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Branch Retinal Artery Occlusion without Morphologic or Electrophysiological Evidence

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Abstract

A 71-year-old man reported sudden, painless loss of the superior visual field of the right eye three days ago. Best-corrected visual acuity was 20/32 on the right and 20/25 on the left eye, a relative afferent pupillary defect was absent. Slitlamp-biomicroscy and dilated fundus exam were normal. Time-domain Optical Coherence Tomography (OCT) of the macula, pattern visual evoked potentials and multifocal electroretinography were normal. Magnetic resonance imaging of the brain showed an infarction located in the right parieto-occipital area, inconsistent with the visual field defect. 2 weeks later fundus examination showed two cholesterol emboli in the inferior temporal retinal artery, OCT showed retinal thinning of the inferior macula.

Conclusion: Branch retinal artery occlusion may initially present without morphologic and electrophysiological evidence. Re-evaluation at a later time should reveal typical findings such as retinal thinning.

Keywords: Retinal artery occlusion; Multifocal electroretinography; Optical coherence tomography

Introduction

We present a case of Branch Retinal Artery Occlusion (BRAO) without initial morphologic or electrophysiological evidence. Retinal artery occlusions are caused by embolization, thrombosis or vasospasm, and affect mainly elderly patients with associated systemic diseases, mainly hypertensive blood pressure [1,2]. Branch retinal artery occlusions represent approximately 38% of all retinal artery obstructions and are clinically characterized by a whitish, thickened area located along the compromised vessel in the acute phase [3].

Case Description

A 71-year-old man complained of sudden painless loss of the superior visual field of the right eye, which had begun three days earlier and had not changed since. There were no complaints suggestive of systemic vasculitis, particularly giant cell arteritis. C-reactive protein (4.2 mg/dl, reference <5 mg/dl) and erythrocyte sedimentation rate (12 mm/hour, reference <20 mm/hour) were within normal limits for his age.

The patient had no history of ocular diseases but reported a transient ischemic attack two months ago and a stroke six weeks ago localized in the right parietal lobe with a corresponding neglect and disturbed coordination of the left hand. He suffered from arterial hypertension and hypercholesterolemia (regular check ups, well controlled) no further systemic diseases.

Best-Corrected Visual Acuity (BCVA) was 20/32 on the right and 20/25 on the left eye. Pupils round, isocor and reacted equally to light without Relative Afferent Pupillary Defect (RAPD). Static (tendency-oriented) 30°-perimetry (Octopus® 900, Haag-Streit, Switzerland) showed an absolute superior altitudinal defect of the right eye and unspecific scotoma of the left eye (Figure 1). The anterior segment and the fundus were normal bilaterally, there was no evidence of optic neuropathy or retinal vessel occlusion noted.

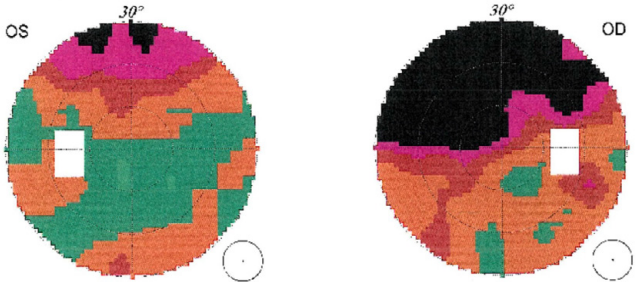

Figure 1: Static perimetry of both eyes (tendency-oriented strategy).

Time-domain optical coherence tomography (Stratus-TD-OCT, macular thickness map, Heidelberg Engineering) showed subtle thickening of the parafoveal inferior retina of the right eye (Figure 2).

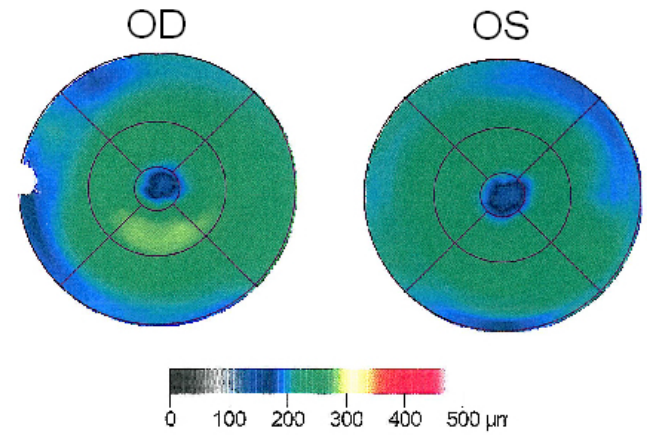

Figure 2: Time-domain optical coherence tomography of both eyes (macula thickness map) three days after the initial event.

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The peripapillary retinal nerve fibre layer was slightly thicker in the inferior half of the right eye compared with the superior half (301 µm vs. 252 µm).

Pattern visual evoked potentials (pVEP, Nicolet Bravo® unit, Hoechberg, Germany) showed mildly reduced amplitudes of the right eye compared to the left eye but no difference of peak times (Figure 3). Multifocal electroretinogram (mfERG, Reti-port unit, Roland Consult, Brandenburg, Germany) was normal in both eyes. No relevant amplitude or peak time differences could be observed between the superior and inferior hemispheres of the right eye or between the superior hemispheres of both eyes (Figure 4).

Since ophthalmologic correlate for the visual field loss could be found, a cerebral magnectic resonance imaging (cMRI) was obtained.

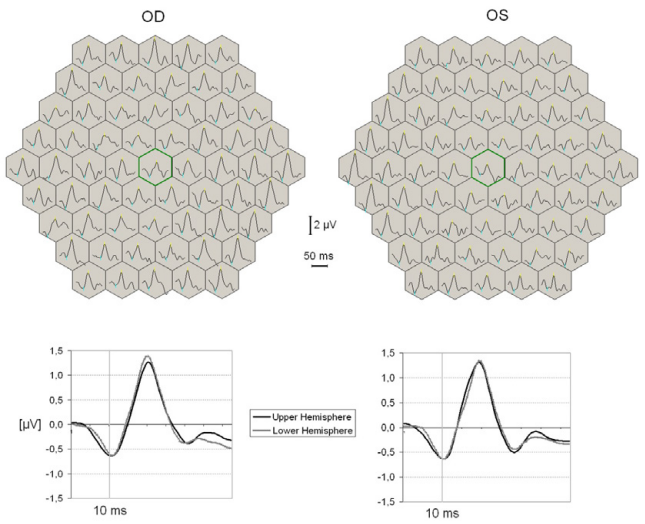

Figure 3: First order kernel of the multifocal electroretinogram. Single answers of each hexagon are depicted on top, below the averaged answers of the upper and lower hemisphere show no relevant differences.

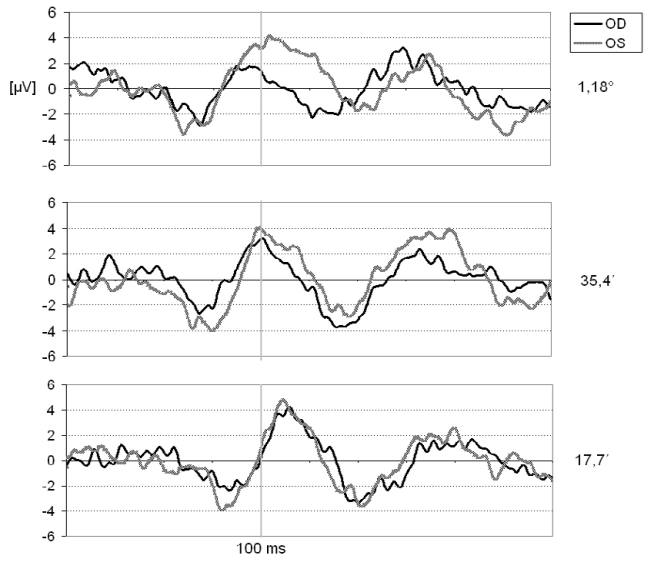

Figure 4: Pattern Visual evoked potentials showing only slight amplitude differences between both eyes. Pattern sizes are depicted on the right.

Compared with previous cMRI exams, an old infarction located in the right parieto-occipital area was noted, not explaining the visual field defect of the right eye (Figure 5). No pathology was noted along the anterior visual pathway of either eye. Additionally, a complete cardiovascular status (24 h-ERG, duplex sonography of carotid arteries, cardiac echography) was imposed to eliminate any possible source of emboli 1-3 days after initial presentation. The duplex sonography showed a 40% stenosis of the right A. carotis communis and a 30-40% stenosis of the left A. carotis interna. The neurologist recommended to increase the daily dose of simvastatin to 40 mg and to repeat the duplex examination at a 6 months interval. The patient did not present noticeable cardiac findings.

Two weeks later, the patient´s symptoms were unchanged. Visual acuity and visual fields were stable, but fundus examination now revealed two yellowish crystalline plaques in the inferior temporal artery without edema in the adjacent retina. Macular OCT showed marked thinning of the inferior retina compared with the superior retina of the right eye (271 vs. 316 µm) (Figures 6 and 7).

We analysed this fact more deeply and measured the thickness of the inner retina complex. For the inner retinal layer 88 um of the superior retina compared to 83 um of the inferior retina were measured. The nerve fibre layer showed similar results: 25 um superior retina compared to 23 um inferior retina. Especially the inner plexiform layer showed a marked thinning of the inferior retina (IPL 21 um superior retina vs. 10 um inferior retina). In addition, the Inner Nuclear Layer (INL) showed a remarkable thinning of the inferior (23 um) compared to the superior retina (28 um).

6 months post branch occlusion a thinning of the inferior inner

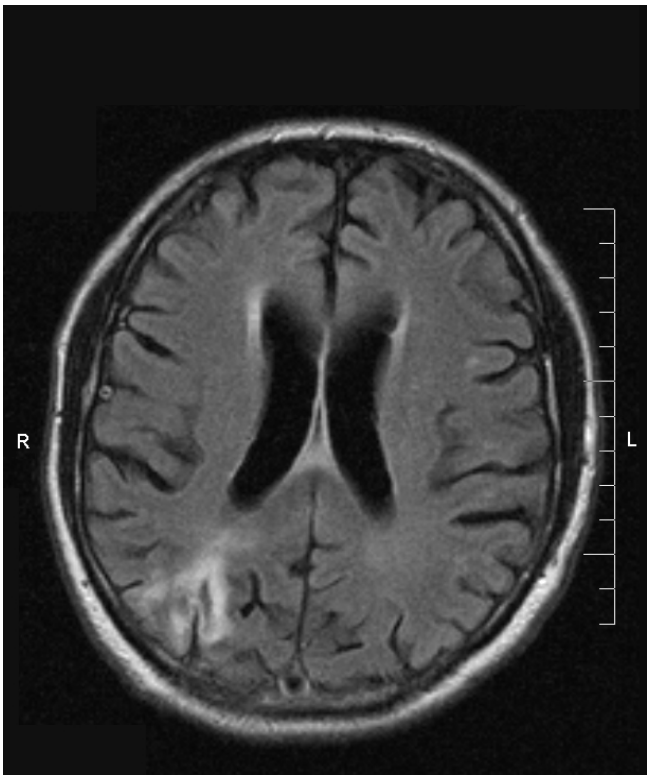

Figure 5: MRI-scan of the head about one week after the initial event showing an infarction located in the right parieto-occipital area, inconsistent with the visual field defect.

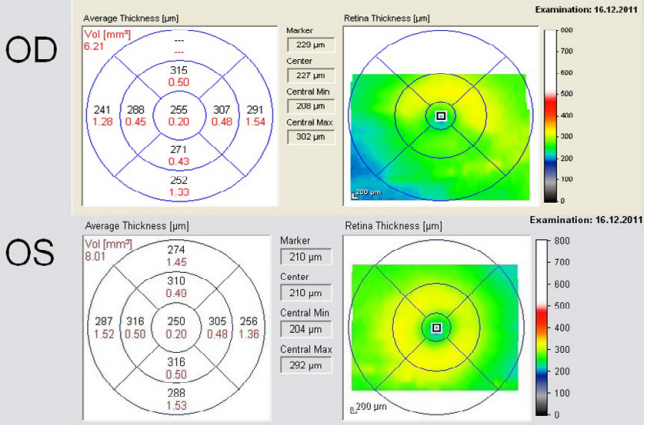

Figure 6: Spectral-domain optical coherence tomography of both eyes (macular thickness map) about two weeks after onset of symptoms. On the right eye the inferior half of the macular retina is markedly thinner.

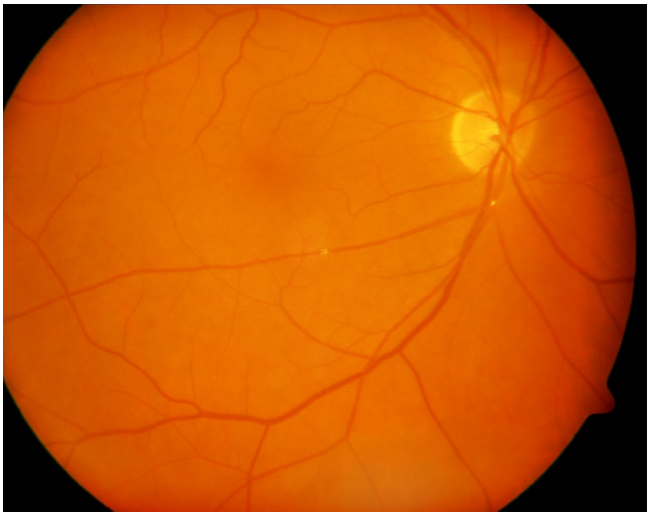

Figure 7: Fundus photography of the right eye taken about 2 weeks after the acute visual field loss and showing two emboli in the inferotemporal branch retinal artery.

retinal layer (75 um) and general inferior retinal thickness (250 um) was still analyzed as the patient presented himself at follow up.

Fluorescein angiography was accomplished another four weeks later, which depicted normal retinal perfusion in both eyes (Figure 8).

Discussion

Why did the examinations performed in the first place fail to detect BRAO?

Swinging-flashlight-test (SWIFT): The SWIFT is an objective test, but can be hard to evaluate, especially if the pupillary light reaction even in the fellow ("normal") eye is slow or scarce as seen in our patient. On the other hand the loss of about half the visual field may not have been enough to lead to a distinguishable RAPD [4].

Ophthalmoscopy/ OCT: In the acute phase of retinal artery occlusion cell death and breakdown of cellular water transport leads to measurable swelling of the retina, which can be measured using OCT. In the later course of the disease atrophy sets in, leading to

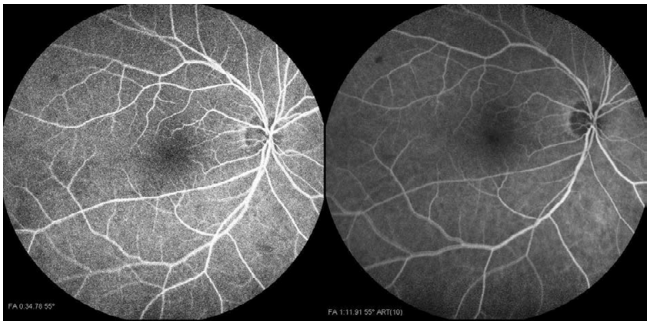

Figure 8: Fluorescein angiography of the right eye 6 weeks after the initial event. Normal retinal perfusion without any signs of edema is depicted. Left side: 34 seconds after infusion, right side: 1 minute 11 seconds after infusion.

retinal thinning [2,5]. In our case, three days after onset of the visual field loss, ophthalmoscopic appearance of the retina was normal and only subtle changes of macular thickness or retinal nerve fibre layer thickness were notedx with OCT. It is possible that the initial retinal swelling had resolved but retinal thinning due to atrophy had not yet developed.

Pattern visual evoked potentials (pVEP): Unlike the flash-VEP the pVEP reflects mainly the activity of the central retina and corresponds better with visual acuity than with the size/ depth of the visual field loss. As visual acuity was only slightly reduced in our patient the amplitudes exhibited only a minor reduction compared with the fellow eye [6]. However, this might be attributed to the relatively larger contribution of the perifoveal retina of the right eye. mfERG: Ohshima et al. [7] showed that the first order kernel of the mfERG may show only slightly decreased amplitudes in the area of a BRAO [7,8]. The second order kernel - reflecting the activity of inner retinal layers –may show retinal dysfunction. Retinal artery occlusion affects mainly the inner retinal layers while the outer retinal layers are still perfused via the choroidal circulation. To the best of our knowledge, this is the first case-description of BRAO with a completely unremarkable first order kernel on mf-ERG.

As there are no therapies available, the study of Ciccone et al. [9] is a promising approach for vascular diseases [9]. They evaluated the acute hemodynamic effects of intranasal 17-beta-estradiol on ophthalmic arterial circulation in postmenopausal women [9]. Their findings showed that nasal administration of E2 induced a significant vasodilatory effect of about 33% in the ophthalmic artery [9]. The nasal application form might be an effective treatment for improving retinal blood supply and therefore to be clinically useful in many pathologies of the retina, especially in this patient' branch occlusion [9]. The systolic and diastolic blood pressure, and heart rate did not significantly differ after drug administration [9].

In conclusion, although funduscopy may not show abnormalities in reperfused areas after retinal artery occlusion, OCT and mfERG would be expected to lead to the correct diagnosis. In our case, even those diagnostic modalities failed to identify the cause of the visual field defect. Therefore, retinal artery occlusion should not be completely ruled out since it may initially present without the typical morphological, imaging and electrophysiological findings. Exams should be repeated, since signs of retinal atrophy and dysfunction after BRAO may appear later during the course. Second order kernel mf-ERG should be measured in cases of where first order kernel is unremarkable.

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