



Ischemic Damage of the Retinal Ganglion Cell Layer in Pseudoexfoliative Glaucoma: A Case Report

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Abstract

Pseudoexfoliative Glaucoma (PXG) patients have been shown to have impaired ocular blood flow. For this reason, in these patients, any added vascular alteration can cause ischemic lesions with functional repercussions on vision despite maintaining good control of the glaucomatous pathology.

We present the case of a 67-year-old man with a 10-year history of PXG, who presented an acute onset inferior nasal scotoma in his right eye. After a normal campimetric study, a ganglion cell layer analysis was made by means of optical coherence tomography, where a focal defect congruent with the symptoms presented by the patient was evidenced. Using complementary tests, an ipsilateral internal carotid artery stenosis was diagnosed.

Introduction

The susceptibility of Retinal Ganglion Cells (RGC) to ischemic damage and the cellular response pathways that converge causing apoptosis have been described by many authors (Figure 1) [1].

This susceptibility to damage becomes clinically relevant in Pseudoexfoliative Glaucoma (PXG), as this condition is associated with impaired ocular blood flow. In PXG, deposits of fibrillar material have been found within the walls of the Central Retinal Artery (CRA), Central Retinal Vein (CRV) and Short Posterior Ciliary Arteries (SPCA) causing alterations in functional parameters such as the peak systolic velocity both in the CRA as in the SPCA [2].

RGC monitoring has a predictive role in glaucoma progression that has been equated with that of the thinning of the per papillary Retinal Nerve Fiber Layer (RNFL) [3,4].

Case Presentation

We present the case of a 67-year-old male with a 10 history of PXG, treated with brimonidine OU with which he maintained an Intraocular Pressure (IOP) of around 15 mmHg. On ophthalmological examinations he had a cup to disc ratio of 0.3 in his right eye (OD) and 0.2 in his left eye (OS), with no focal defects of the Neuroretinal Ring (NRR) OU and no areas of thinning in the RNFL. There were no focal Visual Field (VF) defects.

During a follow up visit, the patient referred an acute onset inferior nasal scotoma in his right eye. He denied any other ophthalmologic or systemic symptom.

A central 30° perimetry was performed (OCTOPUS 900, Haag-Streit Diagnostics) (Figure 2), without any significant alterations. However, the RGC analysis on a 512 × 128 macular cube (Cirrus HD-OCT 4000, Zeiss) showed a focal thinning of the RGC layer in the superior temporal region of the macula congruent with the patient's perceived scotoma (Figure 3).

With these findings, a supra-aortic vessels Doppler ultrasonography was requested and showed a moderate stenosis of the right Internal Carotid Artery (ICA) ipsilateral to the RGC layer thinning.

We concluded that, despite controlled IOP values, the moderate ICA stenosis together with the impaired retinal blood flow caused by the PXG triggered focal RGC layer damage.

Discussion

Hypoxic damage to the retina is known to cause the expression of several apoptosis regulatory genes such as BAX, a homologue of Bcl-2. Furthermore, it decreases the production of Vascular Endothelial Growth Factor (VEGF), Nitric Oxide (NO), proinflammatory cytokines, and promotes the accumulation of extracellular glutamate. The RGC are particularly susceptible to all these

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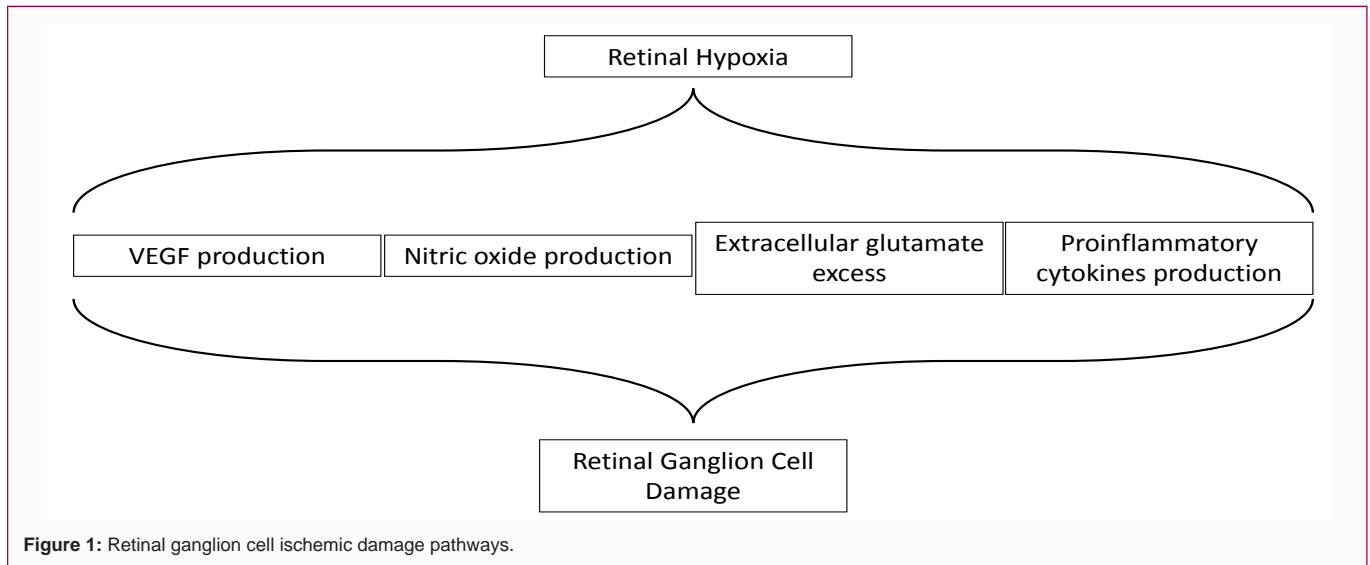


Figure 1: Retinal ganglion cell ischemic damage pathways.

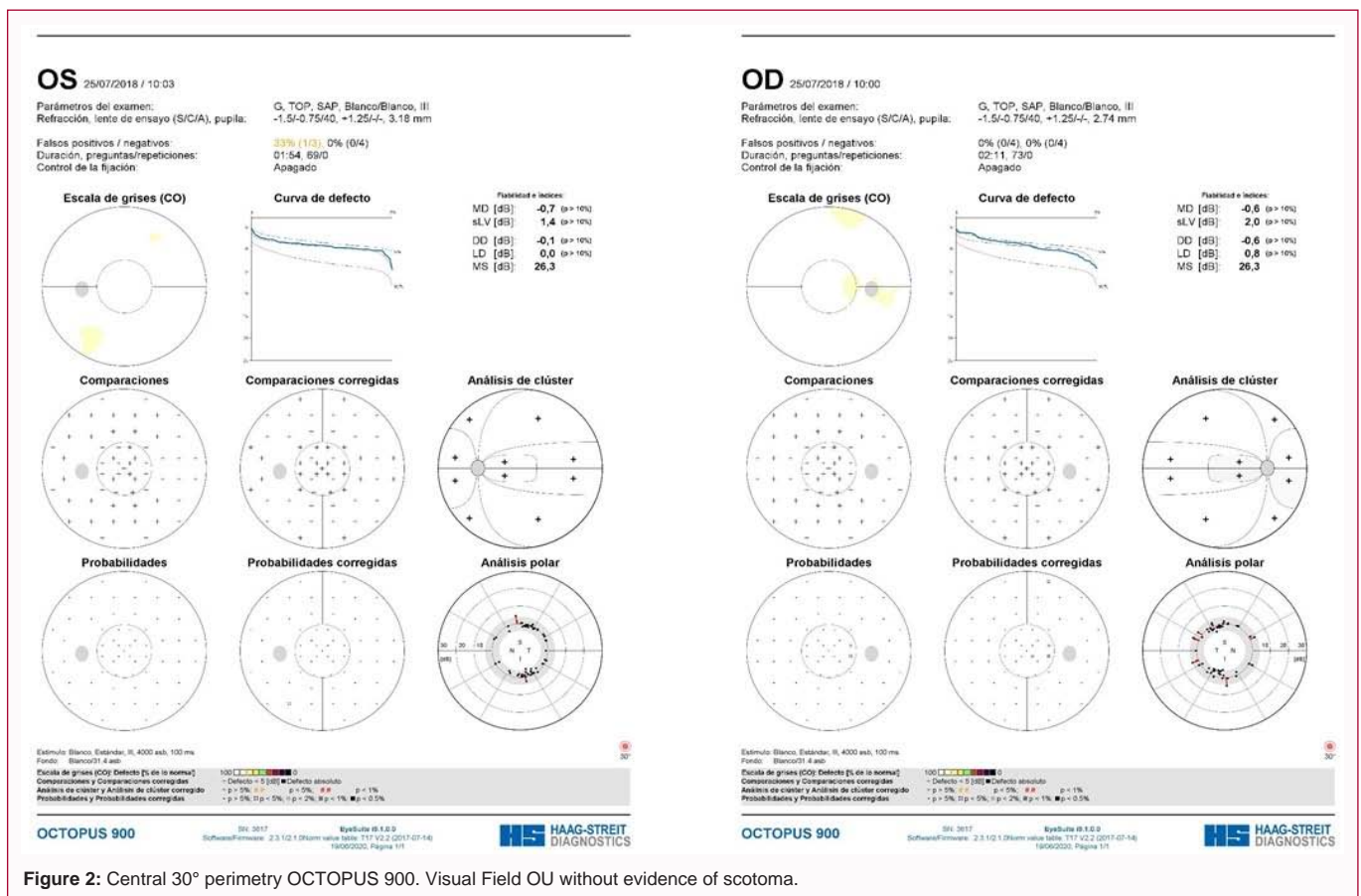


Figure 2: Central 30° perimetry OCTOPUS 900. Visual Field OU without evidence of scotoma.

changes that promote cell death (Figure 1) [1].

PXG is associated with alterations in circulation parameters in the eye; this relates with deposits of fibrillar material within the arterial walls of the vessels that supply the eyeball. Alteration of local hemodynamic values such as peak systolic velocity and arterial resistance indices of the central retinal artery, short posterior ciliary arteries and the ophthalmic artery have been shown in patients with PXG [2].

Although in glaucoma patients, the risk of macular damage

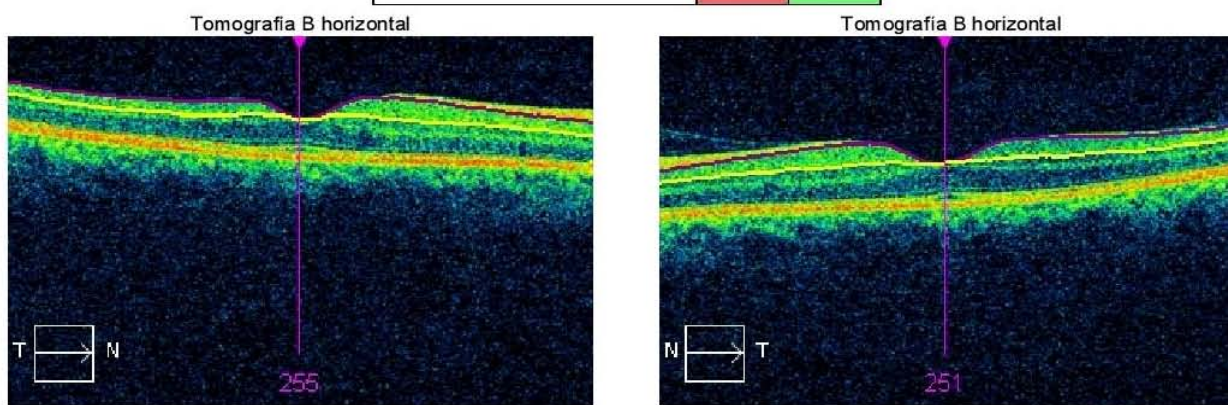
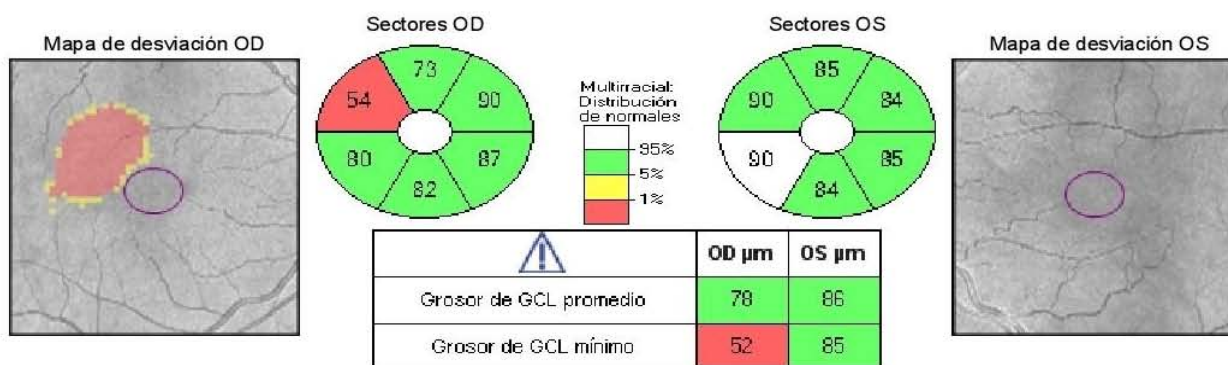
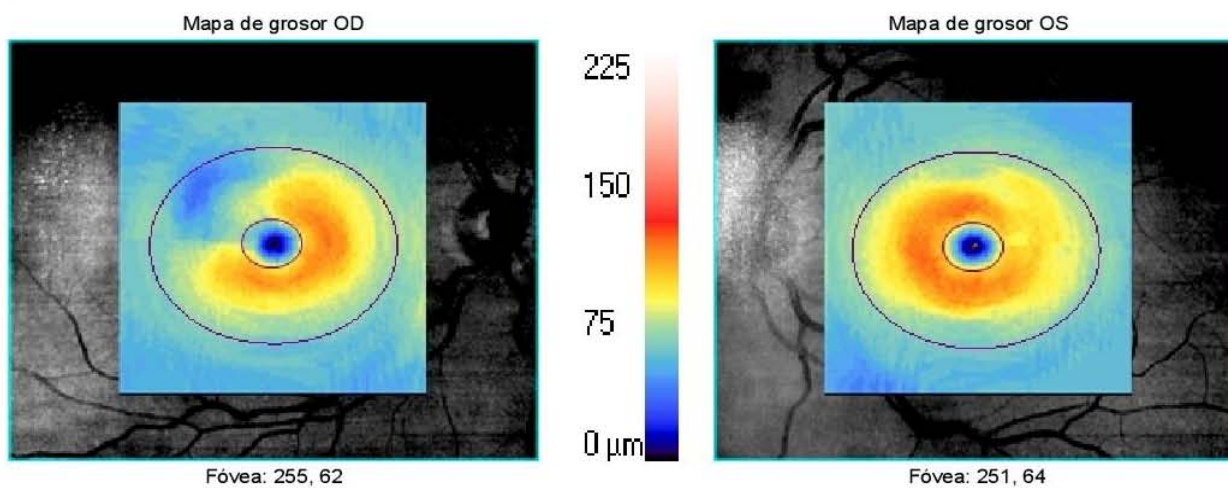
due to loss of RGC correlates with both the VF Mean Defect (MD), and the thinning of the RNFL, in glaucoma and in chronic ocular ischemic lesion due to vascular alterations, macular thinning with loss of the RGC layer may precede these findings. This was the case of our patient in whom the MD of the VF and the RNFL thickness was normal.

The pattern of RGC loss found in our patient matches with what is described in the literature for patients with different types of glaucoma with high IOP values [5]. It's our hypothesis that the

Nombre: OD OS
 ID: 16640302 Fecha de examen: 27/07/2018 27/07/2018
 Fecha de nacimiento: 09/08/1951 Hora del examen: 14:30 14:32
 Sexo: Masculino Número de serie: 4000-6219 4000-6219
 Técnico: Operator, Cirrus Intensidad señal: 9/10 9/10



Análisis de células ganglionares: Macular Cube 512x128 OD ● OS



Comentarios Firma del doctor

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Figure 3: Cirrus HD-OCT 4000 Optical coherence tomography. Macular cube 512 × 128. Ganglion Cell Layer analysis, with thinning in the superior temporal region OD.

hypoxic injury caused by the carotid stenosis aggravated the loss causing the symptomatology referred.

Another important fact to highlight is the absence of campimetric findings despite the scotoma perceived by the patient. In everyday practice, most glaucoma patients are evaluated with perimetry protocols where the evaluation points are 6° apart from each other, this means that the central macular area is not adequately registered [4]; this becomes important when evaluating patients with subjective central visual defects that are not evidenced in campimetric studies.

This case highlights the susceptibility of patients with PXG to ischemic damage due to ocular blood flow alterations and demonstrates the usefulness of the RGC layer analysis as a complementary analysis to detect early damage in patients with otherwise normal tests.

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