

Central Retinal Artery Occlusion after Blunt Trauma Responsive to Hyperbaric Oxygen Therapy

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Abstract

Purpose: To report a case of an indirect traumatic central retinal artery occlusion responsive to hyperbaric oxygen therapy.

Methods: Healthy fifty-five-year-old man presented to the emergency department with sudden and painless vision loss for two hours in his left eye after blunt trauma by pressured air jet that blew out from a manometer. Fundus examination demonstrated diffuse pallor along the macula with subtle cherry-red spot. Central retinal artery occlusion (CRAO) was diagnosed. The patient was immediately referred to hyperbaric oxygen treatment (HBOT) (three sessions at 2.4 atmosphere absolute, over three days) and VA improved to 20/50. Due to COVID-19 restrictions, additional sessions were not performed. One month later, his BCVA was light perception. Optic disc and macular atrophy developed.

Conclusion: CRAO is a commonly blinding ocular emergency. Although highly associated with systemic conditions, it may relate to traumatisms. There are few reported cases of traumatic CRAO, and none successfully treated with HBOT. This case highlights its potential in CRAO management.

Keywords: Central Retinal Artery Occlusion (CRAO); Hyperbaric Oxygen Treatment (HBOT); COVID-19

Introduction

Central retinal artery occlusion (CRAO) is a potentially devasting ophthalmological emergency. Patients often present with profound visual acuity impairment within minutes that will be irreversible for most of them. More than half of cases have final visual outcomes of no light perception [1].

The great majority of CRAO are associated with an emboly originating from atheromatous disease of the carotid artery [2]. As an embolic event, it is mostly associated with cardiovascular disorders such as hypertension, diabetes mellitus and ischemic heart disease [3]. Other associated diseases can be: giant cell arthritis, sickle cell disease, hypercoagulability states, autoimmune disorders. Traumatic occlusions usually follow head contusions but there are few reported cases in the literature [4].

Retina is a metabolic active and highly oxygen demanding tissue. When blood supply is interrupted for longer than the "ischemic penumbra" (four hours), the damage can be irreversible requiring early treatment [5,6].

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The treatment modalities for CRAO, apart from etiology, have relatively limited success. Patients are generally treated conservatively with ocular massage and topical pressure-lowering medications. More invasive strategies such as fibrinolysis and arteriotomy were proposed. However, no treatment modality itself proved to be effective yet. Although hyperbaric oxygen therapy (HBOT) was reported to improve the outcomes the CRAO by numerous case series, it is still underused. Weinberger, *et al.* reported visual acuity improvement in 13 of 21 patients [7]. Cope., *et al.* reported an improvement in 74% of patients, whereas 53% improved two or more lines in Snellen scale [8].

Purpose of the Study

The purpose of this paper is to report a case of traumatic central retinal artery responsive to HBOT.

Case Report

A healthy 55-year-old man presented to our emergency department with a sudden and painless vison loss for two hours in his left eye after blunt trauma by pressured air jet that blew out from a manometer.

On examination, the best corrected visual acuity (BCVA) was 20/20 in the right eye and light perception in the left eye. Intraocular pressure (IOP) was 13 mmHg bilaterally. Extraocular movements were unimpaired on both eyes. The left pupil showed an afferent pupillary defect. Anterior segment examination was unremarkable and there were no visible signs of globe rupture.

Fundus examination of the right eye was normal. The left eye revealed an oval area of pallor along the macula with a subtle cherry-red spot with retinal vessel attenuation (Figure 1). The optic disc was normal in coloration, structure and vascular settings. No retinal hemor-rhages, tears and detachment were seen.



Figure 1: Left eye fundus at initial presentation. Oval area of pallor along the macula with a subtle cherry-red spot. Optic disc unremarkable.

Optical coherence tomography (OCT) showed retinal edema with hyperreflectivity of inner retina, sparing the outer layers. Computed tomography (CT) showed no signs of fracture, hematoma, emphysema and orbital tissue edema.

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Central retinal artery occlusion (CRAO) was diagnosed. The patient was initially treated with ocular massage, eyedrops to lower IOP, intravenous mannitol and oral acetazolamide and was immediately referred to hyperbaric oxygen treatment. He completed three sessions of HBOT at pressure of 2.4 atmospheres absolute over three days in Underwater and Hyperbaric Medicine Center, Armed Forces Hospital (Lisbon). On the fourth day, his left eye visual acuity improved to 20/50 although the fundoscopic examination did not change.

Unfortunately, due to COVID-19 restriction measures, no additional sessions were programmed. One month later, his BCVA was light perception. Optic disc and macular atrophy developed. Fluorescein angiography did not reveal any alteration.

Routine blood investigations, including erythrocyte sedimentation rate, electrocardiogram and carotid ultrasound were unremarkable.



Figure 2A: Left eye fundus one month after HBOT. Macular and optic disc atrophy.



Figure 2B: OCT showing macular atrophy.



Figure 3: Fluorescein angiography one month after HBOT. No evident alterations.

Discussion

Central retinal artery occlusion is a rare ophthalmological condition, with an estimate incidence around 1 in 10,000 to 1 in 100,000 [9]. Although CRAO is the ocular analogue of stroke and most patients have a cardiovascular condition behind, there are some reports of traumatic occlusion following eyeball or head contusion in healthy patients [4,10].

The retina is nourished by two main sources: the inner layers from central retinal artery (CRA) and the outer layers from ciliary arteries coming from choroid circulation. The central fovea is also nourished by choroidal circulation. In CRAO, because choroidal circulation is unimpaired, the pathognomonic "cherry-red spot" against the diffuse retinal whitening appears.

Several mechanisms underlying traumatic CRAO have been proposed. The CRA could be compressed by force transmitted to the orbit, inducing focal vasospasm or retinal vessel stretching with endothelium damage and thrombus formations [4,11-13]. The CRA could also be compressed by hematoma, by air if emphysema is present or by increased IOP from periorbital soft tissue swelling. CT excluded all these conditions.

On other side, it was demonstrated that the arterial walls appear to be swollen as a result of edema and inflammation in response to ischemic insults [14]. Upregulation of contractile receptors, including ET-1 receptors, following ischemic damage is a well-establish mechanism that exacerbates the tissue damage even after reperfusion is established and we believe to be the same mechanism underlying our patient.

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Apart from etiology, every CRAO should be managed in order to promptly identify the occlusion, restore circulation and oxygen supply and prevent further recurrences. There are few studies regarding CRAO management. The ischemia effects could only be reversed in the first four hours and, as such, in CRAO "time is vision".

Ocular massage should be readily performed as it is an easy maneuver that facilitates aqueous outflow, embolus migration and improvement of retinal perfusion [15]. Additionally, IOP should be lowered with topical drops and systemic acetazolamide or mannitol [16]. Other invasive modalities, such as systemic fibrinolysis with tissue plasminogen activator (t-PA) [17] and Nd:Yag laser or surgical arteriotomy [18], are described but did not show clinical benefit.

Hyperbaric oxygen treatment (HBOT) is based on an algorithm published in Journal of the Undersea and Hyperbaric Medical Society [19] and the European Committee of Hyperbaric Medicine (ECHM) indicate that CRAO have Type 2 recommendation and level C evidence for HBOT [20].

HBOT was shown to provide favorable outcomes in CRAO in a review conducted by Olson and Lentz [21]. Menzel-Severing, *et al.* compared VA outcomes in 51 patients that underwent HBOT with 29 patients who did not and found an average improvement of 3 lines of vision in the former versus 1 line in others. [8,22,23]. It relies on the fact that retina has a dual blood supply and if the oxygen levels in the choroidal circulation increases to a sufficient degree, it can diffuse to inner retinal layers and allowing their viability [24]. Under normobaric conditions the choroidal circulation supplies 60% of oxygen needed by retina, which increases to 100% under hyperbaric conditions [19]. HBOT decreases the edema and preserve compromised tissue adjacent to ischemia. Gaydar, *et al.* showed that HBOT diminished cell loss from 58% to 30% which was related to increased survival of cells in the retinal inner layers [25].

Our patient had an increase in BCVA after HBOT. This finding may give additional support the early benefit following CRAO. However, the number of sessions show be considered. Despite HBOT improves the edema, inflammatory mediators remain being released what perpetuates the edema and possibly lead to secondary occlusive events what we believe that happened in our patient. So, it could be beneficial to keep HBOT longer.

Despite the obvious case-effect from trauma, we also requested a systematic investigation in order to rule out other possible underlying cardiovascular disorders that could eventually lead to fellow eye CRAO, stroke or myocardial infraction if not treated.

Conclusion

CRAO is an ocular emergency and requires prompt intervention due to its poor prognosis. Unfortunately, there are no well-established treatment guidelines approved yet. There are little more than 20 clinical reports of traumatic CRAO, making their management even more challenging. Our report highlights that HBOT is a potential and encouraging modality in traumatic CRAO secondary to vasospasm mechanisms.

Disclosure Statement

The authors have no conflicts of interest to declare.

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