Short communication

Delayed diagnosis of ophthalmic artery obstruction due to atrial myxoma

N. Sabater*, S. Alforja, A. Rey, J. Giralt

Unidad de retina, Hospital Clínic i Provincial de Barcelona, Institut Clínic d’Oftalmologia (ICOF), Barcelona, Spain

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ABSTRACT

Case report: A 56-year-old woman with atrial myxoma presented with a visual acuity of no light perception after acute ophthalmic artery obstruction (OAO) associated with stroke. She developed late retinal pigmented changes due choroidal infarction, typical of the OAO.

Discussion: Simultaneous obstruction of the retinal and choroidal circulation was observed in the OAO. Atrial myxoma should be suspected in patients who suffer from OAO associated with stroke. Systemic studies should be performed to find the origin of OAO.

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Diagnóstico tardío de obstrucción de arteria oftálmica por mixoma atrial

RESUMEN

Caso clínico: Une mujer de 56 años con mixoma atrial presentó una agudeza visual de no percepción luminosa tras la obstrucción aguda de la arteria oftálmica (OAO) asociada a accidente vascular cerebral. Tardíamente desarrolló cambios pigmentarios retinianos por infartos coroideos, una característica que diferencia la OAO de la obstrucción de arteria central de la retina.

Discusión: La obstrucción simultánea de las circulaciones retiniana y coroidea se observa en la OAO. Debe sospecharse la presencia de un mixoma atrial en aquellos pacientes con una OAO asociada a accidente vascular cerebral. Deben realizarse estudios sistémicos para encontrar la etiología de la OAO.

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* Corresponding author.
E-mail address: noelia.sabater@gmail.com (N. Sabater).

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Introduction

The ophthalmic artery is the collateral branch of the internal carotid artery that irrigates the eye and the orbit. It divides into several branches, including the central retina artery that irrigates the internal retina and the posterior ciliary arteries that irrigate the external artery, the choroids and the sclera. For this reason, any acute obstruction of the ophthalmic artery could produce choroidal and retina infarcts.  

Case report

Female, aged 56, who visited our practice for evolutionary examination of a central retina artery obstruction (CRAO) in the left eye (LE) diagnosed 6 months earlier.

The pathological history of the patient included ischemic cerebral vascular accident (CVA) of the medial left cerebral artery simultaneously with said CRAO as well as surgical exeresis of an actual myxoma diagnosed during the etiology study of said process.

Examination revealed a visual acuity (VA) measured with Snellen of 0.8 in the RE and no light perception (NLP) in the LE; intraocular pressure (IOP) of 19 and 16 mmHg; LE pupil in medium midriasis with afferent pupil defect and normal anterior segment biomicroscopy in both eyes. The ocular fundus exploration was normal in the RE whereas the LE exhibited optic disk paleness, fiber shaped papillary vessels, some retinal vessels without blood and retinal pigment perifoveal, temporal extrafoveal (Fig. 1) and in the retinal periphery in the form of large pigment groups (Fig. 2).

As the retinal pigment changes could not be explained with the initial CRAO diagnostic, the exploration carried out 6 months earlier when the patient was admitted due to the CVA was reviewed in depth, observing medium LE midriasis with afferent pupil defect in the same eye, RE VA of 0.9 and LE VA of moving hands, IOP of 17 and 16 mmHg and normal anterior segment in RE. The ocular fundus was normal in RE whereas the LE evidenced diffuse retinal edema with papillary involvement and bright red spots with perifoveal hypopigmented halo (Fig. 3). Due to the condition of the patient fluorescein angiography could not be performed. Retinographies were taken at admission time and after 72 h, revealing small hyperpigmented perifoveal small spots and a white-yellowish subretinal loci smaller than one papillary diameter, temporal to the fovea, suggesting choroidal infarct (Fig. 4).

Fig. 1 – Posterior pole image taken in an examination 6 months after the obstructive condition. Pigment changes can be seen in the fovea and the temporal area of the posterior pole, as well as papillary paleness, fiber-shaped vessels and a vessel without blood.

Fig. 2 – LE inferior temporal periphery showing in greater detail aggregates of pigment 6 months after the ophthalmic artery obstruction.

Fig. 3 – LE retinography obtained when obstruction was diagnosed, showing paleness, edema and bright red spots with hypo pigmented halo in the retina.
Discussion

Choroidal infarcts cause damages in the retina pigment epithelium which eventually causes hyperplasia, dispersion with retinal pigment aggregates and probably metaplasia.1 In the initial retinographies, the patient of this report exhibited lesions compatible with choroidal infarcts and at present retinal pigment changes. This led us to conclude that it was not only an occlusion of retinal but also choroidal blood flow. Simultaneous acute obstruction of retinal and choroidal blood flow is known as ophthalmic artery obstruction (OAO). In some cases there is only one obstruction point in the ophthalmic artery (in the trunk), but in other cases simultaneous obstruction of found in multiple locations of retinal vascularization (short posterior ciliary arteries).2 A Doppler echography of the ophthalmic artery would help to locate the site of the occlusion.3

OAO is an infrequent occurrence (between 3% and 5% of arterial occlusions) and could be due to arbitrary trauma, retrobulbar anesthesia, injection of depot corticoids or systemic diseases such as orbital mucormycosis, carotid disease and atrial myxoma. OAO presents as a sudden and very severe visual acuity reduction (light perception or even NLP), optic nerve paleness, fiber-shaped vessels, extensive retina edema, sinuous or absent bright red spots, suggesting simultaneous ischemia of the retina and choroids, as well as delayed pigment alterations.1,3,4 In the cases in which OAO is due to an interruption of the blood flow at the level of the ophthalmic artery trunk, it could present with hypotonia.1 Tests assisting diagnosis include fluorescein angiography (FA) or indocianine green angiography, electroretinogram (ERG) and Doppler echography of the ophthalmic artery. FA shows retinal and choroidal perfusion defects and occasion staining at the level of the retinal pigment. ERG reveals absence of a and b waves, while in the CRAO only the b wave is altered.1 Doppler echography can assist in locating the exact point of the occlusion, which shows as arterial blood flow reduction when the obstruction occurs in the trunk of the OAO.5 Due to the poor general condition of the patient when the CRAO was diagnosed, the only supplementary examinations were retinographies. On the basis of the localized pigment aggregations and the absence of hypotonia, it was concluded that the patient exhibited multiple simultaneous obstructions. It was not considered that additional supplementary tests would provide greater information for the prognosis.

Atrial myxoma is an infrequent primary heart tumor which, in up to 40–50% of cases, expresses initially as distal embolization, particularly to the central nervous system. Up to 50–80% of heart myxoma patients exhibit CVA. Isolated ophthalmic artery or retinal central artery embolization is described but, as in the case of this patient, the majority associate, concomitant embolization to the central nervous system.6

Once the ophthalmological condition has been established there really is no proven effective treatment and very few reports of functional recovery. With a patient suffering simultaneous occlusion of choroidal and retinal blood flow, systemic studies should be carried out to discard carotid or cardiac tumor disease, above all in the presence of associated neurological involvement.

Conflict of interests

No conflict of interests has been declared by the authors.

REFERENCES