A Rare Case Of An Isolated Central Retinal Artery Occlusion Following A Recreational Scuba Diving: A Case Report.

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ABSTRACT

Clinical manifestation of decompression illness after a scuba diving may vary widely. We report a rare case of a 29-year-old man with decompression illness that manifested as isolated retinal artery occlusion in a diver with a single shallow dive profile but missed the obligatory safety stop due to poor buoyancy control. He was treated successfully with one cycle of Treatment Table 5 followed by four cycles of Treatment Table 6 (US Navy Treatment Table) of hyperbaric oxygen therapy.

Key words: Retinal artery occlusion, scuba diving, arterial gas embolism

INTRODUCTION

Scuba diving is associated with a risk of decompression illness (DCI) due to formation of inert nitrogen bubbles under pressure that mainly affect the central nervous system. [1] It is caused by bubbles in blood or tissues during or after a sudden reduction in ambient pressure manifesting in a large range of signs and symptoms. In scuba diving, DCI mostly occurs in divers engaging in excessively long dive time, deep dive or if the mandatory `stops', which are pause during ascend to surface at the end of a dive to safely remove dissolved inert gas from the body, have been omitted. It encompasses two pathophysiological syndromes: pulmonary over-inflation syndrome (POIS) and decompression sickness (DCS). [1] Butler (1995) found that visual disturbances were seen in 7-12% of all DCS that include nystagmus, diplopia, visual field defects, scotoma, homonymous hemianopia, orbicularis oculi pain, cortical blindness, convergence insufficiency, central retinal artery occlusion and optic neuropathy. [2] We describe a rare case of an isolated central retina artery occlusion (CRAO) as a manifestation of decompression illness due to scuba diving, which is of medical interest. This case also highlights the possible occurrence of the incidence albeit a mere single dive and a relatively shallow dive profile which might have an

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implication on scuba diving safety and precautious.

CASE REPORT

A 29-year-old man presented to Hospital Universiti Sains Malaysia with progressive painless blurring of vision of the right eye associated with dizziness after two hours surfacing from a single recreational scuba diving. The dive was to a maximum depth of ten meters with a bottom time of twenty-five minutes. During ascending to five meters depth at the end of the dive he had thirty bar (435 psi) of air pressure left in the tank.[3] With the near empty tank of air he had trouble maintaining neutral buoyancy and went straight up to the surface without being able to perform the mandatory safety stop procedure. He did not complain of any limb weakness, numbness or any joint pain.

On physical examination, he was fully conscious with normal vital signs. His right eye acuity was 6/60 with a positive grade II relative afferent pupillary defect (RAPD). Optic nerve function such as light brightness and red saturation were 20% reduced. Posterior segment examinations of the right eye revealed flat retina with cherry red spot and pale macula (Figure 1). The cherry red spot is a contrast between edematous, pale retina and reddish choroid. The optic disc was pink, not swollen, had well-defined margin and the cup-disc-ratio was 0.3. However, no focal arterial narrowing and optic nerve lesions were seen. The left fundus was normal. Intraocular pressure was 10 mmHg on the left eye and 12 mmHg on the right eye. Other central nervous system examinations were normal.

A diagnosis of central retinal artery occlusion was proposed. The patient was treated with hyperbaric oxygen therapy. He was prescribed one cycle of Treatment Table 5 followed by four cycles of Treatment Table 6

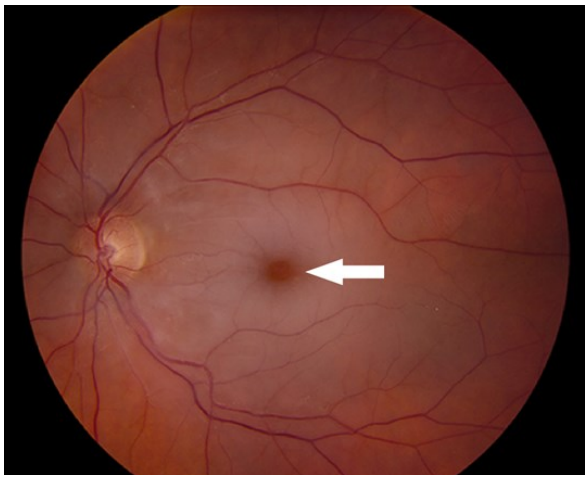

Figure 1: Right eye fundus photography of the diver showing cherry spot (white arrow) before hyperbaric treatment.

(US Navy Treatment Table) which was commenced seven hours after the onset of his symptom.[4] His right visual acuity had remarkably improved from 6/60 to 6/7.5 with no RAPD one day after completed the hyperbaric treatment. Light brightness and red saturation test were also comparable to the left eye and normal. Retina appeared healthy with flat macula and presence of vague fovea reflex. Three weeks after the event, he had no residual eye symptoms and his right eye vision had returned to 6/6 with normal ocular findings on examination.

DISCUSSION

Decompression illness results from both physical and biochemical sequelae of free phase inert gas precipitated by sudden a drop in ambient pressure. [5] In the case of recreational scuba diving not exceeding a non-decompression depth limit as in the case we reported, this is brought about by rapid ascend to surface as well as omitting the three minutes safety stop at five meters at the end of a dive. The safety stop in recreational diving is a standard practice to allow `off-gas' of excess nitrogen in the lung and soft tissue to prevent DCI. [3] Once DCS is precipitated, the bubbles have multiple physical and biochemical effects in the form of compression and

stretching of the surrounding structures as well as activation of complement cascade. [5]

Although scuba diving is considered a safe sport and the overall incidence of DCI in recreational diver of only 0.01-0.019%, the effect of CRAO as a manifestation of DCI has a significant function morbidity. [6] CRAO typically presents with painless, acute and often complete loss of vision and typical ophthalmologic changes occur within few hours or minutes of the event. However, in some cases the presentation may precede with prodromal visual disturbance symptoms and the severity is subtle if the supero-temporal branch is mainly involved. [6] The diagnosis is mainly by history with a dive profile that deviates from standard recommendation. Risk factors of DCI include high body fat content, previous history of DCI, dehydration, advancing age, patent foramen ovale, exercise, repetitive dive, multiple-day diving, deep diving and altitude exposure after a dive. [7] A comparatively shallow dive does not exclude the diagnosis entirely as evidenced from the case we reported. There is a similar report of the occurrence of arterial gas embolism in apnoea diving exercise in very shallow water with a depth of a mere 1.2 meter. [8] However, rapid ascend and omission of safety stop protocol should always alert a physician of a possibility of DCI. [9] The safety stop is a standard procedure in scuba diving to allow slow and adequate nitrogen release from tissues and the lungs. Other ophthalmic manifestations of decompressive sickness include redness of the eye from mask squeeze, orbital cellulitis, nystagmus, double vision and pain upon moving eye. [6]

The goal of treatment in CRAO is to increase retinal blood flow and to dilate its arteriole to improve blood supply to the retina, hence dislodging emboli and overcoming retinal spasm that may be present. [10] The definitive treatment of CRAO is hyperbaric oxygen therapy that promotes angiogenesis and fibroplastic activity of the hypoxic tissue. [10] In our patient, hyperbaric treatment was commenced relatively early with successful outcome.

CONCLUSION

In conclusion, a diagnosis of CRAO is a rare manifestation of DCI and should always be suspected in a diver with sudden painless monocular vision loss after a dive and a suggestive fundus examination. A detailed history of the patient's dive profile should be obtained and hyperbaric treatment should be initiated early.

DISCLOSURE OF INTEREST

The authors declare that they have no conflict of interest concerning this article.

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