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Case Report Open Access

Branch Retinal Artery Occlusion in Retinal Migraine: A Case Report

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Abstract

Permanent consequences of retinal migraine are rare. We present a case of branch retinal artery occlusion (BRAO) in the course of ocular migraine in a 29-year old woman. Scotoma; in the visual field persist in 6-month follow-up. During the time of observation, visual acuity in the affected left eye remained 5/8.0 (0.1 logMAR). After being diagnosed with migraine, the patient in this case remains under neurological and ophthalmologic care.

Keywords: Branch retinal artery occlusion, Retinal migraine; Scotoma

Introduction

Retinal migraine is defined as a temporary loss of vision or presence of scotoma in the visual field in one eye, co-occurring with migraine headache or followed by migraine within one hour. The symptoms are usually reported in women in their fourth decade of life. The etiology has not been fully explained. Permanent deficits are extremely rare and they are usually caused by earlier vascular disorders [1-5].

In this paper, we present a case of the occlusion of branch retinal artery, leading to retinal ischemia in the course of ocular migraine.

Case Report

A 29-year old woman arrived at the ophthalmology emergency ward, complaining of worsening visual acuity from the morning hours of the previous day and the presence of positive scotoma in the upper part of the visual field in the left eye. The symptoms cooccurred with migraine pain in the frontal and left temporal area, and were preceded by visual aura. The medical report mentions the occurrence of migraine pains with aura for two years and asthma. The patient did not report any traumas, surgeries or other general diseases. Her blood pressure was 120/78 mmHg and heart rate 74/min.

A full ophthalmic examination was carried out, including indirect biomicroscopy. Visual acuity in the right eye was 5/6.3 (0.1 logMAR); and in the left eye – 5/8.0 (0.22 logMAR). Intraocular pressure was within normal range (TOD=14 mmHg, TOS=16 mmHg). Pupillary reflexes were correct. No abnormalities in the anterior segment were found. The following findings were noted during the fundus examination of the affected, left eye: retinal pallor and macular edema, above the lower temporal vascular arcade (Figures 1a and 1b). In the right eye the image was within the normal range.

Swept-Source OCT examination (DRI OCT Triton, Topcon, Japan) showed thickening of the inner nuclear layer and inner plexiform layer in the area of the lower temporal vascular arcade (Figure 2a). Swept-Source OCT angiography showed a hyporeflective area in the inner retina and in the choriocapillaries and was correlated with the SS-OCT images (Figure 2b).

Another examination performed in this case was automated perimetry, conducted with the Frey AP-300 perimeter. The result is presented below (Figure 3).

Fluorescein angiography showed a retarded flow of contrast in the blood vessel. Computed tomography of the head did not show intracranial bleeding nor new ischemic lesions of the brain. The ventricular system was without midline shift; subdural fluid reserve was sustained; and neurocranium bones presented as normal.

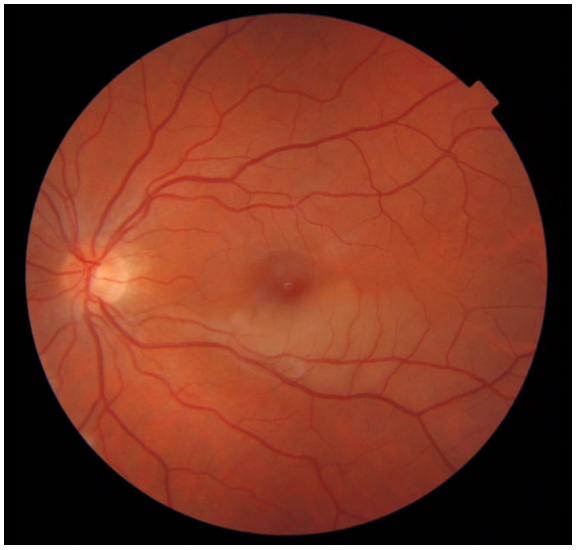

Figure 1a: Color image of the left eye show retinal pallor above the lower temporal vascular arcade.

Neither meningeal nor focal signs were found during a neurological consultation. Following the neurologist's recommendation, 15% Mannitol and then 100 mg Hydrocortisone with ketoprofen were administered intravenously. Although the pain subsided, the scotoma in the visual field remained. The neurologist diagnosed common migraine.

Laboratory tests results: complete blood counts, erythrocyte sedimentation rate, C-reactive protein, electrolytes, glucose, cholesterol, creatinine, coagulation panel, rheumatoid factor were all within the normal range. Increased levels of Cytomegalovirus antibodies IgG were reported.

At the next visits, after 1 day, 1 week, 1 month and 6 months the condition of the fundus gradually returned to normal (Figures 4a and 4b). Visual field defects decreased (Figure 5). During the 6-month period of observation, visual acuity in the right eye remained 5/6.3 (0.22 logMAR); and in the left eye 5/8.0 (0.1 logMAR). The patient is still under ophthalmologic and neurological care.

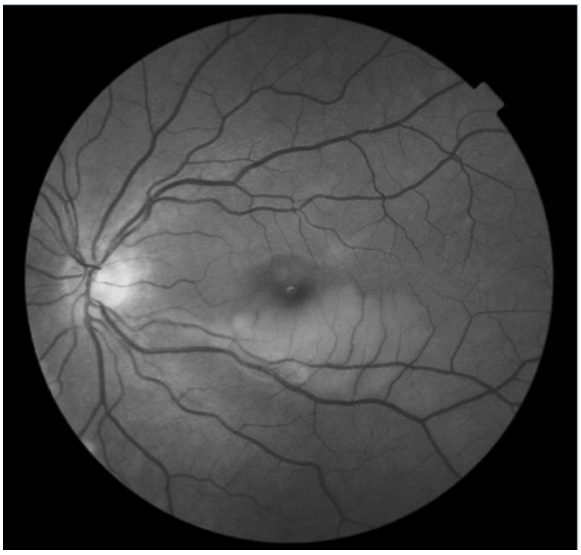

Figure 1b: Red-free image of the left eye.

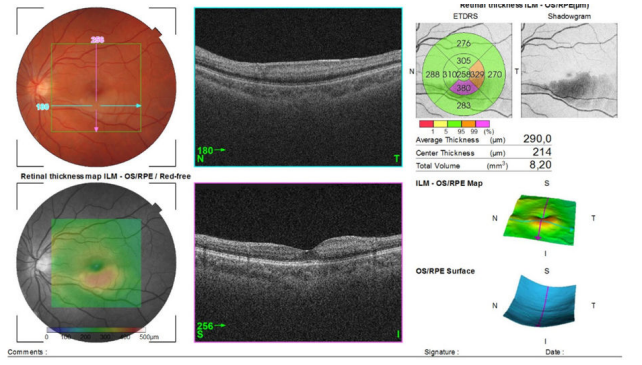

Figure 2a: SS-OCT shows thickening of the inner nuclear layer and inner plexiform layer in the macula area.

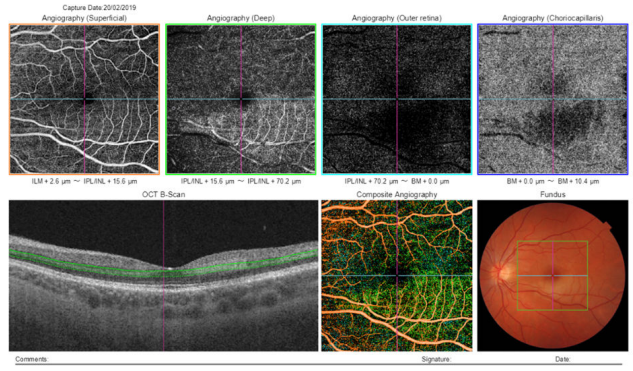

Figure 2b: SS-OCT Angiography shows a hyporeflective area in the inner retina and in the choriocapillaries.

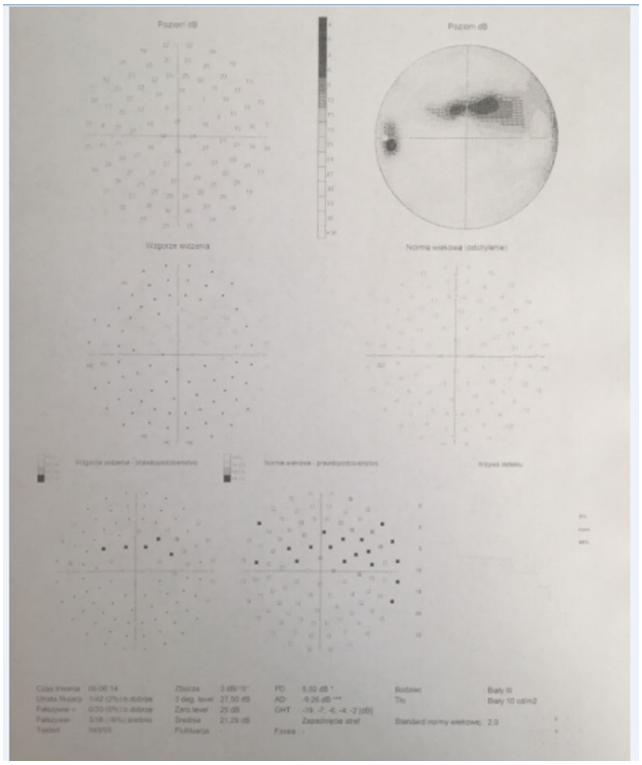

Figure 3: Para-central scotoma in the upper temporal part of visual field.

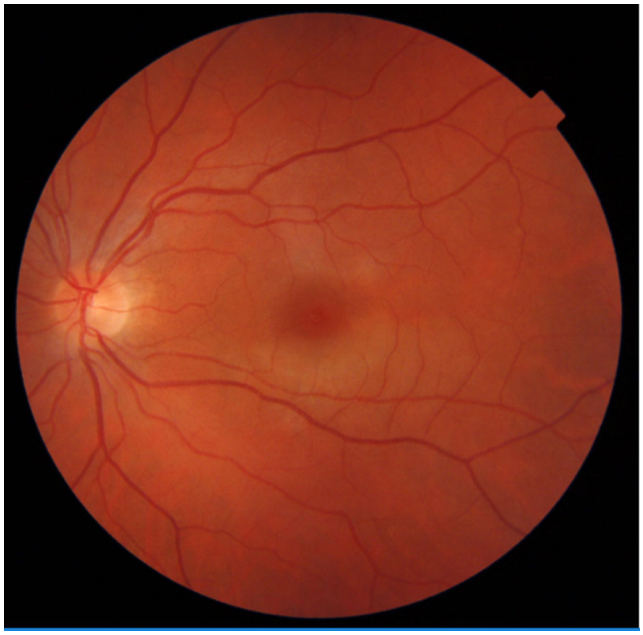

Figure 4a: Color image of the left eye.

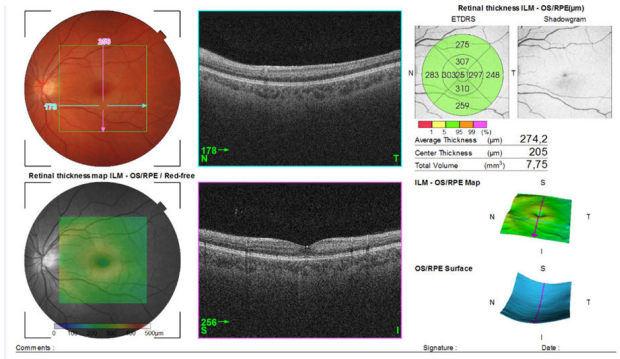

Figure 4b: SS-OCT of the left eye.

Discussion

Retinal migraine is referred to by numerous other terms, including ocular migraine, visual migraine, eye migraine, and anterior visual pathway migraine, but it should not be confused with classic visual auras (cortical-based visual phenomena), ophthalmoplegic migraine (resulting in diplopia), or silent migraine (aura without headache) [1].

In order to make a correct diagnosis, it is necessary to diagnose migraine according to the criteria of the International Classification of Headaches [4]. The therapeutic procedure in retinal migraine comes down to symptomatic treatment. In retinal migraine it is important to avoid the use of Triptans, ergots, and beta blockers in migraines with transient vision loss secondary to risk of potentiating vasoconstriction and increasing the risk of irreversible visual loss [6].

It is believed that the factors triggering retinal migraine are exactly the same as those in classic migraine with aura: stress, smoking, hypertension, oral hormonal contraception, dehydration, hypoglycemia or overheating. Positive family history implies genetic predisposition, inherited in an unspecified way [1,2].

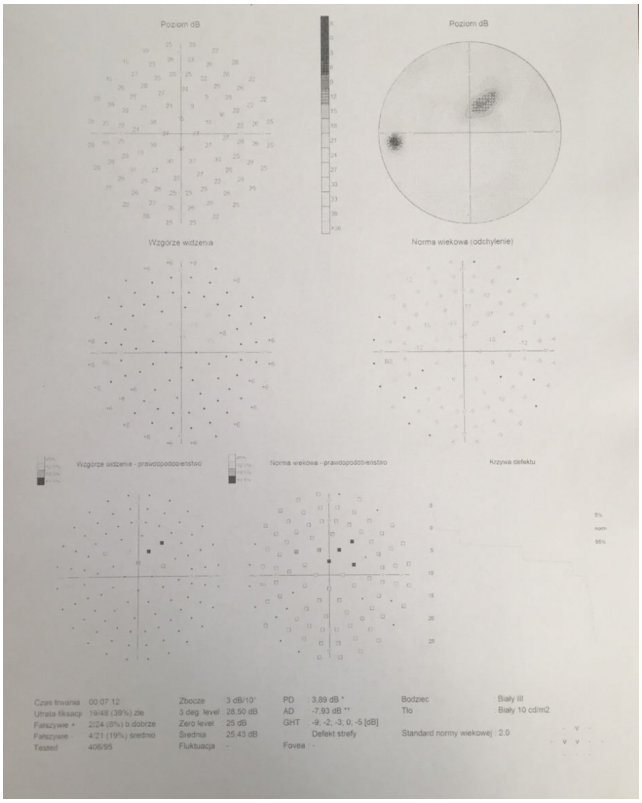

Figure 5: Scotoma in the upper temporal part of visual field decreased.

Permanent consequences of retinal migraine are rare [2-7]. Therefore, it is essential to understand its etiopathogenesis. There are several hypotheses, one of which assumes that retinal migraine is originally caused by a vasospasm within the area of the retinal or the ciliary vascular system. This may be controversial due to the complexity of blood supply to the retina, which has dual vasculature and is characterized by a low blood flow while flow in the choroid is high. The central retinal artery supplies blood to the anterior two-third of the neurosensory retina. Here, there is no autonomic innervation; sensory nerves undergo self-regulation and retinal circulation is mainly influenced by local factors. Choroidal supply occurs in outer onethird retina, containing mainly autonomic fibers which are not self-regulating [1,2].

According to another theory, the etiopathogenesis of retinal migraine is concerned with a spreading depression of neuronal function in the retina, similar to the spreading depression of Leão found in the cerebral cortex, which may be involved in migraine with aura where both eyes are affected by visual disturbances. The signs then include diffuse and segmental narrowing of arteries and veins. Fluorescein angiography confirms the diagnosis. It shows retarded or blocked flow in retinal arteries or their branches with correct choroidal flow or spot irregularity of the retina [1,2].

It seems possible that the neuron and glial depolarization caused by a spreading depression could trigger an inflammatory response (similar to cortical spreading depression's potential to activate the trigeminal nerve) leading to vasoconstriction or vasospasm which could then cause the amaurosis, or the hypersensitivity caused by migraine could trigger a neovascular response in the form of hypo-perfusion again leading to the vasoconstriction/vasospasm and thus amaurosis.

To sum up, the occlusion of the branch retinal artery in retinal migraine is a rare consequence of migraine. The probable cause may be the disrupted functioning of neurons, leading to vascular occlusion. In the case we present, vasoconstriction was a long-lasting condition and caused retinal ischemia. Scotoma in the visual field and lowered visual acuity persist. After being diagnosed with migraine, the patient in this case remains under neurological and ophthalmologic care.

Conclusion

This study, probably a pioneering one, observes the effects of vitamin B, C and E on the surrogate markers of responsible biochemical derangements, and the structural and functional capabilities of RBCs related to development of DR in diabetic subjects for a considerably longer period of time.

References

1. Pula JH, Kwan K, Yuen CA, Kattah JC (2016) Update on the evaluation of transient vision loss. Clin Ophthalmol 10: 297-303.
2. Pradhan S, Chung SM (2004) Retinal, ophthalmic, or ocular migraine. Curr Neurol Neurosci Rep 4: 391-397.
3. Khalili YA, King KC (2019) Retinal Migraine Headache. Treasure Island (FL): StatPearls Publishing.
4. Headache Classification Committee of the International Headache Society (2018) The International Classification Of Headache Disorders. Cephalalgia 38: 1211.
5. Evans RW, Grosberg BM (2008) Retinal migraine: Migraine associated with monocular visual symptoms. Headache 48: 142-145.
6. Loehrer E, Vernooij MW, Van DLA, Hofman A, Ikram MA (2015) Migraine and cerebral blood flow in the general population. Cephalalgia 35: 190-198.
7. Reggio E, Chisari CG, Ferrigno G, Patti F, Donzuso G, et al. (2017) Migraine causes retinal and choroidal structural changes: Evaluation with ocular coherence tomography. J Neurol 264: 494-502.