

Geç Dönemde Başvuran Santral Retinal Arter Oklüzyon Vakasında Hiperbarik Oksijen Tedavisi Sonrası Görme Artışı

Visual Improvement After Hyperbaric Oxygen Therapy in a Late Referral Central Retinal Artery Occlusion Case

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ÖZ

40 yaşında bayan hasta Karaman devlet hastanesi göz kliniğine sağ gözde 9 gün önce başlayan akut görme azalması ile başvurdu. Başvuru sırasında hastanın sağ gözde görme keskinliği el hareketleri, sol gözde ise düzeltilmesiz 10/10 düzeyinde idi. Fundus muayenesinde superior arka kutbun korunduğu santral retinal arter oklüzyonu (SRAO) görünümü mevcut idi. Hastanın yapılan fundus floresans anjiyografisinde (FFA) retinal arteriyel dolaşımın rekanalize olduğu ve siliyoretinal arterin açık olduğu tespit edildi. Hastanın ayrıntılı anemnezi sorgulanarak hematoloji ve kardioloji ile konsulte edildi, gerekli etiyolojik tetkikleri yapıldı. Hasta aynı gün santral retinal arter oklüzyonu tanısıyla hiperbarik oksijen tedavisi için ilgili merkeze yönlendirildi. 20 seans hiperbarik oksijen tedavisi sonrası 6. hafta kontrolünde SRAO olan gözde düzeltilmesiz 10/10 düzeyinde görme elde edildi.

Anahtar Kelimeler: santral retinal arter, oklüzyon, hiperbarik oksijen

ABSTRACT

40 years old female patient was presented to Karaman State Hospital (Karaman/Turkey) eye clinic with acute vision loss in the right eye, beginning 9 days before. The patient's initial visual acuity was at the level of hand movements in the right eye and 10/10 in the left eye. There was a marked central retinal artery occlusion (CRAO) view with preserved superior posterior pole in right eye fundus examination. Fundus fluorescein angiography (FFA) revealed central retinal artery recanalization, and patent cilioretinal artery. Hematological, cardiovascular and other etiologic examinations were also performed. Subsequently patient redirected to the hyperbaric oxygen therapy with the diagnosis of central retinal artery occlusion. Visual acuity improved at 10/10 level, after 20 sessions of HBO therapy at 6th week control in the right eye.

Key words: central retinal artery, occlusion, hyperbaric oxygen

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INTRODUCTION

Central retinal artery occlusion (CRAO) is a rare clinical condition and causes an acute dramatic reduction in visual acuity. The inner retinal layers are nourished from the retinal and the outer one-third part are from the choroidal vascular circulation. Therefore, the inner retinal layers are affected in the CRAO.

If the blood partial oxygen pressure is increased adequately, oxygen can diffuse from the choroidal circulation to the inner retinal layers. In this way, retinal functions can be recovered. In some cases, normobaric oxygen support is needed, while in some cases, there is a need for hyperbaric oxygen support.

The prognosis in CRAO may vary depending on whether the presence of retinal and choroidal occlusion, the degree of occlusion and the presence of the patent cilioretinal artery. The cilioretinal artery is preserved in 15-30% of cases.¹ Thus, central macular vision is preserved in an amount of CRAO cases. The goal is to provide adequate oxygen support before the development of the irreversible retinal damage and vision loss and to restore retinal functions and vision.

CASE REPORT

Before the case presentation, the "Informed consent" form obtained from the patient. A 40-year-old female patient has presented with acute visual loss beginning 9 days ago in the right eye. As learned from the patient's medical questionnaire, she had essential hypertension and any other systemic or ocular disease and used oral antihypertensive, no oral anticoagulant. The uncorrected initial visual acuities of the patient were at the level of hand movements in the right eye and 10/10 in the left eye (with Snellen scales). Intraocular pressure (IOP) was measured 14 mmHg in both eyes with Goldman applanation tonometry. The direct light reflex in the right eye was impaired. Anterior segment examination was bilaterally unremarkable. Fundoscopic examination showed a view of CRAO with a preserved superior posterior pole in the right eye after pupillary dilatation with 2.5% of phenylephrine and 0.5% of tropicamide (**Figure 1**).

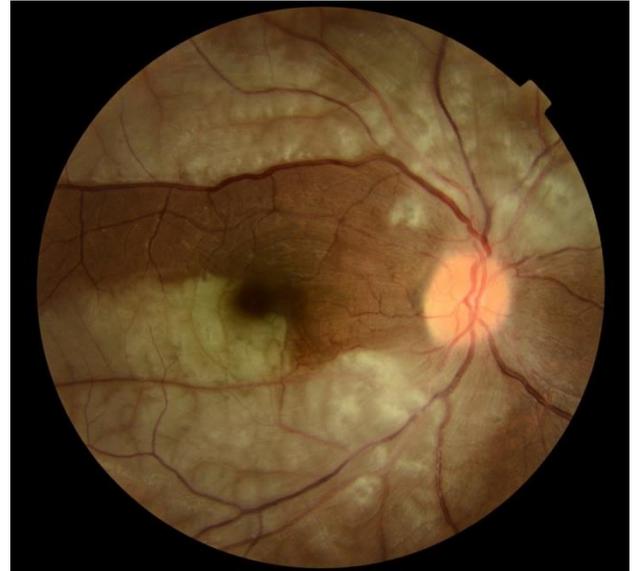


Figure 1. Initial color fundus photograph of the patient.

Acute CRAO treatment procedures were not performed in this late-referral patient. FFA images revealed recanalized arterial circulation and the patent cilioretinal artery in the right eye (**Figure 2**). After cardiology and hematology consultation, 100 mg/day acetylsalicylic acid was prescribed to the patient. Renal and carotid Doppler ultrasonography and other hematological examinations were also unremarkable.

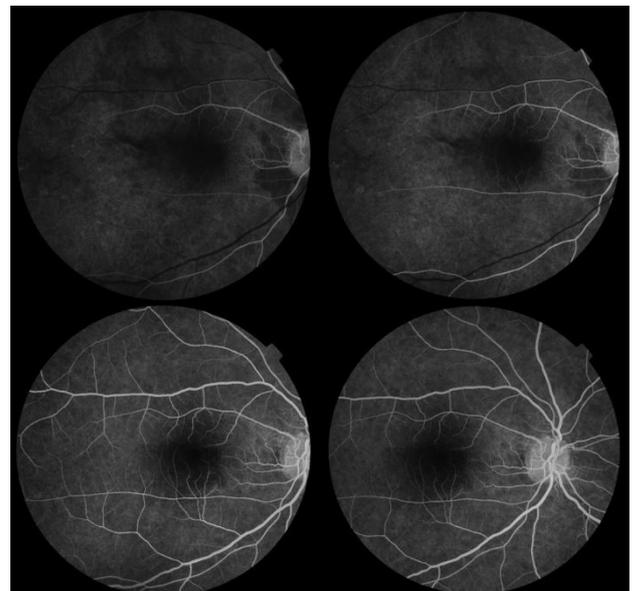


Figure 2. Initial FFA images of the patient

On the same day, the patient was referred to the hyperbaric oxygen therapy center. The patient underwent a total of 20 sessions hyperbaric oxygen

therapy, two sessions per day for the first week and one session per day for the next 6 days. The procedure was performed in multi-person pressure chambers, each with a pressure of 2.4 ATA, in 120-minute sessions.

The best corrected visual acuity (BCVA) was 6/10 in the 3rd-week visit. At the 6th week visit, the BCVA was at the 10/10 level, anterior segment and fundus examination were normal, IOP was 16 mmHg, direct and indirect light reflexes were normal.

Color fundus images, optical coherence tomography (OCT) records were taken at the 3rd-month follow-up, also visual field test was performed. Besides the extensive peripheral concentric narrowing, the central visual field was preserved in the perimetry (Figure 3,4).



Figure 3. The color fundus photo of the patient at 3rd month visit after hyperbaric oxygen therapy

A marked ganglion cell-inner plexiform layer thinning was observed in the nasal quadrant comparing with temporal quadrant in the OCT maps (Figure 5).

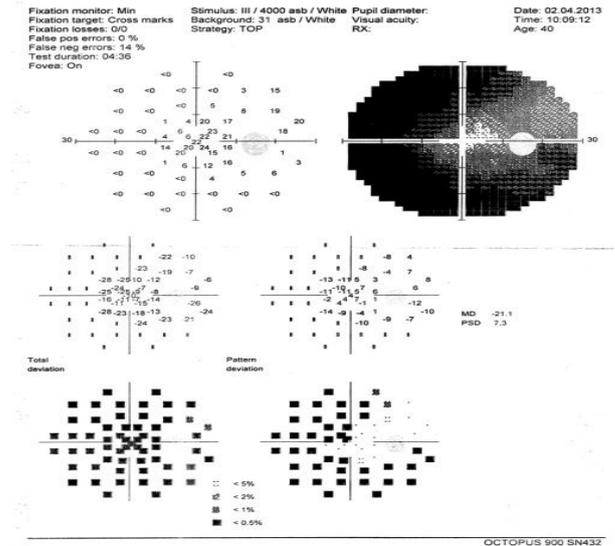


Figure 4. The perimetry analysis of the patient at 3rd month visit after hyperbaric oxygen therapy

The BCVA was persisted at the 10/10 level and there was no change in the visual field and OCT records at the 6th month's follow-up.

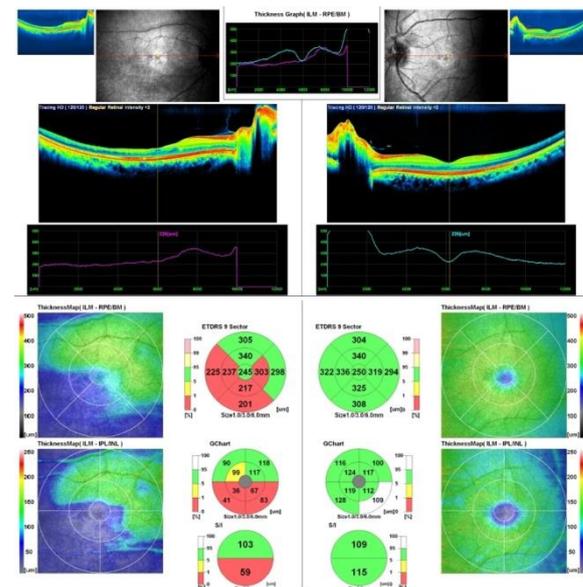


Figure 5. The OCT analysis of the patient at 3rd month visit after hyperbaric oxygen therapy

DISCUSSION

CRAO is a rare devastating eye emergency presented with sudden, painless vision loss. Various etiologies such as thrombosis, embolism, vasospasm, giant cell arteritis can affect the treatment algorithm and the prognosis. The retina is the most active tissue in the body in terms of

oxygen requirement. (13ml / 100g / min). For this reason, sufficient oxygen support must be provided in the CRAO until retinal circulation recanalizes. Vascular recanalization usually occurs within 72 hours after occlusion.

The ganglion cell-inner plexiform layers that nourished from retinal circulation lose their vitality in the CRAO. The visual prognosis is poor despite the emergency interventions those are ocular massage, IOP lowering treatments, paracentesis, vasodilators, and diuretics.²⁻⁴ Hayreh et al.^{2,5} argued that these treatments are not effective except for the hyperbaric oxygen therapy. Moreover, Hayreh² reported that the final vision was at finger counting levels in more than 80% of the patient group without receiving hyperbaric oxygen support and the absence of transient occlusion and patent cilioretinal artery in the natural course of the CRAO. In addition, only 1.5% of this patient group had a visual gain at 5/10 levels.²

If the blood partial oxygen pressure is increased sufficiently by hyperbaric oxygen support, retinal layers can ensure oxygen demand from the choroidal circulation and maintain its vitality. Animal studies have shown that retinal layers can be fed with choroidal diffusion even in the complete retinal arterial occlusion.¹ In normoxic conditions 60% and in hyperbaric conditions 100% of the retinal oxygen demand can be supported by the choroidal circulation.⁶

Hayreh et al. classify the CRAO into 4 subtypes: Non-arteritic (NA) CRAO, NA-CRAO with patent cilioretinal artery, Arteritic CRAO (secondary to Giant cell arteritis, cilioretinal artery +/-), Transient NA-CRAO (cilioretinal artery +/-).⁶ These four CRAO clinical types, cilioretinal artery occlusion, location and degree of vascular occlusion are important parameters affecting visual prognosis. Hayreh noted that the visual gain in late referral patients (7th day and after and with finger count vision levels) may vary according to the CRAO subtype and most prominent vision increase was observed in 82% of patients with transient NA-CRAO (cilioretinal artery +) subtype.⁶

An important point in treatment is the timing of hyperbaric oxygen therapy. Hyperbaric oxygen therapy should be initiated before irreversible

retinal damage occurs. Hayreh's work on rhesus monkeys has shown that cessation of 105 minutes of retinal blood flow leads to irreversible retinal damage. Thus, Hayreh determined the critical time threshold for recovery of retinal functions to be 97 minutes.⁷ Hyperbaric oxygen support should be provided immediately in patients with CRAO who are referred within the first 24 hours.

There are limited late referral cases in the literature with marked visual gain. Aktaş et al. reported a case, 26-year-old male patient who had symptoms starting at 20 hours ago before the presentation and diagnosed with isolated cilioretinal artery occlusion.⁸ Initial visual acuity was 20/200 and hyperbaric oxygen therapy was started at 22nd hour in this patient. After 5 sessions (3 days) hyperbaric oxygen therapy, the visual acuity remained unchanged, and after 20 sessions in total, the visual acuity level reached 10/10.⁸

In another late referral 81-year-old CRAO case with mitral valve dysfunction, visual acuity was improved from 20/640 to 20/50 after eight sessions of hyperbaric oxygen therapy which started 12th day.⁹ Gokce et al. reported a case diagnosed with cilioretinal artery and central retinal vein occlusion due to high altitude exposure. Hyperbaric oxygen therapy was started to the patient after the 11th day, and the initial visual was advanced from 10/20 to 10/10 after the treatment.¹⁰ In another case report in the literature, central retinal artery and posterior temporal ciliary artery occlusion were detected in FFA in a 70-year-old patient who presented with light perception vision in one eye. This patient was treated with carbogen (95% hyperbaric oxygen + 5% CO₂). At the end of 96 hours, spontaneous resolution has been achieved and vision progressed to 6/10 at the end of the first week.³

Miyake presented a case series comprising 53 patients with CRAO and 19 patients with branch retinal artery occlusion. Only 3 of these patients were able to receive hyperbaric oxygen therapy within the first 24 hours. Despite exceeding the critical time threshold for treatment, a significant visual gain was determined in the 44% of these patients. However, the presence of patent cilioretinal artery or transient occlusion were not stated in this case series.¹¹

Even in the complete CRAO the presence of residual retinal blood flows had been demonstrated in FFA images in some experimental and clinical studies. These residual blood flows may clarify the significant increase in visual acuity obtained in some patients with complete CRAO, even days or weeks after the treatment.^{2,12} The site of obstruction is also very important in terms of visual prognosis. Considering occlusions along the course of the optic nerve in the dural sheath, the pial and intraneural collaterals of the central retinal artery remain intact at the distal site of the occlusion and these collaterals constitute the source of retinal residual blood flows.¹³

We argued that a probable transient photoreceptor distress not resulting in complete necrosis due to the presence of a transient CRAO accompanying cilioretinal artery occlusion may explain the poor initial vision in our case presented at the 9th day. In addition, another cause of the significant increase in visual acuity in our patient may be due to retinal residual blood flow at the time of the occlusion. We can also mention that hyperbaric oxygen therapy, which is known to have beneficial effects on tissues exposed to ischemic stress, has a positive effect on the reversibility of pathology.¹⁴

Nevertheless, the most appropriate treatment approach is to initiate hyperbaric oxygen therapy as soon as possible. Emergency treatment procedures were not applied to our late referral patient. After performing the required examinations and consultations patient was scheduled for the hyperbaric oxygen therapy immediately. Considering poor initial visual acuity in our case report, the effect of hyperbaric oxygen supplementation on visual acuity is noteworthy. Finally, we argued that hyperbaric oxygen support should be provided in late-referral CRAO cases.

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