Short communication

Macular atrophy in Terson’s syndrome

J.L. Sánchez-Vicente a, L. Frau-Aguilera a,*, P. Sánchez-Vicente b, A. Herrador-Montiel c, T. Rueda-Rueda a, A. Castilla-Lázpita d, A. Romera-Piñero a, A. Medina-Tapia a

a Unidad de Gestión Clínica de Oftalmología, Hospital Universitario Virgen del Rocío, Sevilla, Spain
b Centro de Salud de Vejer, Vejer de la Frontera, Cádiz, Spain
c Servicio de Oftalmología, Hospital Universitario Reina Sofía, Córdoba, Spain
d Servicio de Oftalmología, Hospital INGESA, Melilla, Spain

ARTICLE INFO

Article history:
Received 17 November 2013
Accepted 9 February 2014
Available online 5 March 2015

Keywords:
Terson’s syndrome
Macular atrophy
Macular hemorrhage
Subarachnoid hemorrhage
Cerebral aneurysm

ABSTRACT

Case report: The case is presented on a 63-year-old patient with Terson’s syndrome who complained of loss of visual acuity. The optical coherence tomography showed macular atrophy.

Discussion: The patient developed macular atrophy probably secondary to macular hemorrhage caused by the rupture of a cerebral aneurysm.

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Atrofía macular en el síndrome de Terson

RESUMEN

Caso clínico: Se presenta el caso clínico de un varón de 63 años con una atrofía macular en el curso de un síndrome de Terson. El paciente mostraba una disminución de la agudeza visual con adelgazamiento macular observado en la tomografía óptica de coherencia.

Discusión: El paciente presentó una atrofía macular probablemente secundaria a una hemorragia en polo posterior, tras síndrome de Terson causado por la rotura de un aneurisma cerebral.

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* Corresponding author.
E-mail address: laura.frau.a@gmail.com (L. Frau-Aguilera).

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Introduction

In 1900, Terson described the association between acute subarachnoid hemorrhage and vitreous hemorrhage. At present, this syndrome comprises vitreous and retinal intraocular hemorrhages caused by intracranial bleeding.

Complications described in the Terson’s syndrome include loss of vision, formation of macular holes, epiretinal membranes and/or retinal folds as well as the development of proliferative vitreoretinopathy and retinal detachment.

A Terson’s syndrome case is presented in which the patient experienced loss of visual acuity (VA) associated to significant macular thinning in both eyes.

Clinic case

Patient aged 63 admitted to the Intensive Care Unit due to subarachnoid hemorrhage Fisher Grade III secondary to aneurysm rupture in the anterior communicating artery. The aneurysm was submitted to endovascular treatment by means of arteriography which achieved the closure thereof without additional complications.

The patient was examined for the first time 5 weeks after admittance in the hospital room, finding significant visual acuity loss. Intrinsic and extrinsic ocular motility were normal. Ocular fundus (OF) examination revealed hemorrhage in vitreous as well as pre-, intra- and subretinal hemorrhages. The optic discs (OD) were normal.

At week 6 of evolution, the patient was examined in the practice, exhibiting VA of 0.1 in RE and 0.04 in LE. Intraocular pressure (IOP) was 12 mmHg in both eyes. Biomicroscopy did not reveal pathological findings. The most relevant findings appeared in OF, where slight vitreous turbidity was observed due to the presence of blood together with the existence of hemorrhages in the macula and along the arcs in pre-, intra- and subretinal locations. The OD did not exhibit edema or alterations (Figs. 1 and 2).

Fluorescein angiograph (AGF) performed during week 8 revealed the presence of macular hypo-fluorescence due to blockage which did not exhibit modifications in late times, corresponding to subretinal hemorrhage at that level (Figs. 3 and 4).

Optical Coherence Tomograph (OCT) (Topcon® 3D OCT-1000, Topcon Corporation, Tokyo, Japan) carried out 4 months later when the hemorrhages were resolved revealed a significant retinal thickness reduction at the macular level, with a thickness of 111 μm in RE and 107 μm in LE (Figs. 5 and 6).

Fig. 1 – Right eye retinography showing slight vitreous turbidity, pre- and subretinal hemorrhages. Optic disc appears to be normal.

Fig. 2 – Left eye retinography showing pre- and subretinal hemorrhages. Optic disc normal.

Fig. 3 – Right eye angiography, showing screen effect due to macular subretinal hemorrhage.

Fig. 4 – Left eye angiography, subretinal macular hemorrhage.
Fig. 5 – Right eye OCT showing diminished foveal thickness and loss of external and internal photoreceptor strip.

Fig. 6 – Left eye OCT showing macular atrophy.
Discussion

Terson’s syndrome appears in 8–14.5% of subarachnoid hemorrhages and accounts for 5.5% of non-traumatic or diabetic vitreous hemorrhages. The presence of intraocular hemorrhage seems to be related to the severity of the subarachnoid hemorrhage, the most frequent cause being aneurysm rupture in the brain, specifically aneurysms located in the anterior communicating artery.

Several mechanisms have been described to explain the association between intraocular bleeding and subarachnoid hemorrhage, but the most broadly accepted explanation assumes that the ocular hemorrhage is caused by sudden intracranial pressure increases which cause rapid increases in the intraocular venous pressure with ensuing bleeding from the peripapillary retinal vessels.

The nature of the damage produced by subretinal hemorrhages could be due to at least 4 effects: the toxicity of iron on receptors, the shear forces upon receptors due to the contraction of the clot, obstructed distribution of oxygen, glucose and other vital substances for the external retina from choroidal circulation and metabolic deprivation and irreversible toxicity suffered by the retina pigment epithelium.

The visual prognostic after subretinal hemorrhage is variable and largely depends on the amount of blood and above all the cause of the hemorrhage. The best prognostic can be assumed when the hemorrhage is not due to choroidal neovascular membranes as in this case.

The patient of the present case exhibited poor VA which could be due to the macular atrophy demonstrated by OCT, probably secondary to the effects derived from the existence of a large macular subretinal hemorrhage (VA and macular condition prior to the cerebral-vascular accident were unknown). Accordingly, the tomographic image revealed significant macular thinning at the expense of the external layers with loss of the strip corresponding to the external and internal photoreceptor segments.

The differential diagnostic should include other causes of macular atrophy such as vascular and inflammatory alterations of the retina, sickle cell retinopathy, toxic diseases such as chloroquine retinopathy, macular infarcts due to the administration of intravitreal aminoglycosides, optic neuropathy, neurological diseases such as multiple sclerosis or spinocerebellar ataxia, as well as degenerative diseases such as myopia magnus and retinal dystrophies.

Terson’s syndrome complications described in the literature do not include the macular atrophy of the present case, which would appear as a consequence of the subretinal hemorrhage. Accordingly, the authors consider that early assessment and treatment of these patients is essential whenever the general condition allows it.

Conflict of interests

The authors declare no conflict of interest.

References