Mystery Case: White matter lesion related to decompression sickness following extreme breath-hold diving

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Figure Axial brain MRI

(A) Initial FLAIR shows a hyperintense subcortical linear left frontal lesion, sparing the subcortical U-fibers. On diffusion-weighted imaging (B), the lesion is hyperintense, without corresponding low apparent diffusion coefficient map values (C), showing no restricted diffusion. (D) FLAIR at 1-month follow-up shows subtotal lesion regression.

A 39-year-old man developed transient expressive aphasia and headache following extreme breath-hold diving. He had performed around 30 breath-hold dives, lasting approximately 2 minutes, 30 meters deep, for 5 hours, with short surface intervals. Brain MRI (figure, A–C) revealed a left frontal white matter lesion, without restricted diffusion, contrast enhancement, or mass effect.

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At 1-month follow-up, after 3 treatment sessions of hyperbaric oxygen therapy, the patient remained asymptomatic and the lesion had regressed almost completely (figure, D).

Accumulation of nitrogen in blood and tissues after repetitive breath-hold diving¹ has been suggested to cause endothelial dysfunction and disruption of the blood–brain barrier, with subsequent hyperpermeability in microvasculature and vasogenic edema.²

Author contributions

C. Guerreiro: acquisition of data and manuscript elaboration. A. Teixeira: acquisition of data and manuscript elaboration. T. Marques: manuscript revision. Sofia Reimão: supervision and critical revision.

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Disclosure

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Mystery Case Responses: A 39-year-old man with transient aphasia

The Mystery Case series was initiated by the *Neurology*® Resident & Fellow Section to develop the clinical reasoning skills of trainees. Residency programs, medical student preceptors, and individuals were invited to use this Mystery Case as an educational tool. Responses to multiple choice questions formulated using this case were solicited through a group email sent to the American Academy of Neurology Consortium of Neurology Residents and Fellows and through social media. We received 161 responses. The majority of respondents (70%) had been in practice for 1–4 years; 65% were residents or fellows while 29% were faculty/board-certified physicians; the remainder were medical students or advanced practice providers. A total of 48% resided outside the United States. A wide range of practice settings was represented.

When presented with this patient's history of transient expressive aphasia and headache following extreme breath-hold

diving, and shown the initial MRI brain, 63% of respondents correctly recognized the findings of hyperintense left frontal fluid-attenuated inversion recovery (FLAIR) lesion (sparing subcortical U-fibers), and 33% also recognized the FLAIR/diffusion-weighted imaging (DWI)/apparent diffusion coefficient (ADC) findings as being suggestive of vasogenic edema. When asked what treatments they would consider, 73% correctly selected hyperbaric oxygen therapy and 40% selected initial high flow oxygen. A total of 18% incorrectly selected aspirin, which is not indicated in this case as the patient did not actually have a stroke/TIA.

Upon then being provided the patient's follow-up MRI showing regression of the lesion after 3 treatment sessions of hyperbaric oxygen therapy, and asked to identify potential mechanisms for this presentation, the following appropriate mechanisms were identified: (1) accumulation of nitrogen in blood and tissues after repetitive breath-hold deep-diving (by 44%), (2) endothelial dysfunction and disruption of the blood–brain barrier (by 18%), (3) bubble formation in the vasculature from excess dissolved gases (45%), and (4) hyperpermeability in brain microvasculature contributing to vasogenic edema (24%). One favored incorrect option was vaso-occlusion due to the dissolving of nitrogen in blood under high pressure (33%). Small nitrogen emboli are an important complication of deep sea and scuba diving, resulting in Caisson disease, with ischemia of the brain and spinal cord and prominent signs of spinal cord dysfunction; hyperbaric oxygen is the usual treatment.¹ However, such occlusive emboli are unlikely to have occurred in this case given the absence of true diffusion restriction (bright on DWI, dark on ADC) as expected with ischemia on neuroimaging.

This case highlights a complication of decompression sickness following extreme breath-hold diving that may present to neurologists.

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