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Case report

CASE REPORT

Hyperbaric oxygen therapy for combined branch retinal artery and branch retinal vein occlusion

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Learning points for clinicians

Combined branch retinal artery occlusion (BRAO) and branch retinal vein occlusion (BRVO) is very rare and leads to sudden vision loss. Amaurosis fugax is a premonitory symptom. Hyperbaric oxygen therapy might be a feasible and effective treatment for combined BRAO and BRVO.

Introduction

Retinal vascular occlusion is a blockage of the retinal vascular system which results in sudden vision change. Simultaneous occlusion of retinal artery and vein has been reported in the past but very rare. The effective treatment for retinal vascular occlusion is still controversial. Recently, hyperbaric oxygen therapy (HBOT) has been reported to be an effective and safe option in treating patients with retinal artery occlusion.[1,2] However, the experience for treating combined branch retinal artery occlusion (BRAO) and branch retinal vascular occlusion (BRVO) is extremely rare. We will discuss the efficacy of HBOT in combined BRAO and BRVO. To the best of our knowledge, this is the first case with combined BRVO and BRAO who is treated successfully by HBOT.

Case

A 45-year-old male with uncontrolled hypertension presented to the emergency department due to sudden vision loss in his left eye with upper visual field loss. He had one episode of temporary visual loss (about 5 min) in the same eye about 2 weeks ago after he drove for a long time. On examination, his best-corrected visual acuity (BCVA) was 20/20 in the right eye and 20/200 in the left eye. The anterior segment was unremarkable, and the pupil reflex was normal without a relative afferent pupillary defect. Eye movements were free and full in all directions. Dilated fundus examination revealed mild disc edema, narrowing of the lower temporal branch retinal artery, small emboli at upper temporal branch retinal artery and retinal ischemia at the lower part of retina OS (Figure 1A). Fluorescein angiography demonstrated significant delayed filling of inferior temporal retinal arcade with nonperfusion at the lower part of retina, and total occlusion at lower temporal retinal vein (Figure 1B). Visual field test revealed profound upper visual field defect in the left eye (Figure 1C). Laboratory tests included hemogram, biochemistry, glycated hemoglobin, high sensitivity C-reactive protein and coagulant function were within normal limits. Carotid duplex sonography did not have evidence of significant stenosis or occlusion over the left carotid artery. The patient then received 20 sessions of HBOT. After 2 months, he had brighter visual field subjectively in his lesion eye but his BCVA remained 20/200. However, at the 5-month follow-up, his BCVA returned to 20/20 in the left eye and the visual field showed great improvement (Figure 1D).

Discussion

Combined BRAO and BRVO is a rare event that has not been well-studied. The exact etiology is unclear and the visual

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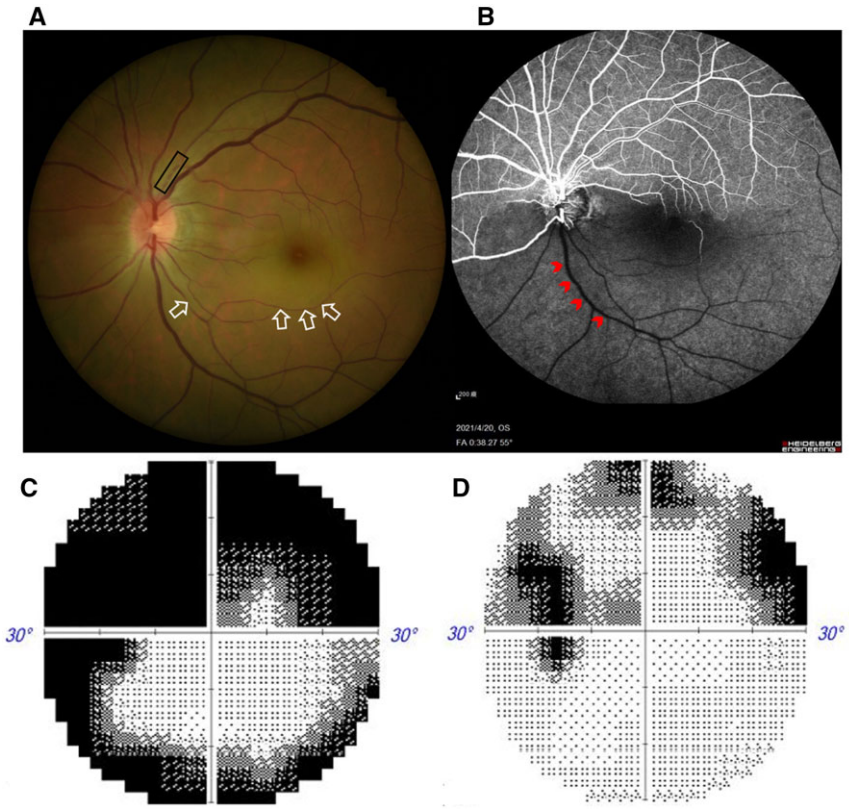

Figure 1. **(A)** Color fundus photograph revealed narrowing of lower temporal branched retinal artery (*white arrows*) and emboli at upper temporal branched retinal artery (*black rectangle*). **(B)** Fluorescein angiography demonstrated retina ischemia at lower arcade and total occlusion of lower temporal branched retinal vein (*red arrow heads*). **(C)** Visual field examination before the hyperbaric oxygen therapy showed profound longitudinal visual field defect. **(D)** Visual field examination after the hyperbaric oxygen therapy (about 5 months) revealed great improvement compared to (C).

prognosis is very poor. Most of the patients have multiple cardiovascular risk factors such as diabetes, hypertension, dyslipidemia and hyperhomocysteinemia.[3,4] One hypothesis is that hypertension causes distal compression of the retinal vein by the retinal artery at the crossing point, which leads to elevation of the branch retinal vein pressure and BRVO. This high pressure then returned to the crossing point and re-elevated the pressure of the branch retinal artery, eventually causes hemorrhage due to vascular breakdown and BRAO.[3] Amaurosis fugax usually occurs in patients who have vascular risk factors. Shuler *et al.*[5] reported two cases from recurrent amaurosis fugax and finally developed with CRVO. They concluded that amaurosis fugax may be as a premonitory symptom of CRVO, just the same situation in our patient.

Unfortunately, there is no proven effective therapy for this disease at present. Several treatments had been reported, including intravitreal antivascular endothelial growth factor injection, or scattered laser photocoagulation when retinal neovascularization or macular edema was observed. HBOT delivers 100% oxygen by a pressure greater than one atmosphere, increasing plasma oxygen transportation and diffusion, with positive effects in vascular perfusion and neuroprotection. HBOT was proved to increase the volume of oxygen delivered to the damaged or ischemic tissue until spontaneous or assisted reperfusion occurs. Its pathophysiological mechanisms are based on the retinal double vascular supply, in which the choroidal capillary vessels that supply the outer retina will bypass the occlusion and oxygenate the inner ischemic retina until spontaneous reperfusion occurs.[6]

Our patient had one episode of amaurosis fugax and uncontrolled hypertension. These are important risk factors for both BRAO and BRVO. He received 20 sessions of HBOT, and his BCVA and VF improved dramatically.

Conflict of interest. None declared.

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