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Multimodal imaging analysis of retinal and choroidal microvascular abnormalities in a case of ocular decompression sickness

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ABSTRACT

Purpose: Decompression sickness can result in a variety of ocular manifestations due to barotrauma. The retinal complications of this illness are less defined. In this case report, we describe a case of pigment epithelial detachment (PED) with retinal and choroidal microvasculature changes secondary to ocular decompression sickness in a scuba diver.

Observations: The parafoveal serous pigment epithelial detachment resulted in a scotoma associated with a kaleidoscope-like visual disturbance and mildly decreased vision which started immediately after the accident. Multimodal imaging was obtained revealing a serous PED without exudation, pooling of dye on fluorescein angiography, and decreased flow signal on optical coherence tomography angiography (OCT-A) in the deep capillary plexus and choriocapillaris in the area of the PED. Over the course of three months, the serous PED spontaneously resolved leaving behind subtle retinal pigment epithelium (RPE) alterations. Visual acuity also improved over the same time period however the visual disturbance had not completely resolved at the date of last follow-up.

Conclusions and importance: The imaging findings, temporal association with the diving accident, and short timeframe to resolution of this PED favor an etiology related to ocular decompression sickness. PED formation in this context may be secondary to 1) RPE dysfunction due to endothelial cell damage from free radicals and 2) choroidal ischemia resulting from gas emboli. To our knowledge, this is the first reported case where OCT-A has been used to demonstrate choroidal ischemia in ocular decompression sickness. The patient received hyperbaric oxygen treatments for several weeks following the accident which may have contributed to the rapid resolution of the PED supporting the role of choroidal ischemia in its pathogenesis.

1. Introduction

Decompression sickness (DCS) is a potentially life-threatening illness which is caused by barotrauma from dissolved gases forming bubbles in the bloodstream and tissues of the body.[1] This is typically due to ambient pressure changes, such as during a rapid ascent from a dive or from flying at high altitudes in an unpressurized aircraft. During a dive, gases exist in equilibrium under pressure in their dissolved forms in the tissues of the body. A rapid ascent causes these gases (primarily nitrogen) to exit tissues and form bubbles, which can result in vascular occlusions, inflammation, and tissue damage (involving soft tissues, skin, lungs, and the brain). Ocular decompression sickness (ODS) is a subset of DCS in which the eye sustains barotrauma from the rapid decrease in ambient pressure and the liberation of gas bubbles. Various ocular manifestations have been reported including subconjunctival hemorrhage, refractive changes, retinal vascular occlusions, accommodative

Abbreviations: PED, pigment epithelial detachment; RPE, retinal pigment epithelium; DCS, decompression sickness; ODS, ocular decompression sickness; HBOT, hyperbaric oxygen therapy; OCT, optical coherence tomography; FA, fluorescein angiography; OCT-A, optical coherence tomography angiography; IS, inner segment; OS, outer segment; FMD, flow mediated dilation; VEGF, vascular endothelial growth factor; PDT, photodynamic therapy.

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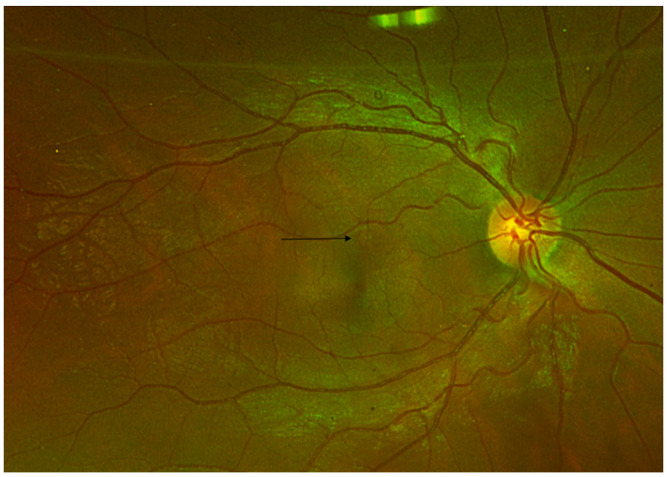

Fig. 1. Fundus Photograph Pseudocolor wide-field fundus image of the right eye revealing a small pigment epithelial detachment (black arrow) located in the superonasal parafoveal region.

and convergence insufficiency, and orbital hemorrhage.[2] The most common symptoms are decreased vision, diplopia, and scotomata.[3] There is little reported regarding specifically the choroidal and retinal complications of ODS. In this paper, we seek to address this by reporting a case of a symptomatic serous pigment epithelium detachment in a scuba diver after an episode of decompression sickness.

2. Case report

A 39-year-old male commercial scuba diver presented to the West Virginia University Eye Institute with a kaleidoscope-like visual disturbance and blind spot in his right eye resulting in decreased vision. These symptoms started immediately upon surfacing following an accidental rapid ascent during a dive 2 weeks prior to presentation. This resulted in decompression sickness, which was his first incident of DCS in his 11-year diving career. The patient experienced nystagmus, bilateral shoulder pain radiating to his jaw and teeth, and numbness and paresthesia of his bilateral upper extremities. The patient underwent hyperbaric oxygen therapy (HBOT) using the US Navy recompression treatment tables for patients with pain-only decompression sickness.[4] His pain improved significantly after the initial HBOT, but he was advised to continue treatment due to residual right upper extremity weakness and temperature dysregulation, and nystagmus. He completed two additional HBOT treatments.

On presentation to our institution, the patient's nystagmus and systemic symptoms had resolved, however he reported persistent kaleidoscope-like visual disturbance and a scotoma. Uncorrected visual acuity for the right eye was 20/40, and the left eye was 20/15. Intraocular pressure, pupillary reflexes, and extraocular motility were normal in both eyes. Slit lamp examination of the anterior segment of both eyes was unremarkable. The fundus examination of the right eye revealed a small pigment epithelial detachment located superonasal to the foveal center without hemorrhage, exudate, or subretinal fluid. The fundus examination of the left eye was unremarkable.

Multimodal retinal imaging was obtained including fundus photographs, spectral-domain optical coherence tomography (OCT), fluorescein angiography (FA), and optical coherence tomography angiography (OCT-A). The aforementioned imaging of the left eye was normal. The widefield pseudocolor fundus photographs of the right eye confirmed the clinical examination findings and did not reveal any additional pathology (Fig. 1). The near infrared reflectance image of the right eye revealed an area of hyporeflectivity superonasal to the foveal center which corresponded to the small solitary serous PED on the OCT raster scan (Fig. 2). A Heidelberg Spectralis 55-degree FA was obtained which demonstrated pooling in the area of the serous PED. No leakage was noted (Fig. 3).

The patient was reevaluated at our institution three months following the initial consultation and it was noted that the PED had resolved spontaneously. There were RPE alterations with mild hyporeflectivity of the IS/OS/RPE complex on OCT in the area of the previous PED (Fig. 4). OCT-A was performed at this visit showing decreased flow signal on the deep capillary plexus and choriocapillaris slabs in the area where the PED was located (Fig. 5). The patient noted that the kaleidoscope-like visual disturbance had improved but had not completely resolved. His uncorrected visual acuity had improved to 20/25 in the right eye.

3. Discussion

Ocular decompression sickness is a subset of decompression sickness, or "the bends". The manifestations of ODS are variable and are related to the rapid change in ambient pressure as gas bubble emboli enter the orbit and choroidal circulation. Subconjunctival and orbital hemorrhages may be present and arise from the sudden decrease in pressure

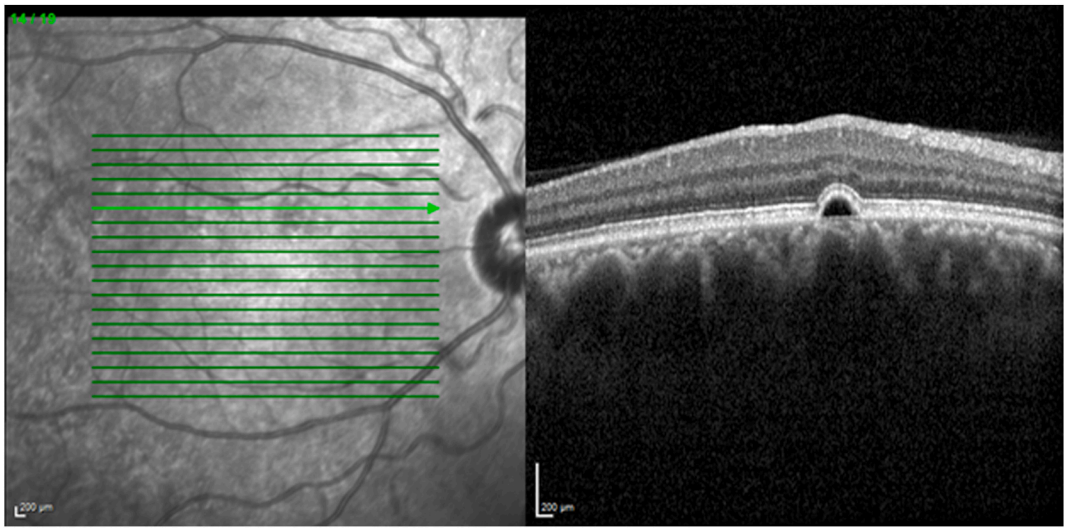

Fig. 2. Spectral domain optical coherence tomography OCT image demonstrating normal foveal architecture with a small serous PED just superonasal to the foveal center.

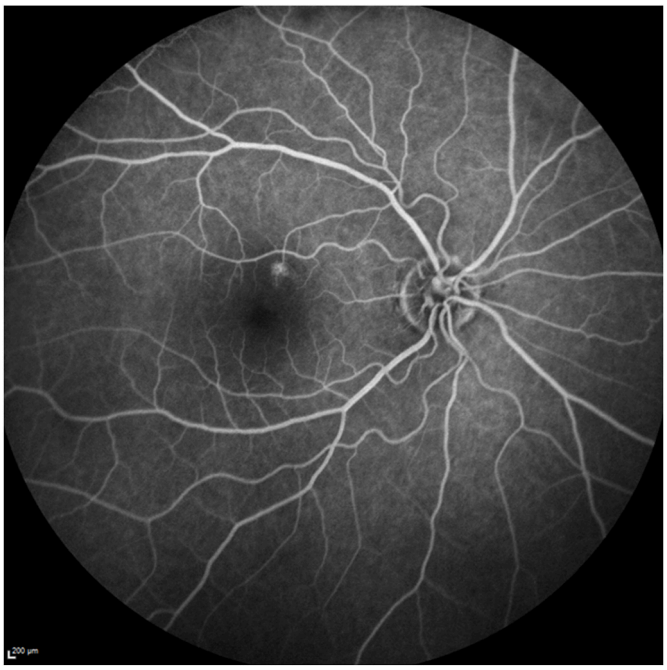

Fig. 3. Fluorescein angiography Late phase FA showing hyperfluorescence due to pooling within the serous pigment epithelial detachment.

leading to rupture of vessels.[5] Orbital hemorrhages have also been postulated to be caused by the negative pressure exerted by the divers' face mask, which creates a vacuum that causes suctioning of orbital tissues.[6] Changes within the crystalline lens from high pressure environments are speculated to be the cause of the refractive changes seen in ODS; typically, a myopic shift is present.[7] Neurologic sequelae of DCS include nystagmus, as well as accommodative and convergence insufficiency.[8] The effect of ODS on the choroid and retina are not well defined as there is scarce literature on the topic.

Following a deep-sea dive, it has been demonstrated that there is a decrease in the subfoveal choroidal thickness.[9] It is hypothesized that during a dive, vasoconstriction of the choroidal vessels occurs with subsequent delayed flow mediated dilation (FMD) following the dive due to endothelial cell dysfunction leading to arterial stiffness. Diving is known to increase the partial pressure of oxygen leading to oxidative stress and free radical formation resulting in damage to endothelial cells. Insufficient relaxation of the choroidal smooth muscle cells may also play a role in the reduction of subfoveal choroidal thickness. We speculate that free radical formation within the choriocapillaris may be transmitted to the retinal pigment epithelium (RPE) resulting in cell damage. An additional possible mechanism for PED formation may involve vascular gas emboli inducing choroidal ischemia.[10] The combined effects of endothelial cell dysfunction and choroidal ischemia may be the driving factors for the development of pigment epithelial detachments.

A pigment epithelial detachment (PED) is a separation of the basal lamina of the retinal pigment epithelium from the inner collagenous layer of the Bruch's membrane. The space within can be occupied by serous exudate, blood, fibrovascular tissue or drusenoid material. PEDs have been associated with various ocular diseases such as age-related macular degeneration, central serous chorioretinopathy, polypoidal choroidal vasculopathy, myopic choroidal neovascularization, Vogt-Koyanagi-Harada (VKH) disease, retinal angiomatous proliferation and many other ocular conditions.[11] The association of PEDs with ocular decompression sickness has not been well described in the literature. There have been some studies evaluating divers with fluorescein angiography which have had differing results. One study comparing 55 Royal Navy divers to 24 non-divers showed no difference in foveal avascular zone size or RPE defects on angiography[12]; another analysis of 84 divers however demonstrated lower foveal retinal capillary density and microaneurysms with areas of capillary nonperfusion in the diver group compared to the control group.[13]

The management of PEDs varies based on the underlying etiology and the type. Vascularized PEDs are primarily treated with anti-VEGF intravitreal injections. Treatment of serous PEDs depends on the etiology and may involve anti-VEGF intravitreal injections, photodynamic therapy (PDT), and/or observation.[10] In our case, the serous PED was not leaking on angiography. The decrease in vision was likely related to the lesion's proximity to the foveal center. The presumed, mild decreased flow signal in the deep capillary plexus and choriocapillaris demonstrated on OCT-A supports the hypothesis that choroidal ischemia may have been implicated in the development of the PED. Furthermore, the onset coinciding with the diving accident and the spontaneous resolution with observation also favor the idea that this serous PED was a manifestation of ocular decompression sickness. While a possible explanation for his findings, central serous chorioretinopathy seemed less likely given the temporal association of his symptoms with the rapid ascent and the absence of classic risk factors for central serous chorioretinopathy such as stress, or use of steroid or sympathomimetic

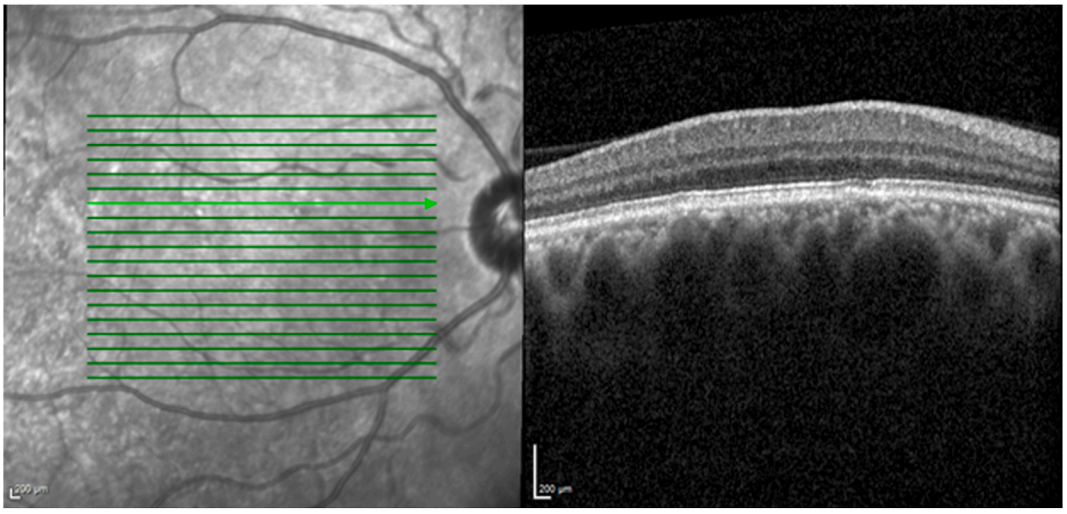

Fig. 4. Follow-up spectral domain optical coherence tomography OCT image demonstrating resolution of the serous pigment epithelial detachment and IS/OS/RPE complex alterations.

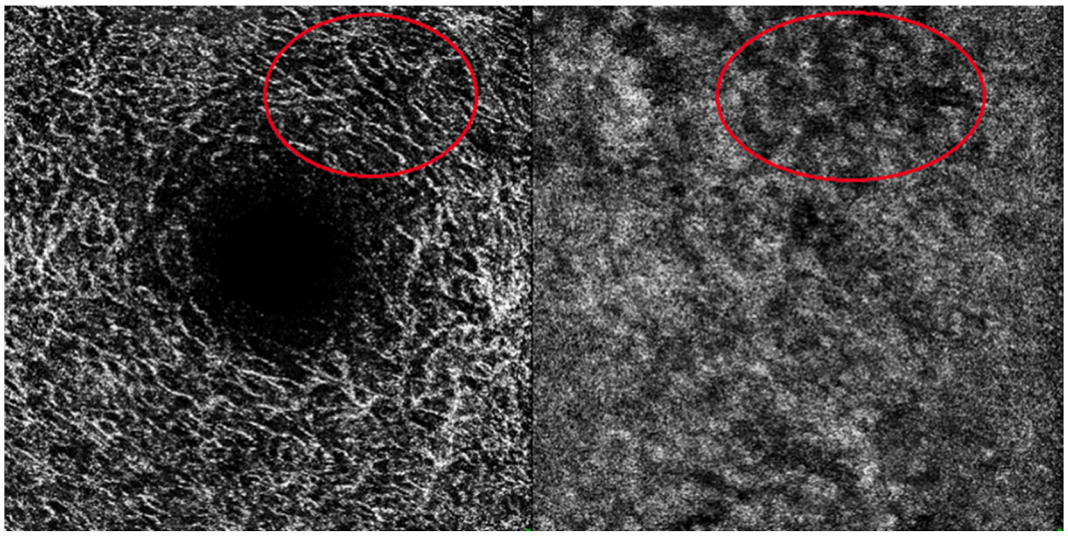

Fig. 5. OCT angiography Image of the deep capillary plexus (left) revealing mild decreased flow signal in the area of the PED (red circle). OCT angiography of the choriocapillaris (right) also demonstrating subtle flow signal void in the area of the PED (red circle). (For interpretation of the references to color in this figure legend, the reader is referred to the Web version of this article.)

drugs.

In summary, we present a novel presentation of ocular decompression sickness from a symptomatic serous PED in a patient with decompression sickness following a scuba diving accident. In addition to previously described hypotheses, we have proposed choroidal ischemia as a factor in the underlying pathophysiology of this condition and evaluated choroidal perfusion in this case with OCT-A. Further research should be done to better understand retinal and choroidal complications of decompression sickness. Routine ocular screening of divers with DCS should be considered as part of the initial work-up. Decompression sickness treatment protocols may be influenced by the presence of ocular findings.

CRediT authorship contribution statement

Christine Clavell: Writing – review & editing, Writing – original draft, Investigation, Formal analysis, Conceptualization. **James Dossett:** Writing – review & editing, Investigation, Formal analysis, Conceptualization. **Sanya Yadav:** Writing – review & editing. **Ami Patel:** Writing – review & editing. **L. Carol Laxson:** Writing – review & editing. **Ghassan Ghorayeb:** Writing – review & editing, Supervision.

Patient consent

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

Authorship

All authors attest that they meet the current ICJME criteria for Authorship.

Funding

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Declaration of competing interest

The authors declare the following financial interests/personal relationships which may be considered as potential competing interests: The authors have no conflict of interest.

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