Short communication

Peripapillary intrachoroidal cavitation in pathological myopia

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ABSTRACT

Case report: A 54-year-old woman with pathological myopia, presented with an elevated, yellowish-white lesion at the inferior border of the myopic conus in her left eye. The optical coherence tomography (OCT) demonstrated an intrachoroidal hyporeflective space. The fluorescein angiography examination (FA) showed early hypofluorescence with delayed staining, with no leakage of contrast.

Discussion: Recognition of «peripapillary intrachoroidal cavitation» as an own entity associated with pathological myopia is important to avoid confusion with other possible retinal lesions which require further investigation and treatment.

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Excavación intracoroidea peripapilar en miopía patológica

RESUMEN

Caso clínico: Mujer de 54 años, miope magna, que presentaba una lesión sobreelevada y blanco-amarillenta en la parte inferior del conus miópico de su ojo izquierdo. La tomografía de coherencia óptica (OCT) mostró un espacio hiporreflectivo intracoroideo. En la angiografía con fluoresceína (AFG) se apreciaba una hipofluorescencia precoz con tinción tardía, sin fuga del contraste.

Discusión: El reconocimiento de la «excavación intracoroidea peripapilar» como una entidad propia asociada a la miopía patológica es importante para evitar la confusión con otras posibles lesiones retinianas susceptibles de más estudios y tratamiento.

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Introduction

Pathological myopia is defined as any refractive error equal to or greater than −6 D and axial length greater than 26 mm. It affects 0.5% of the general population and 30% of myopic individuals. Progressive anteroposterior elongation of the globe is responsible for funduscopic changes, i.e., posterior staphyloma, peripapillary conus, lacquer striae or focal chorioretinal atrophy.

In 2003, Freund et al.\(^1\) reported a peripapillary disease in pathological myopia patients, which they call “peripapillary detachment.” It consisted of an elevated, orange-yellowish lesion on the inferior border of the conus, which caused elevation in the neurosensory retina and retinal pigment epithelium (RPE). Its prevalence in pathological myopia is 4.9%\(^2\).

Subsequently, Toranzo et al.\(^3\) suggested the term “intrachoroidal peripapillary cavitation,” when OCT reveals an intrachoroidal hyporeflective area beneath an unaltered RPE.

Recently, Spaide et al.\(^4\) have studied morphological changes and pathophysiologic mechanisms of this lesion by spectral domain OCT.

We report the case of a patient with pathological myopia related to peripapillary intrachoroidal cavitation.

Case report

A 54-year-old female referred to the retina unit for an asymptomatic inferior border peripapillary lesion in her left eye (OS). Her ophthalmological history indicates she had undergone astigmatic myopic LASIK surgery (axial length 27.85 mm) and was using latanoprost in both eyes.

Best corrected visual acuity was 0.7 in her right eye (OD) and 0.8 in her left (OS). Anterior segment biomicroscopy was normal. Intraocular pressure (IOP) was 17 and 18 mmHg in OD and OS with treatment. Funduscopic

Fig. 1 – Elevated, yellowish-white lesion with diffuse borders on the lower end of peripapillary conus.

Fig. 2 – Hyporeflective intrachoroidal space (white arrow) located beneath RPE (blue dashed lines). The yellow arrow points to a thinning of choroid thickness and the red arrow a slight displacement of posterior sclera.

Fig. 3 – Early hypofluorescence of the lesion.

Fig. 4 – Late staining of lesion without contrast leakage.
examination in OS showed oblique papilla with 4/10 cavitation and inferior temporal conus. Its inferior border revealed an elevated, yellowish-white and diffuse-border lesion of approximately one disk diameter, which hampered visualization of the underlying choroidal vessels (Fig. 1). Retina showed slight diffuse chorioretinal atrophy.

OCT lesion examination with Cirrus (Carl Zeiss Meditec Inc., Dublin, CA) showed an intrachoroidal hyporeflective space beneath RPE (Fig. 2 White arrow and blue dashed lines, respectively). Likewise, choroid thinning thickness was found (Fig. 2, yellow arrow) along with slight posterior sclera displacement (Fig. 2, red arrow).

FA showed early hypofluorescence in the lesion with late staining, without contrast leakage (Figs. 3 and 4).

Ocular ultrasound and vision field assessment were performed, showing no abnormalities (Fig. 5).

Additional tests ruled out retinal lesions amenable to treatment, continuing with patient follow-up. Lesion has remained stable for 2 years.

**Discussion**

A comprehensive differential diagnosis must be performed for a peripapillary lesion with the reported characteristics.

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**Fig. 5** – Visual field without significant alterations.
Peripapillary choroidal neovascularization would show hemorrhage, dye leakage under FA and would change toward progression or disciform scar. Idiopathic polypoidal choroidal vasculopathy would show dilated choroidal vascular channels, hemorrhage and exudation.

For a central serous chorioretinopathy we would have found RPE serous neurosensory in OCT with corresponding angiographic findings.

Exophytic retinal hemangioblastoma would include edema, exudates and hemorrhages and tumor hyperfluorescence in angiography.

Choroidal tumor metastases would appear as multifocal lesions, without predilection for the peripapillary area or myopic eye. Large exudative detachments would occur with size variation.

Abnormalities were ruled out in our patient’s optic nerve (drusen, hypoplasia or dysplasia, coloboma, optic disk pit).

Multiple hypotheses have been proposed for the lesion’s origin (incomplete choroidal coloboma, vitreous fluid passing into the subretinal space). Spaide et al. suggest a series of pathophysiologic events. First, the loss of overlying layers at conus level causes this area to be susceptible to deformation. Posterior sclera slope may contribute to intrachoroidal cavitation expansion. Choroidal thinning at the optic nerve junction may stretch border tissues of Jacoby (a thin astrocyte layer separating choroid from optic nerve), whose rupture would cause communication from intrachoroidal cavitation to vitreous cavity, allowing IOP to be applied directly on the sclera. We found no such communication in this patient.

There is disagreement about the prevalence of perimetric defects in pathological myopic subjects, with or without peripapillary choroidal cavitation. Our patient had no fiber bundle defects in her OS.

Although new spectral domain OCT allows visualization of deep eyeball structures, further studies are still needed to clarify the origin of these lesions and possible relation to glaucomatous defects.

In view of this case, we believe it is important to recognize intrachoroidal peripapillary cavitation in clinical practice as a single condition related to pathological myopia to avoid confusion with other possible retinal lesions amenable to further study and treatment.

**Conflict of interest**

The authors declare that they have no conflicts of interest.

**REFERENCES**