

Brain multi-infarct and decompression sickness

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Dear Editor

Scuba diving is associated with an important risk of developing decompression sickness secondary to formation of gas bubbles inside the body. The latter is formed mainly by nitrogen in the body on the diver's way to the surface (1,2). In some cases, it might injure the central nervous system. Several decompression cases that have been associated with neurologic symptoms are described in the literature; however, brain multi-infarct with lethal outcome has never been described.

A 41-year-old male, came to the ER with clinical suspicion of decompression sickness (he dived for 15 minutes at a depth of 50 m). Among his health history, he was a heavy smoker, 30 minutes after leaving the water; he suddenly had dysarthria and vertigo.

Upon admission, the patient had a poor general condition, Glasgow Coma Scale (GCS) 9 points, and horizontal nystagmus with right hemiparesis 4/5. Brain magnetic resonance imaging (MRI) demonstrated multiple cerebral and cerebellar infarctions. Echocardiography did not reveal the presence of patent foramen ovale (Figure 1). The patient was required to be transferred to intensive care unit and to a hyperbaric chamber session. He progressed to hemodynamic instability, dying within 36 hours after admission.

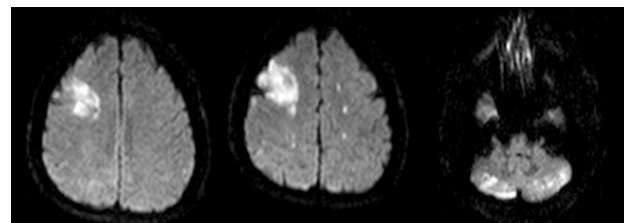


Figure 1. Brain MRI showing brain multiple and cerebellar infarct.

Decompression sickness is thought to be associated with thrombotic events due to intravascular gas bubbles. It is accepted that mechanical abrasion to vessel's walls may induce endothelial dysfunction and activation of the blood cells that are responsible to start the inflammatory reaction, vasomotor dysfunction, platelet aggregation pathway and coagulopathy. This disease has been classified into two types: type I and II, based on the severity of signs and symptoms. Type I is a mild form that requires less recompression and treatment, type II is associated with neurologic symptoms. (3). The latter requires an extensive therapy and may lead to significant neurologic sequelae. Our case report represents a type II decompression sickness. It is known that the delay to start recompression treatment represents a poor prognosis. Early start of hyperbaric therapy will reduce the size of the gas bubbles, improve perfusion and re-oxygenate ischemic tissue before late inflammatory processes might occur. These include cytokines, platelet activation and complement activation. (4,5).

Ethical issues

Not applicable.

Authors' contributions

All authors contributed equally.

Received: 13 January 2017; Accepted: 20 March 2017;

Published online: 27 May 2017

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Competing interests: Authors declare that they have no competing interests.

Funding information: There is none to be declared.

Citation: Moscote-Salazar LR, Satyarthee GD, Hernandez NE, Moscote-Salazar LR, Matus JA, Calderon-Miranda WG, Blancas-Varas MA, et al. Brain multi-infarct and decompression sickness. Journal of Emergency Practice and Trauma 2017; x(x): x-x. doi: 10.15171/jept.2017.17.



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