Clinical Case Reports

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CASE REPORT

Migration of emboli in the retinal artery: case report

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Key Clinical Message

Branch retinal artery occlusion (BRAO) is a prevalent vascular occlusive disorder of the eye. In transient BRAO, an embolus temporarily blocks the vessel and then moves on resulting in recovery of blood circulation. In this case, we have documented migration of the embolus with recovery of perfusion of the retina and improvement of visual acuity in a patient with transient BRAO. Early diagnosis and fast action are important in the case of patients with retinal artery occlusion.

Keywords

Embolus, OCT-angiography, transient branch retinal artery occlusion (BRAO).

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Introduction

Retinal artery occlusion (RAO) is a prevalent, visually disabling, vascular occlusive disorder of the eye. There is usually a dramatic, sudden onset of visual loss, particularly in central RAO (CRAO), which was first described by von Graefe in 1859 [1].

Thromboembolus is the most common cause of RAO, which occurs at the narrowest part of the retinal artery. Calcific emboli usually do not migrate due to their coarse texture, whereas cholesterol and especially platelet emboli migrate more frequently [5]. For example, in 14.5% eyes with RAO, a plaque was seen at least once during multiple visits [5]; in 69%, the plaque was not successively visible at all of the visits [9].

Nonarteritic transient RAO accounts for 15–17% of RAOs and has the best visual prognosis [3, 4].

Case Presentation

A 65-year-old woman presented to the ophthalmology clinic in Hradec Kralove (Czech Republic) with a one-hour history of acute painless vision loss in her left eye. Her long-term self-medication included levothyroxinum 0.137 mg a day orally for hypothyroidism and flavonoidorum 500 mg for venous insufficiency of the lower extremities (history of venous thrombosis in 2011). The ocular history included amblyopia of the right eye and presbyopia. Best corrected visual acuity (BCVA) at presentation was 20/32 in the right eye, with amblyopia and light perception from right and from above in the left eye. There was an afferent pupillary defect on the left eye, but no ocular movement limitation, anterior segments were both otherwise normal, and applanation tonometry was normal. Dilated fundus examination of the left eye revealed a visible motile embolus in the superior branch of the retinal artery bifurcation (Fig. 1A), with whitish edema along the superior arcades and perifoveal ischemia (Fig. 2). The right eye was normal. Conventional structural optical coherence tomography (OCT Cirrus, Zeiss, Germany) of the left eye showed increased thickness of the inner layers of the involved retina due to retinal swelling (edema), caused by acute ischemic retinal infarction (Fig. 3). A diagnosis of branch retinal artery occlusion (BRAO) was made, and ocular massage, eye drops with dorzolamidum 20.0 mg and timololum

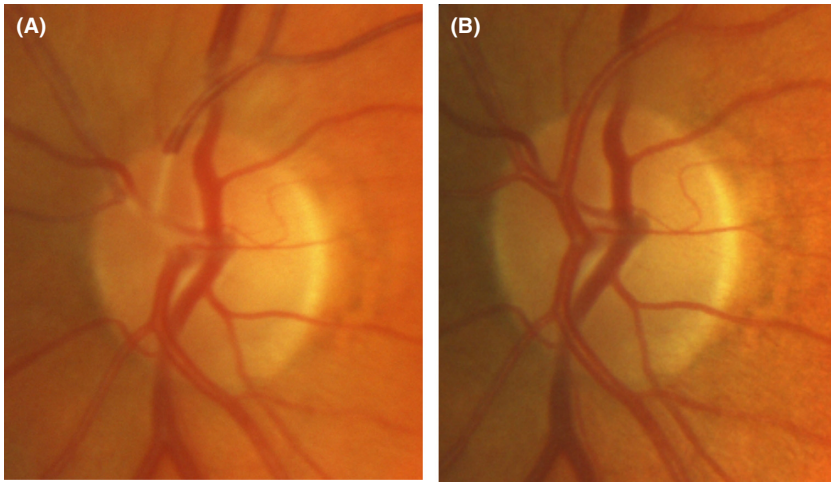

Figure 1. (A) Visible motile embolus in the superior branch of the retinal artery bifurcation. (B) Disappearance of the embolus.

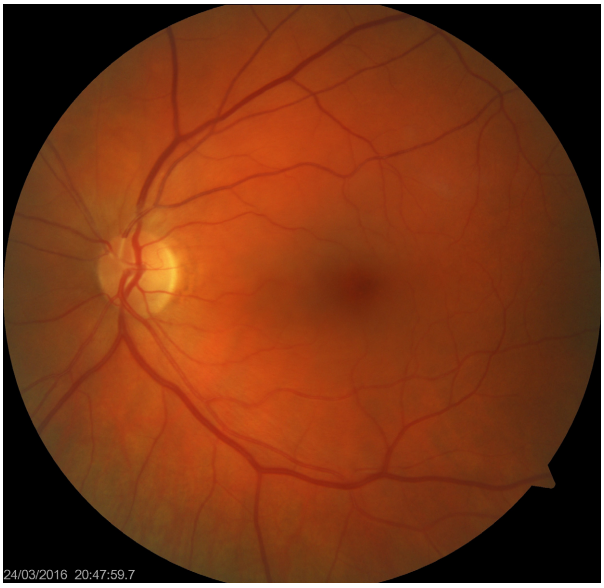

Figure 2. Perifoveal ischemia of the upper part of the macula.

5.0 mg, and acetazolamide 500-mg tablet in oral prescription were administered. The following laboratory tests were normal: complete blood count, kidney and liver function tests, lipid profile, blood clotting test, erythrocyte sedimentation rate, C-reactive protein, and homocysteine levels. Echocardiography and carotid ultrasound Doppler were normal. After a consultation with cardiologists, vasodilatatory therapy was administered, consisting of intravenous infusion of normal saline solution with Oxantil (etophylline 160 mg and theophylline 40 mg in 2 mL; HBM Pharma, Martin, Slovakia) and pentoxifylline 100 mg. The next day BCVA of the left eye had improved to 20/32, the embolus had resolved, and the afferent pupillary defect had also disappeared. Optical coherence tomography documented the gradual restoration of normal retinal configuration. But the en face optical coherence tomography angiography (OCT-A) (Zeiss AngioPlex) of the superficial retinal layer (Fig. 4A) still showed the hyperreflective area of retinal edema in the upper part of the macula. The superficial retinal OCT-angiogram revealed dilation of blood vessels due to decrease in vascular resistance (Fig. 4B). A diagnosis of transient BRAO was made, and antiplatelet therapy with acidum acetylsalicylicum 100 mg once daily was administered. Visual field testing of the left eye after 1 month revealed a small scotoma of the lower part of the visual field (Fig. 5) and BCVA of the left eye was 20/32.

Discussions

Disease of the carotid artery is the most common cause of RAO. It may cause retinal arterial occlusion by two basic mechanisms.

1 Embolism, which is the most common reason. Aortic and mitral valvular lesions, tumor in the left atrium, patent foramen ovale, and myxoma are the sources of plaques in the heart [6].
2 Hemodynamic. In order to produce hemodynamically caused ocular and/or retinal ischemia, by greatly reducing the ocular blood flow, the internal carotid artery has to be noticeably stenosed (70%), or fully occluded. Hayreh reported that 80% stenosis of the internal carotid artery was seen in 14% of BRAO cases and 18% of CRAO, but carotid Doppler/angiography revealed the presence of emboli in 66% in BRAO and 71% in CRAO [5].

Hayreh described that an abnormal echocardiogram with embolic source presented on carotid Doppler/

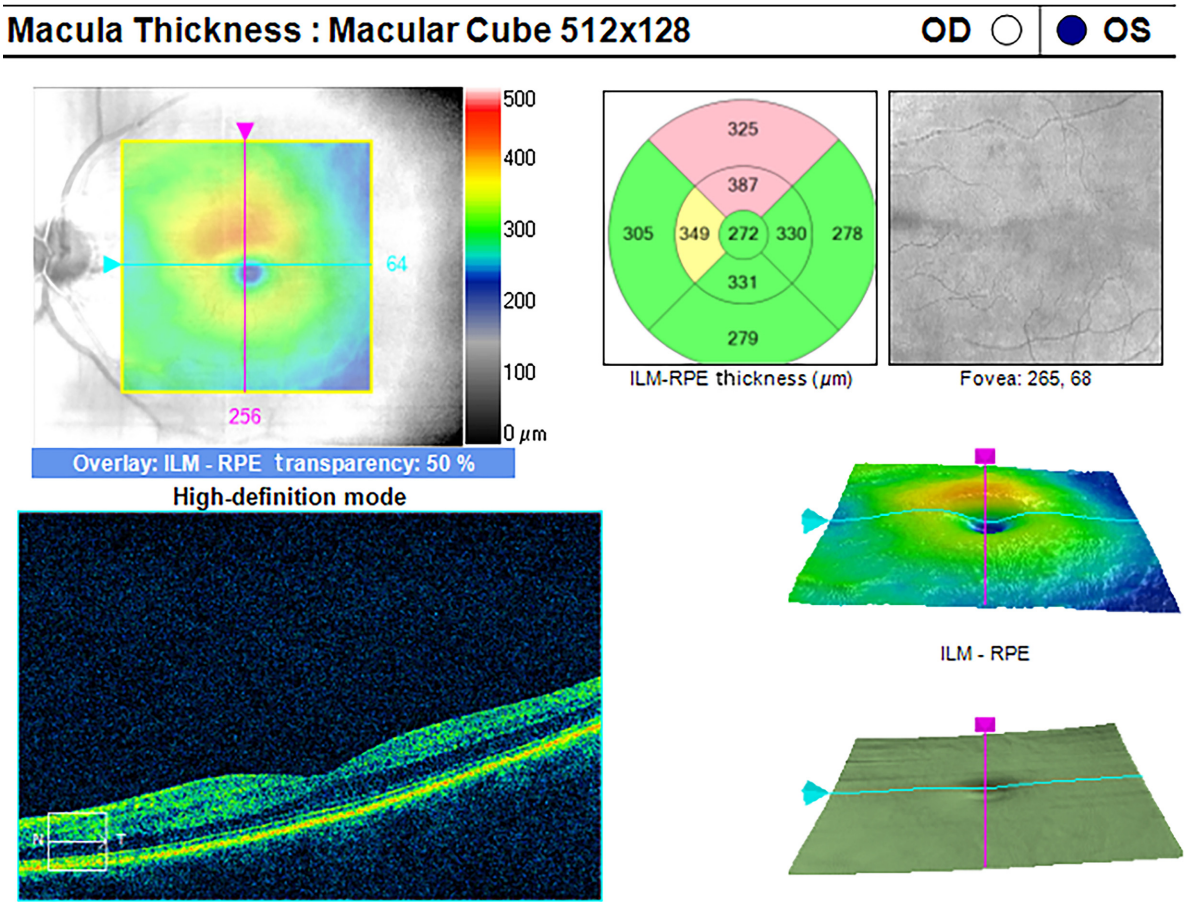

Figure 3. OCT. Increased thickness of the inner layers of the involved retina.

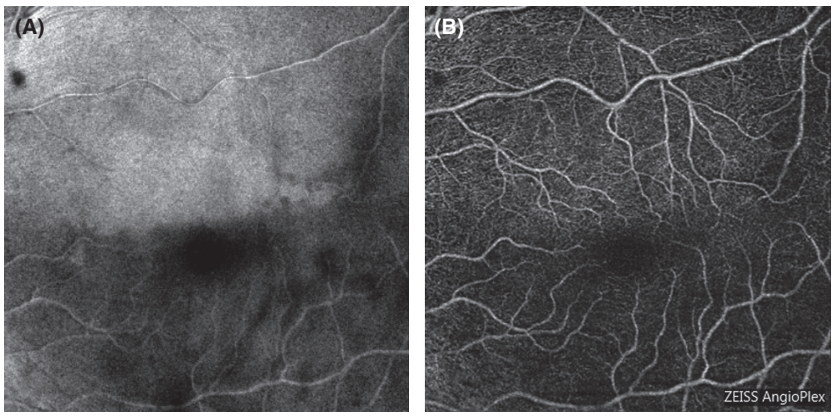

Figure 4. (A) The en face OCT-A of the superficial retina. Hyperreflective area of retinal edema. (B) OCT-A. Dilation of blood vessels of the involved retina.

angiography in 62% of patients with plaque in CRAO and in 44% of the patients with BRAO [5]. This means that the emboli in such cases could have come from either the heart, the carotid artery, or also possibly both, and hence one has to evaluate both the heart and the carotid artery for possible embolism in all patients with BRAO and CRAO.

On the basis of this, Hayreh decided that most RAOs are due to small emboli ("microemboli"), which may be presented as nonhemodynamically significant obstruction.

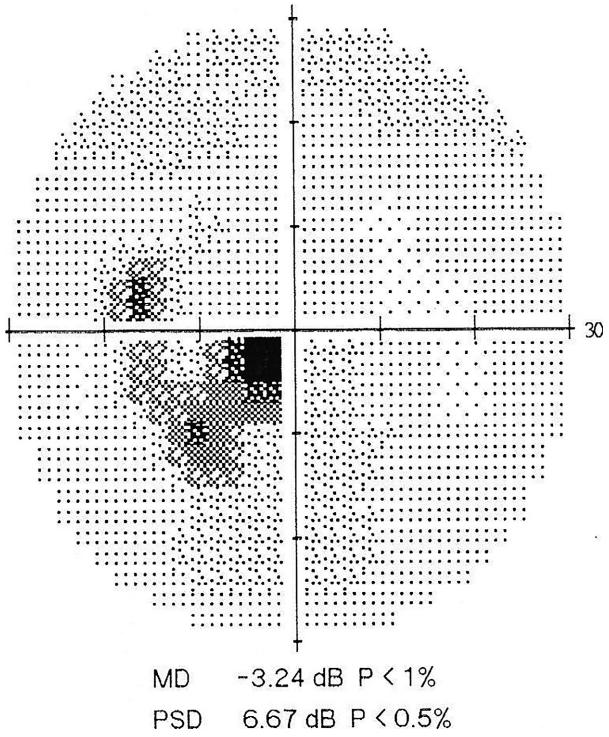

Figure 5. A small scotoma of the lower part of the visual field 1 month after onset of symptoms.

The absence or presence of emboli is generally of much more importance than the grade of stenosis.

In a like manner, the lack of any anomaly shown by echocardiography does not in any way rule out the heart as the origin of embolism, as the resolution may not be sufficient to reveal very small lesions. The source of emboli in the heart is mainly valve disease and rarely any other cardiac anomaly, including patent foramen ovale, mitral valve prolapse, or myxoma [8, 10]. In this case, both carotid ultrasound Doppler and echocardiography were normal.

It has been suggested that foveal retinal thickening may prevent reperfusion of the fovea, which then results in foveal ischemia. This mechanism has been called "no-reflow phenomenon," and may explain residual ischemic changes and central scotoma [13], as is seen in most reports of patients with transient BRAO. Although our patient's BCVA improved, a small scotoma has still persisted.

When the junction between the infarcted and normal retina in BRAO passes through the fovea, the visual acuity may suddenly initially deteriorate; however, visual acuity improvement may take place within days or weeks, from ≤20/200 even to normal [13]. Hayreh et al. in a retrospective study of 18 eyes with transient BRAO reported that initially 6% had visual acuity of ≤20/40 and 94% ≥20/40, which improved to ≥20/30 during follow-up of 100% of all eyes [1]. In our case, the patient had initial BCVA of the left eye light perception with incorrect light projection, which improved to 20/32 the day after the onset of symptoms.

A number of therapies have been used in the treatment of RAO [11, 12]. These include carbogen inhalation, acetazolamide infusion, ocular massage, and paracentesis, as well as various vasodilators [14]. Pentoxifylline is a trisubstituted xanthine derivative that works by increasing erythrocyte flexibility, reducing blood viscosity, and increasing microcirculatory flow and tissue perfusion. In a randomized control trial, a small number of patients with CRAO were randomized to either pentoxifylline or placebo for 4 weeks [7]. The endpoint measurements included objective CRA blood flow measured by duplex scanning. The authors noted an increase in the peak-systolic and end-diastolic flow velocities by 550% and 400%, respectively, in patients treated with pentoxifylline, versus 288% and 200%, respectively, in a placebo group. In our case, we used intravenous infusion with pentoxifylline 100 mg, which resulted in improvement of the condition.

Experimental studies on CRAO in atherosclerotic, elderly, and hypertensive rhesus monkeys (a model for most patients with RAO) revealed that the retina suffers no perceptible damage with RAO of up to 97 min, but beyond this time the longer the RAO, the more irreversible and extensive the damage. RAO for longer than about 240 min results in irreversible, massive retinal damage [2, 5]. In our case, the patient visited the physician one hour after onset of symptoms, and early treatment resulted in migration of the embolus and subsequent improvement in BCVA.

Conclusions

Retinal artery occlusion is a visually disabling, ocular vascular occlusive disorder. In transient RAO, the embolus temporarily blocks the retinal artery and then moves on resulting in recovery of retinal circulation. In this case, using modern noninvasive imaging techniques we have documented the phenomenon of migratory emboli with recovery of perfusion of the retina and improvement of BCVA in a patient with transient BRAO. Early diagnosis and fast action are important in the case of patients with retinal artery occlusion.

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Authorship

AS (Ph.D, FEBO) and MM (Ph.D): performed examination of the patient, wrote the manuscript. TR: wrote the manuscript. NJ (Ph.D, FEBO) and JL (CSc., MSc., FEBO, MBA, LL.A, DBA, FAOG): wrote the manuscript, supervised the project. All authors contributed equally to this work.

Conflict of Interest

The authors state that there are no conflicts of interest regarding the publication of this article. None declared. The authors are grateful to Ian McColl MD, PhD for assistance with the manuscript.

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