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RESEARCH ARTICLE

SPONTANEOUS ASSOCIATION OF MACULAR HOLE AND CHOROIDAL NEOVASCULARIZATION: A CASE REPORT

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Abstract

Myopic maculopathy is characterized by the presence of degenerative abnormalities of the central retina. We report the co-existence of a macular hole and atrophic choroidal neovascularization in a highly myopic patient. The occurrence of this association in the same eye without a history of intraocular surgery has been reported very rarely in the literature. OCT is a reliable diagnostic tool for a full thickness macular hole. When an atypical-appearing macular hole is detected, fluorescein angiography and OCT-A should be performed to detect associated choroidal neovascularization. The association described is of exceptional occurrence but with a serious prognosis and very limited chances of visual recovery.

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Introduction:-

Pathological myopia is defined by an axial length above a certain threshold (usually 26.5 mm), corresponding to a refractive error of at least -6.0 diopters, accompanied by degenerative structural changes.

Myopic maculopathy is characterized by the presence of one or more of the following changes: Posterior staphyloma, rupture of Bruch's membrane, choroidal neovascularization, macular hole and posterior polar chorioretinal atrophy. (1)

The occurrence of choroidal neovascularization with macular holes in the same eye without a history of intraocular surgery has been reported very rarely in the literature.

Our work reports the coexistence of macular holes associated with choroidal neovascularization at the atrophic stage in a highly myopic patient, as well as a review of the literature in order to determine the pathogenesis, the clinical presentation and the diagnostic approach of this atypical association.

Case Report:

A 58-year-old female patient, with high myopia in both eyes : at -8 D in the right eye and -10 D in the left eye, presented to us with a progressive decrease in visual acuity in the right eye for several months.

The corrected visual acuity was finger movement in the right eye and 6/10 in the left eye according to the Snellen scale.

The fundus of the right eye detected a macular hole associated with a large area of macular atrophy and changes in the retinal pigment epithelium (Figure 1A).

Fluorescein angiography shows a window effect in the macula related to macular retinal atrophy and reveals a progressive dye impregnation in the perifoveolar area from the early phase, well limited without diffusion characteristics at the late stage, pointing to a neovascular scar (Figure 1B and 1C).

Spectral domain OCT (optical coherence tomography) revealed a stage 4 full thickness macular hole associated with a well-limited hyperreflective lesion in front of the plane of the retinal pigment epithelium compatible with a type 2 pre-epithelial choroidal neovascularization. The absence of exudative signs indicates the inactive nature of the choroidal neovascularization. (Figure 2).

On OCT-A (OCT- angiography), the neovessel is visualized in the choriocapillaris layer. (Figure 3).

Discussion:-

Only a handful of cases with a macular hole with choroidal neovascularization in the same myopic eye have been reported.

The majority of cases reported in the literature have presented choroidal neovascularization after a first observation or repair of a macular hole. The pathogenesis can then be divided into operative and non-operative causes. In non-operative cases, the hypothesis is that chronic retention of subretinal fluid under a macular hole produces inflammatory mediators that will disrupt Bruch's membrane leading to the development of choroidal neovascularization. (2)

Another hypothesis suggests that macular hole development may follow choroidal neovascularization.

This hypothesis was proposed following the study by Shimada et al. In this study, the incidence of macular holes was relatively high (14.0%) in myopic eyes in the atrophic stage of choroidal neovascularization. It has been suggested that the fibrovascular tissue of the neovessel produces adhesion between the choroidal neovascularization and the neurosensory retina (3). In parallel, a histopathologic study performed on myopic human eyes in the atrophic stage of myopic choroidal neovascularization showed the presence of loss of the pigment epithelium layer and the outer layers of the retina in the area of chorioretinal atrophy (4). Together, these two observations have led to the pathogenic hypothesis that the retraction and regression of the choroidal neovessel and the enlargement of the chorioretinal atrophy causes centrifugal and tangential stretching of the extremely thin atrophic retina and ultimately causes the formation of a macular hole at the edge of the choroidal neovascularization (3).

When the neovessel is active, it appears on fluorescein angiography by a well-defined hyperfluorescence in the early stages and a diffusion of the dye in the late stages.

When it is inactive, the fibrosis becomes impregnated with dye and appears progressively hyperfluorescent, well limited, but does not diffuse at late times, as in our case.

Angiographic diffusion, even in the absence of exudation on OCT, is an argument for treatment. (5) (6)

OCT is a non-invasive method for the quantitative assessment of retinal thickness, it is the gold standard for the management of myopic choroidal neovascularization. The vast majority of myopic choroidal neovessels are classically located above the pigment epithelium layer and therefore easily visualized on OCT. They have the appearance of a hyper-reflective dome-shaped elevation above the pigment epithelium with negligible subretinal fluid accumulation. When active, it is often poorly limited, whereas a well-limited lesion with a hyperreflective shell points to an inactive lesion, which is sometimes accompanied by signal attenuation behind the choroidal thickness.

OCT can be useful to identify coexisting pathologies related to myopia, such as macular hole, retinoschisis and atrophic patches. (1) (6)

Although fluorescein angiography and structural SD-OCT remain the diagnostic gold standard for choroidal neovascular activity, they have inherent limitations that are partially offset by A-OCT.

OCT does not provide functional information of the retinal microcirculation. Fluorescein angiography does not provide 3D images and requires intravenous injection of dye that can cause side effects ranging from nausea to severe anaphylactic reactions, thus limiting its application.

OCT-A could be a useful tool to evaluate patients with high myopia, especially patients with a history of allergy. (5)

A study by Lea Querques et al proposed an OCT-A description of the morphological characteristics of myopic choroidal neovascularization correlating with different degrees of neovascular activity. In many cases, myopic choroidalneovessels had an irregular shape, with ill-defined margins, and no visible nucleus.

Active myopic choroidalneovessels appeared to be predominantly intertwined, whereas in inactive forms, the neovascular network appeared to be predominantly entangled.

The entangled network was defined on OCT-A by a loose appearance with long filamentous vessels, a few large branches, and a thick vessel wall. Several myopic lesions may interfere with the detection of myopic choroidalneovessels on OCT-A: extensive myopic staphyloma, bruch's membrane ruptures, retinal pigment epithelium atrophy, and retinoschisis. (5)

The prognosis of a macular hole developed at the edge of an atrophic CNV is not known. The incidence of retinal detachment in strong myopic eyes with a macular hole is 97.6%. This incidence was higher when there was concomitant severe chorioretinal atrophy. (3)

Patients with large areas of chorioretinal atrophy do not show additional visual acuity loss during the development of the macular hole probably because they already have severely impaired vision. Periodic OCT examinations are recommended for macular holes and retinal detachment in asymptomatic patients with myopic choroidal neovascularization even when the neovessel has completely regressed. (3)

Conclusion:-

Although this polypathology presented is associated with an exceptional occurrence, it has a significant prognosis and a very limited chance of visual recovery. OCT is a reliable tool for diagnosing full-thickness macular hole. If an atypical macular hole is confirmed, fluorescein angiography and A-OCT should be performed to detect associated choroidal neovascularization. (1)

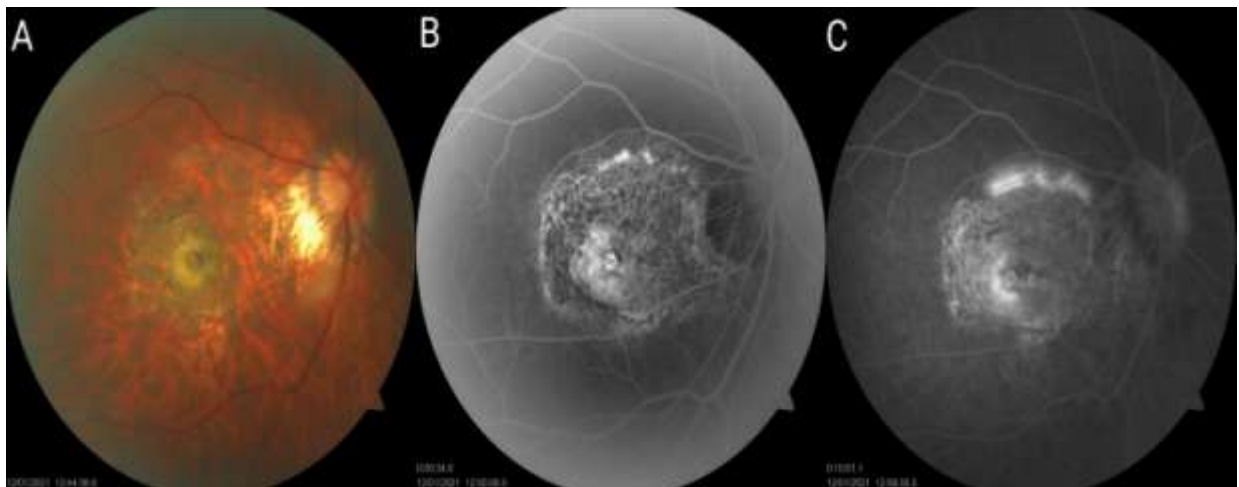


Figure 1:- Fundus photograph of the right eye :Chorioretinal atrophy is observed around the Choroidal neovascularization with a detectable macular hole (A). Angiographic sequence showing a window defect corresponding to the area of chorioretinal atrophy. A perifoveolar impregnation of fluorescein is also observed from the early stage (B) without diffusion at the late stage (C) suggestive of an inactive type 2 (classic) choroidalneovessel.

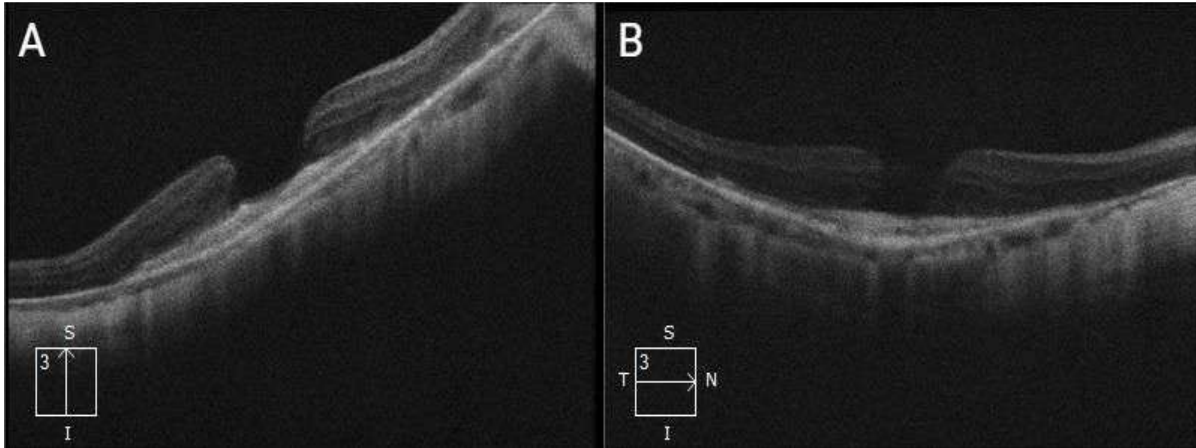


Figure 2:- Vertical (A) and horizontal (B) B-scans showing a full thickness stage 4 macular hole associated with hyperreflective material above the retinal pigment epithelium layer with no associated exudative signs in favor of inactive choroidal neovascularization type 2.

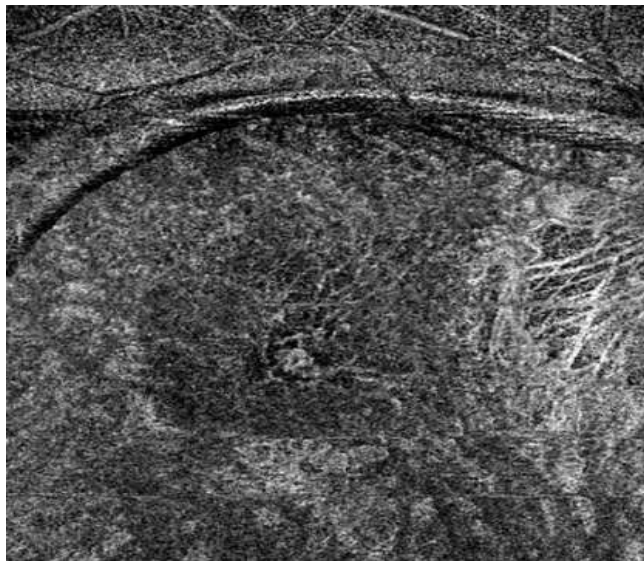


Figure 3:- OCT-A: scan passing through the choriocapillaris allowing visualization of the choroidal neovascularization.

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