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Case Report

Bilateral Extramacular Choroidal Neovascularization in Wet Age-Related Macular Degeneration

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Abstract

Purpose: Choroidal neovascularization is a common complication following several retinal conditions, most commonly age-related macular degeneration (AMD). We present a case of bilateral extramacular choroidal neovascularization in a patient with wet AMD.

Methods/Patients: Interventional Case Report. Clinical, radiologic, and angiographic correlation.

Results: An 89 year old Caucasian male with Age-related Macular Degeneration (AMD) developed bilateral vitreous hemorrhages and subretinal hemorrhages after which were noted bilateral extramacular choroidal neovascular membranes. The vitreous hemorrhage in the left eye cleared on its own, while vitrectomy was performed in the right eye. Bevacizumab (Avastin) was injected into both eyes. Eighteen months after initial presentation and three months after Bevacizumab, the patient was asymptomatic, both neovascular membranes were inactive, and visual acuity had improved to 20/70 OD and 20/100 OS.

Conclusions: We report the first case of aggressive bilateral extramacular choroidal neovascular membranes in a patient with wet AMD. The pathophysiology that causes AMD to target the macula and fovea in most cases, but the extramacular region in this case, has yet to be elucidated.

Disclosures

None of the authors has a financial or proprietary disclosure or conflict of interest with the submission. No financial support was received for this submission. An Institutional Review Board (IRB) waiver was granted. All research was performed in accordance with the Declaration of Helsinki and all local, regional, and national law.

Summary statement

This is the first case of aggressive bilateral extramacular choroidal neovascular membranes in a patient with wet AMD. Eighteen months after initial presentation and three months after Bevacizumab treatment, both membranes were inactive, and visual acuity was improved. The development of bilateral extramacular choroidal neovascular membranes is atypical of AMD.

Introduction

Choroidal neovascularization (CNV) is a common complication following several retinal conditions, including age-related macular degeneration, uveitis, angioid streaks, ocular histoplasmosis syndrome, myopia, trauma, and tumors. CNV commonly affects the macula. We present the first case report of bilateral extramacular choroidal neovascularization in a patient with wet AMD.

Case report

An 89-year-old Caucasian man with a history of sick sinus syndrome, hyperlipidemia, aortic stenosis, mitral regurgitation, and low-grade prostate cancer presented with blurry vision in the left eye for 1 week without pain. Visual acuity was 20/60 OD and 20/150 OS. Dilated fundus examination revealed a dense vitreous hemorrhage in the left eye. An OCT and FA could not be performed in the setting of dense vitreous hemorrhage. B-scan ultrasonography (Figure 1A) revealed vitreous hemorrhage and 2 smooth dome shaped lesions in the left eye, suggestive of possible subretinal hemorrhages. MRI was performed to further characterize these masses and showed two small enhancing left ocular lesions with high T1 and low T2 signal: one was 5 mm and the second 2-3 mm (Figure 1B). The vitreous hemorrhage improved over several months and the masses resolved, revealing peripheral choroidal neovascular membranes.

At thirteen months, he presented with floaters in the right eye. Examination revealed a new, large subretinal hemorrhage anterior to the equator associated with extramacular choroidal neovascularization in the right eye and vitreous hemorrhage. Bevacizumab was administered

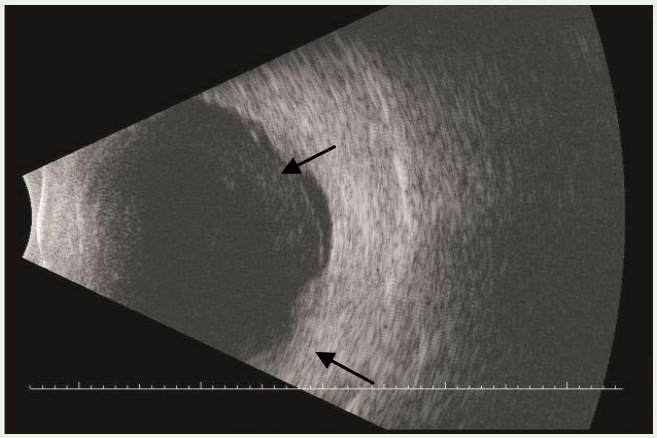

Figure 1A: B scan ultrasonography of the left eye transverse 3 o'clock view revealing 2 temporal, smooth, dome-shaped lesions (arrows) with associated vitreous detachment.

intravitreally in the right eye. After a month of nonclearing vitreous hemorrhage in the right eye, pars plana vitrectomy with gas and endolaser were performed.

The patient received injections of 1.25mg Bevacizumab twice OD and once OS. At eighteen months after initial presentation, his visual acuity was 20/70 OD and 20/100 OS, the extramacular choroidal neovascular membranes were inactive, and the vitreous hemorrhage had resolved in both eyes (Figure 2). Fluorescein angiogram demonstrated cystoid macular edema in the left eye (Figure 3) and age-related macular degeneration bilaterally.

Conclusions

CNV in wet AMD is highly selective, targeting the macula and the fovea, but the mechanism of this localization is unknown. The development of AMD with CNV has been linked to levels of complement factors H, B, and C3 [1-3]. In AMD, the macular tissues undergo thinning and pigment deposition, and the extracellular matrix undergoes several significant changes [4]. There is greater deposition of collagen I, lipids, and glycosaminoglycans in the

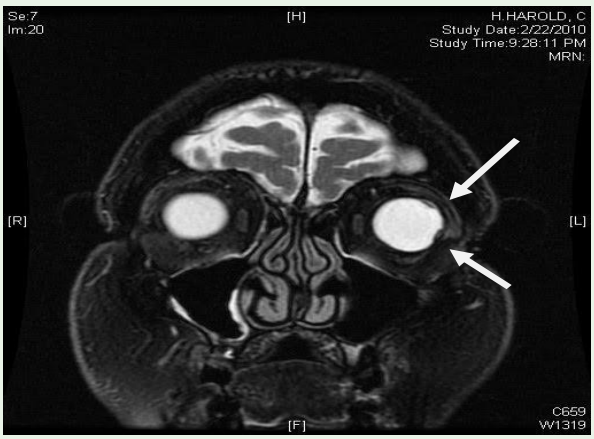

Figure 1B: Coronal MRI T2 reveals 2 small hypoenhancing lesions of the left eye (arrows), one in the upper outer quadrant and one in the lower outer quadrant, arising from the retina or choroid.

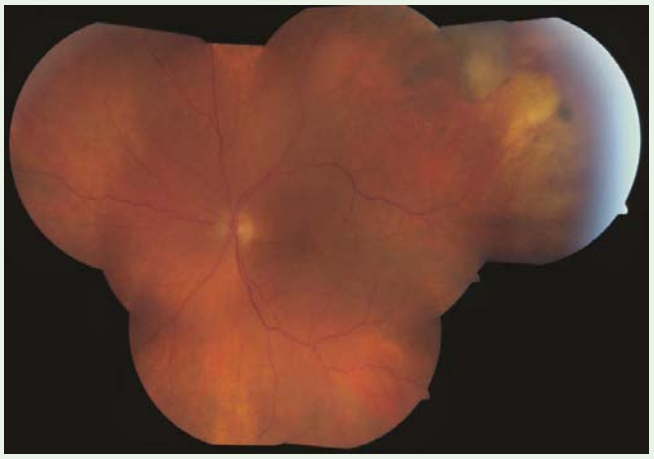

Figure 2: Montage fundus photograph left eye demonstrating superotemporal chorioretinal scar from prior extramacular choroidal neovascular membrane.

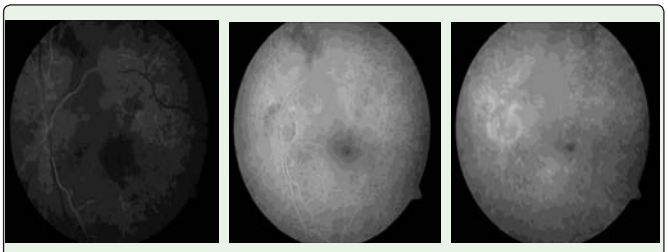

Figure 3: FA of the left eye (early, middle, late phases) demonstrates patchy choroidal filling and cystoid macular edema in this patient with known AMD.

submacular portion of the Bruch membrane than in the periphery, with greater levels of cross-linking and protein-containing debris [5]. There are also more inactive MMPs in the submacular region, leading to impaired degradation of ECM components and thickening [6]. A third contributory factor to CNV is a decrease in choroidal blood flow and blood volume - these are inversely related to the total area of retinal drusen and suggest ischemic change as an important mechanism of disease progression [7]. Decreased subfoveolar choroidal blood flow and loss of choriocapillary endothelial cell processes also limits the clearing of debris from the submacula [8]. These factors likely work in concert to predispose the fovea and macula to CNV.

This case report demonstrates, however, that AMD can be associated with aggressive extramacular choroidal neovascular membranes and retinal and vitreous hemorrhages. The treatment approach is similar to macular CNV to stabilize the extramacular membranes, preserve retinal function, and prevent vision loss, including bevacizumab and vitrectomy. Photocoagulation could also be considered in extramacular CNV, and laser treatment has been shown to improve the hemodynamics of the impaired choroidal blood flow seen in advanced AMD; improved circulation may also explain the disappearance of drusen following therapy [9]. The pathophysiology of extramacular CNV formation, the prevalence, and its significance is an area that has yet to be fully elucidated.

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