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Declarations

Conflict of interest: The authors declare that there is no conflict of interest.

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Acute monocular oligemia in a patient with migraine with aura demonstrated using OCT-angiography: a case report.

Abstract

Introduction

Migraine is one of the most common causes of transient visual loss. Optical coherence tomography angiography (OCTA) provides fast and non-invasive imaging of the retinal vessels. We report the first case of monocular retinal oligemia demonstrated using OCTA during a migraine attack with aura.

Case description

A 27-year-old man with a previous history of migraine with visual aura was seen in the emergency room due to acute left hemicranial pain with positive visual symptoms in his right eye. The patient reported a blue stain in his right eye. Optical coherence tomography angiography (OCT-A) showed an extensive area of hypoperfusion in the macular region of his right eye. Forty-eight hours later visual symptoms had improved and the OCT-A showed a significant reduction in the area of hypoperfusion. Seven days later the patient was asymptomatic and retinal perfusion had returned to normal values.

Conclusion

Monocular involvement suggests that these retinal vascular changes are independent from cerebral vascular changes, supporting the hypothesis of selective retinal ganglion cell layer spreading depression as the possible cause of some cases of retinal migraine.

Keywords: retinal migraine, OCTA, retina, ischemia, retinal ganglion cell layer, cortical spreading depression, case report.

Introduction

Migraine affects approximately 15% of the global population. It is the most prevalent neurological condition and the third most frequent global health disorder in both genders[1]. Migraine is also one of the most common causes of transient visual loss [2]. Visual field loss and positive visual symptoms are usually homonymous; nevertheless, some patients develop monocular visual symptoms. The terminology used in these monocular cases has been confusing, but nowadays retinal migraine is the accepted term by the HIS 3 classification[3].

From a physiological point of view, retinal migraine is thought to be a neuro-vascular phenomenon, in which cortical spreading depression seems to be the main mechanism, with vascular changes as a secondary phenomenon[4]. Non-arteritic ischemic optic neuropathies and arterial vascular occlusions have been reported in patients with migraine and there is evidence of vasospasm of the retinal arteries occurring in some patients[5, 6].

These episodes of ocular ischemia may explain why structural changes in the optic nerve and retina have been consistently reported in populations that suffer from migraines. Several studies have shown that there is a decrease in the retinal nerve fiber layer, with more severe changes in patients that suffer migraine with aura than in those without aura[1].

Optical coherence tomography (OCT) was invented in 1991 and has evolved tremendously since then. Traditionally, fluorescein angiography was used to evaluate the retinal vasculature. However, nowadays OCT angiography (OCTA) provides fast and non-invasive imaging of the retinal vessels. Although fluorescein angiography is still used in certain cases [7], it has been largely displaced by OCTA.

Case description

We report the case of a 27-year-old, right-handed man who suffered from recurrent headaches since he was 14 which fulfilled HIS III criteria for migraine with aura. A CT scan performed when he was seventeen was normal. He had a family history of migraine with aura. The episode for which he consulted had begun 48 hours earlier as a typical episode of migraine with aura with left hemicranial pain and flashes in the contralateral eye. Forty-eight hours later he perceived a bluish stain in the center of his right eye, that spread gradually . The headache was similar to those he had suffered periodically. He referred a non-pulsatile pain in the left side of the head, without nausea and vomiting. He also had photophobia and sonophobia but not osmophobia. The patient was under treatment with Tryptizol and had taken Naproxen at home.

Neurological examination was normal and the neuroophthalmological evaluation showed a decimal uncorrected visual acuity of 0.9 in both eyes. Intraocular pressure, pupil examination and ocular fundus were normal, but optical coherence tomography angiography (OCT-A) performed with OCT Triton, Topcon, Tokyo, revealed the presence of an extensive area of hypoperfusion in his right retina. These changes were more severe in the superficial retinal plexus than in the deep retinal plexus (Figure 1).

Macular OCT showed normal foveal profiles without edema in any of the retinal layers, and normal ganglion cell layer thickness. Optic disc OCT was not performed during the acute phase, but was carried out on the one-year follow-up consultation and did not show any abnormalities (peripapillary retinal nerve fiber layer average thickness was 112 microns in his RE and 109 microns in his LE) Despite the presence of this area of oligemia, central visual fields were normal in both eyes. He was treated with intravenous Metamizol, which reduced the pain. Forty-eight hours later, only mild pain persisted in the left side of his head and the bluish stain was very small and limited to the superotemporal area, next to center of the visual field. OCTA showed normal retinal vasculature (Figure 2). Eight days later the patient was asymptomatic and OCTA remained normal. Cerebral magnetic resonance imaging performed one month later did not reveal any abnormalities.

Conclusion

The development of new technologies reshapes our way of thinking. For example, OCT technology has allowed us to measure the retinal ganglion cell layer, showing that occipital lesions can induce anterograde degeneration of these cells. This finding has proven that the paradigm that stated that retrograde trans-synaptic neuronal degeneration did not take place in the human brain was wrong[8].

A decade ago, in an editorial, Winterkorn reported that vasospasm during a migraine attack had been photographically documented in fewer than 10 patients[9]. However fundus photography can only detect vasospasm in the main retinal arterioles and most cases of hypoperfusion are probably caused by changes in neuronal activity and platelet function, not by vasospasm.[9] OCTA can detect hypoperfusion in smaller

vessels and thereby constitutes a much more sensitive method to detect retinal oligemia. In a recent article, Atilla et al reported reversible bilateral retinal hypoperfusion in the macular area in one patient with migraine.[10]

Our case is not easy to understand from a topographical point of view, since the retinal changes were monocular and contralateral to the headache. They may be related processes but they do not seem to respond to the same migraine phase pathophysiology. The oligemia could be the consequence of a primary retinal spreading depression phenomena, similar to classic aura. In our patient, retinal oligemia was more severe in the superficial retinal plexus and therefore it might have been due to selective spreading depression of the retinal ganglion cell layer (Figure 1). This might explain why it was contralateral to the headache as well as the absence of a retinal lesion after the resolution of the retinal aura (Figure 2). There is one other report of OCTA performed during a migraine attack. In this case, oligemia affected both retinas, and the severity of vascular changes was similar in the superficial and deep plexus[10].

We conclude that OCTA allows fast and non-invasive measurements of ocular circulation and it is probably going to provide new insights into the pathophysiology of retinal migraine in the future, leading to better characterization and classification. In this patient, vascular changes were more severe in the superficial plexus, supporting the hypothesis of selective retinal ganglion cell layer spreading depression as the possible cause of some cases of retinal migraine. Nevertheless, this idea of selective retinal ganglion cell layer spreading depression as the cause of some cases of monocular should be confirmed in the future by more extensive case report series.

Declarations

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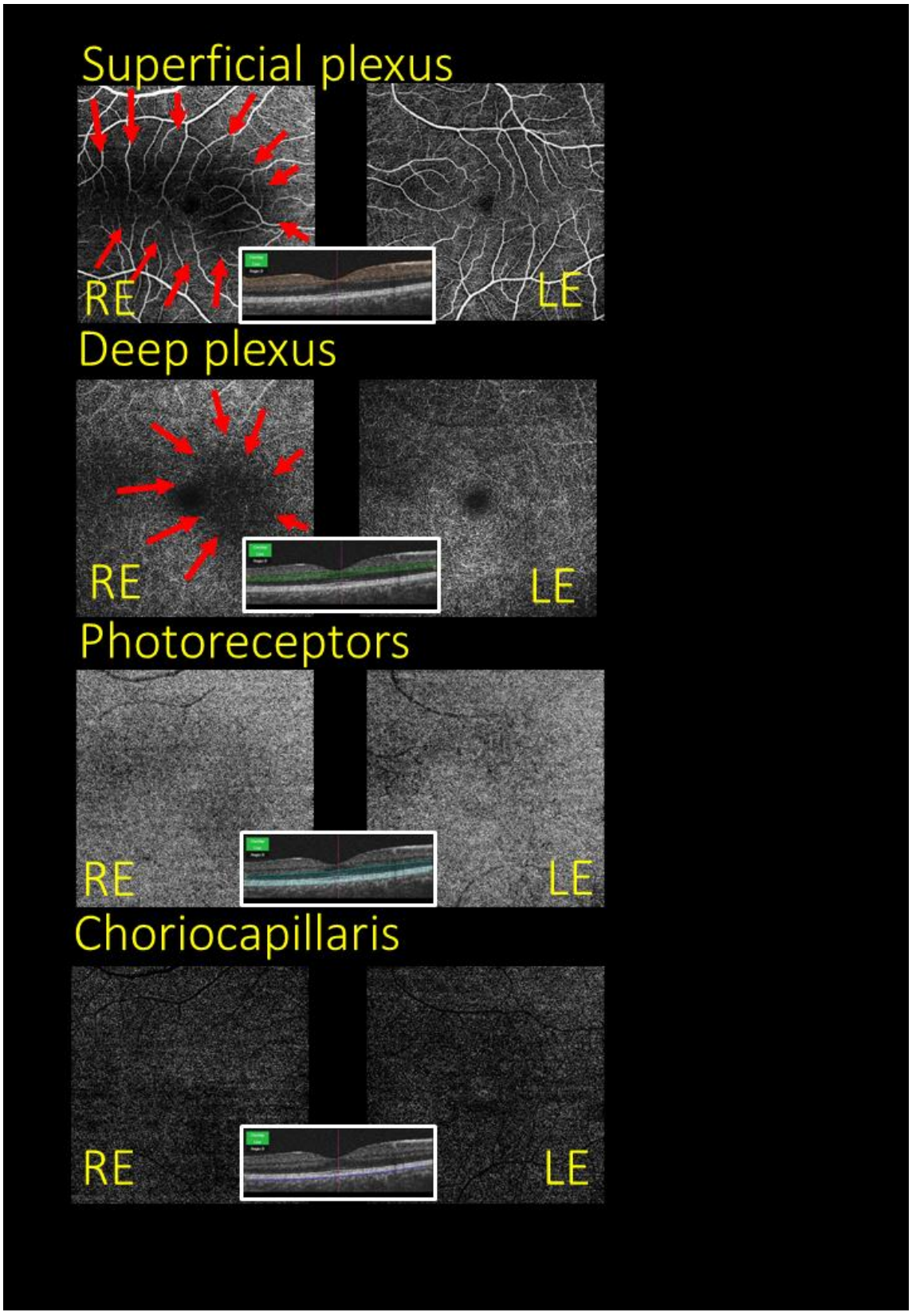
Superficial plexus
RE
LE
Deep plexus
RE
LE
Photoreceptors
RE
LE
Choriocapillaris
RE
LE

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Figure 1. An extensive area of oligemia was present during the attack. It was more severe in the superficial plexus than in the deep plexus (red arrows). Figures show OCT-A maps of the retinal superficial plexus, retinal deep plexus, photoreceptor (in healthy people this level should be avascular) and choriocapillaris. RE=right eye, LE=left eye.

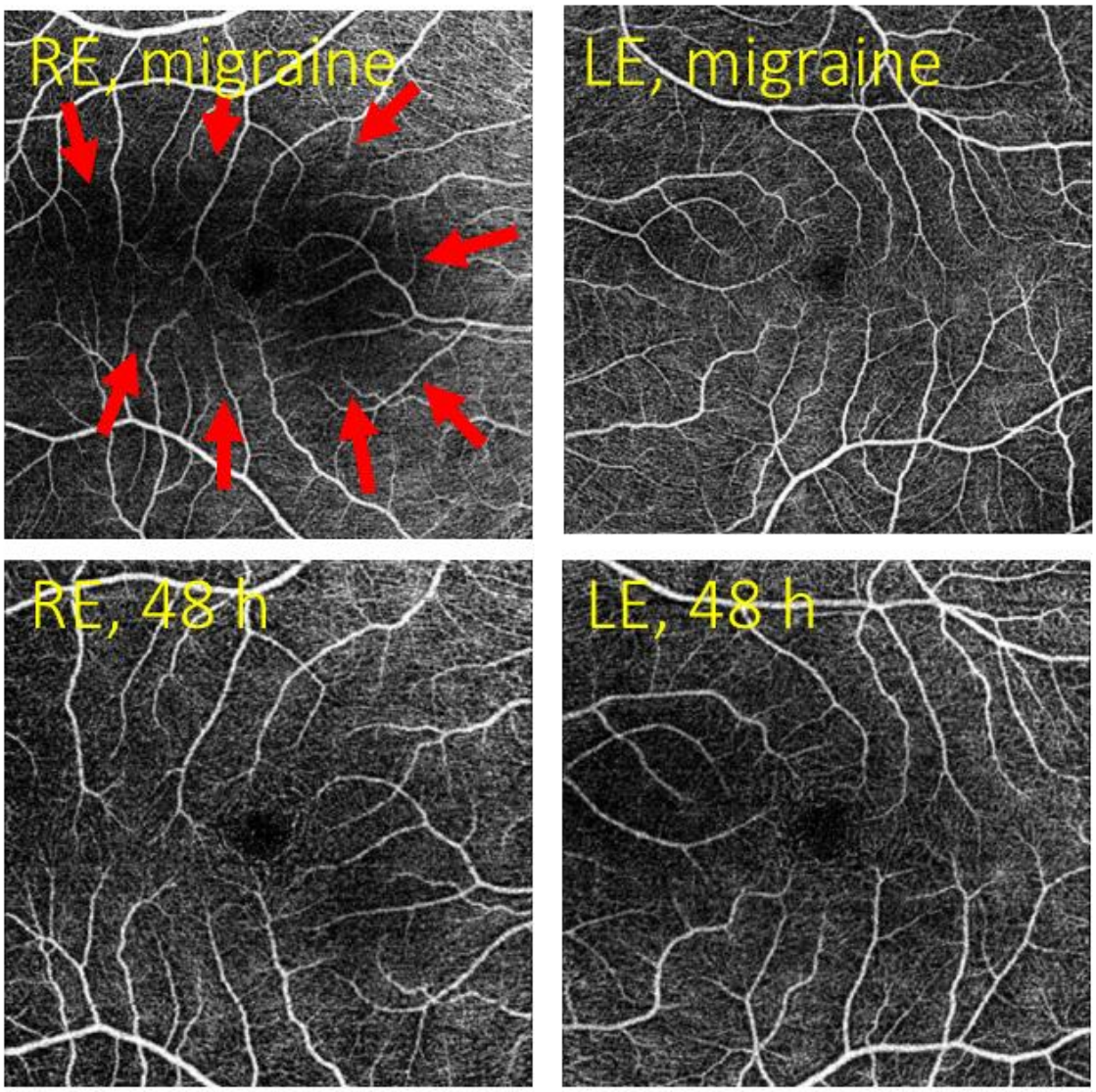

Figure 2. An extensive area of oligemia was present during the attack in the retina superficial plexus in the right eye. Forty-eight hours later the retinal plexus had returned to normal. RE=right eye; LE=left eye.

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CARE

CARE Checklist of information to include when writing a case report

Topic	Item	Checklist item description	Reported on Line
Title	1	The diagnosis or intervention of primary focus followed by the words "case report"	40, 41
Key Words	2	2 to 5 key words that identify diagnoses or interventions in this case report, including "case report"	60,61
Abstract (no references)	3a	Introduction: What is unique about this case and what does it add to the scientific literature?	83-85
	3b	Main symptoms and/or important clinical findings	88-109
	3c	The main diagnoses, therapeutic interventions, and outcomes	88-109
	3d	Conclusion—What is the main "take-away" lesson(s) from this case?	135-140
Introduction	4	One or two paragraphs summarizing why this case is unique (**may include references**)	135-140
Patient Information	5a	De-identified patient specific information.	88-89
	5b	Primary concerns and symptoms of the patient.	91-97
	5c	Medical, family, and psycho-social history including relevant genetic information	88-91
	5d	Relevant past interventions with outcomes	94-97
Clinical Findings	6	Describe significant physical examination (PE) and important clinical findings.	98-109
Timeline	7	Historical and current information from this episode of care organized as a timeline	88-109
Diagnostic Assessment	8a	Diagnostic testing (such as PE, laboratory testing, imaging, surveys).	100-103; 109-110
	8b	Diagnostic challenges (such as access to testing, financial, or cultural)	
	8c	Diagnosis (including other diagnoses considered)	89
	8d	Prognosis (such as staging in oncology) where applicable	
Therapeutic Intervention	9a	Types of therapeutic intervention (such as pharmacologic, surgical, preventive, self-care)	96-97
	9b	Administration of therapeutic intervention (such as dosage, strength, duration)	96-97
	9c	Changes in therapeutic intervention (with rationale)	96-97
Follow-up and Outcomes	10a	Clinician and patient-assessed outcomes (if available)	106-108
	10b	Important follow-up diagnostic and other test results	106-108
	10c	Intervention adherence and tolerability (How was this assessed?)	96-97
	10d	Adverse and unanticipated events	
Discussion	11a	A scientific discussion of the strengths AND limitations associated with this case report	125-135
	11b	Discussion of the relevant medical literature **with references**.	112-124
	11c	The scientific rationale for any conclusions (including assessment of possible causes)	136-141
	11d	The primary "take-away" lessons of this case report (without references) in a one paragraph conclusion	136-141
Patient Perspective	12	The patient should share their perspective in one to two paragraphs on the treatment(s) they received	
Informed Consent	13	Did the patient give informed consent? Please provide if requested	Yes ☒ No ☐

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