstrated right hemiparesis during an ascent after free diving at a depth of 5 metres' sea water. This report suggests the usefulness of on-site ultrasound for making a differential diagnosis of DCS from endogenous cerebral ischaemia. Further clinical studies of this management approach are warranted.

Introduction

It was previously believed that a diver must be exposed to a certain minimum depth before bubbles could form and that a diver could spend an unlimited amount of time at shallow depths (< 10 meters' sea water, msw).[1] However, it is now known that bubble formation can occur even after shallow dives and that it is inappropriate to exclude the diagnosis of decompression sickness (DCS) based on a perceived minimum depth.[1] Even when a diver follows the decompression tables, excess nitrogen remains in the blood for hours after a dive. Doppler studies have shown that up to 10% of remaining nitrogen may be released as bubbles after a normal dive.[2]

Commercial or occupational breath-hold (BH) harvest divers along the coast and islands of Japan are collectively known as Ama divers.[3] Repetitive BH diving can lead to the accumulation of nitrogen (N_2) in blood and tissues, which may give rise to DCS,[4] and previous studies have suggested that Ama divers are at risk of neurological DCS.[3–5] We report a patient with endogenous transient ischaemia who demonstrated right hemiparesis during her ascent after free diving to a depth of 5 metres' sea water (msw).

Case report

A 74-year-old Japanese female Ama diver was rescued by colleagues after she developed right hemiparesis during ascent while free diving to a depth of 5 msw. She had made multiple dives over three hours to similar depths collecting turban shells and abalone. The exact times of the dives could not be determined. She had known hypertension on medication. She was taken ashore, and an emergency call resulted in an ambulance being dispatched. A physician-staffed helicopter from Eastern Shizuoka was also requested, as divers with DCS are transported to a hospital located further away for recompression since there are no suitable hospitals on the Izu Peninsula.

When examined on site by a physician an hour post dive, she was asymptomatic. As portable ultrasound (Vscan®; General Electric, USA) using the longitudinal sub-xiphoid window did not reveal any bubbles in her inferior vena cava, she was diagnosed with endogenous cerebral ischaemia not induced by DCS or air embolism from barotrauma. She was transported to our hospital (which does not have recompression facilities) by helicopter whilst receiving intravenous Ringer's solution and on oxygen.

On arrival, she was fully conscious and orientated, with isocoric, reactive pupils. Neurological examination was unremarkable. Whole-body computed tomography (CT) revealed no gas in her body, and there were no blebs, small bullae or air trapping in the lungs. Magnetic resonance imaging (MRI) of her head two hours after the incident, including diffusion-weighted imaging and angiography, were negative. Biochemical analyses of blood revealed no abnormalities, including for D-dimer. Right-to-left shunt, including a persistent foramen ovale (PFO), was excluded on

ultrasound. A dagnosis of a transient endogenous ischaemic attack and hypertension was made, and she received anti-platelet therapy without recompression treatment. Her post-admission course was uneventful and she was discharged on hospital day five without sequelae.

Discussion

This report suggests the potential usefulness of on-site ultrasound for the differential diagnosis of a central neurological event. The different diagnosis of DCS, air embolism from pulmonary barotrauma or endogenous stroke was necessary in the present case. Free diving as performed by Japanese Ama is a rare cause of DCS but still one that should be considered.[3] The mechanism of stroke induced by DCS or air embolism is intravascular gas produced by abrupt decompression or barotrauma. Also there is a strong correlation between DCS and PFO, where venous bubbles can become arterialized via the right-to-left shunt and, thus, result in cerebral symptoms.[6] Repetitive deep breath-hold diving, long dive times and short surface intervals result in N_2 loading predisposing divers to DCS, which characteristically manifests as cerebral stroke.[3,7–9] This elderly Ama had no air bubbles on ultrasound at the scene or on subsequent CT, a negative D-dimer level and no right-to-left shunt, so the possibility of DCS was unlikely.

Even in asymptomatic divers, gas bubbles can be detected by ultrasound immediately after diving.[10] Loss of consciousness during diving may be from decompression illness (DCI), hypoxia induced by drowning, immersion pulmonary oedema or a cardiac event from immersion and exercise.[11] In such cases, physicians on board the Eastern Shizuoka helicopter routinely use ultrasound to make a differential diagnosis at the scene in order to decide to which hospital the diver will be transported, dependent on the availability of recompression facilities. However, the use of this device in making a differential diagnosis has not been validated in the literature. There may be criticism that the absence of bubbles in the inferior vena cava, even in the acute phase, cannot exclude the presence of bubbles in the whole body of a diver, so divers with focal neurological signs should always be transported to a hospital with a recompression chamber as a precaution.

Conclusion

This report suggests the usefulness of on-site ultrasound for the differential diagnosis of DCS from endogenous cerebral ischaemia in an elderly Ama diver. Further clinical experience with ultrasound for the acute differential diagnosis of divers with neurological symptoms and signs is merited.

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