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American Journal of Ophthalmology Case Reports

Epiretinal membrane-induced intraretinal neovascularization

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ARTICLE INFO

ABSTRACT

Purpose: To report a 71-year-old male patient diagnosed with epiretinal membrane-induced intraretinal neovascularization.

Observations: The presence of an epiretinal membrane (ERM) was confirmed by Optical Coherence Tomography (OCT), fluorescein and indocyanine angiography. Optical coherence tomography angiography (OCT-A) revealed a neovascular membrane within the ERM. Intravitreal ranibizumab injections were administered three times at four-week intervals. Imaging revealed a stable membrane with no leakage. Five months after the third injection, OCT revealed intraretinal fluid. OCT-A showed a new branch of the neo-vascular membrane at the superficial capillary plexus. Following an additional ranibizumab injection, the membrane stabilized.

Conclusions and importance: It is conceivable that neovascularization developed due to, or in close conjunction with an epiretinal membranes already in place.

1. Introduction

Idiopathic epiretinal membranes are macular disorders with an incidence of 2–20 %, frequently diagnosed in the elderly.[1] ERMs are thought to result from fibroglial proliferation on the inner retinal surface secondary to small internal limiting membrane (ILM) breaks occurring during posterior vitreous detachment (PVD). ERM contraction or shrinkage may create irregular folds in the membrane itself, and exert anteroposterior or tangential tractional forces on the retina and retinal vasculature. Anteroposterior forces produce vertical traction and increases retinal thickness whereas tangential forces pull the superficial retinal layers away from their original position, causing retinal deformation and displacement.

ERMs also alter the morphology, location and permeability of the retinal vasculature. Morphological abnormalities include straightening and curling of retinal blood vessels and shrinkage of the foveal avascular zone (FAZ).[2] Eyes with ERMs often have macular edema and tortuous vessels with abnormal hemodynamics in the perifoveal capillaries and disturbance of the macular microcirculation.[3] Some reports suggest a partial similarity between idiopathic ERM and central retinal vein occlusion (CRVO)[4,5] and venous outflow from the macula can be impeded in eyes with ERM. Patients may experience metamorphopsia, micropsia, monocular diplopia, and, depending on the location, decreased visual acuity.

2. Case report

A 71-year-old Caucasian male, presented with gradual vision loss in his right eye (OD) over the past four months. Snellen best-corrected visual acuity (BCVA) was 20/50 OD and 20/20 in his left eye (OS). Fundus examination revealed an epiretinal membrane OD as well as sub-hyaloid and intraretinal hemorrhage inferior to the foveola (Fig. 1A) without any apparent PVD or any other rhegmatogenous, vascular, inflammatory or hamartomatous findings. No findings were observed in OS. Fluorescein angiography (FA) revealed leakage in both early and late phases, corresponding to the intraretinal neovascularization inferior to the foveola OD (Fig. 1B and C). Indocyanine green angiography confirmed the fluorescein angiography results.

Optical Coherence Tomography (OCT, Heidelberg Engineering, Ltd., Heidelberg, Germany) confirmed the presence of an epiretinal membrane in the macula OD with intraretinal cystic macular edema and a hyperreflective area in the inner retinal layer corresponding to the intraretinal hemorrhage (Fig. 1D). Optical coherence tomography angiography (OCT-A) revealed increased flow only in the retinal superficial capillary plexus, corresponding to an area of intraretinal neovascularization, a dense and apparently active membrane. There were

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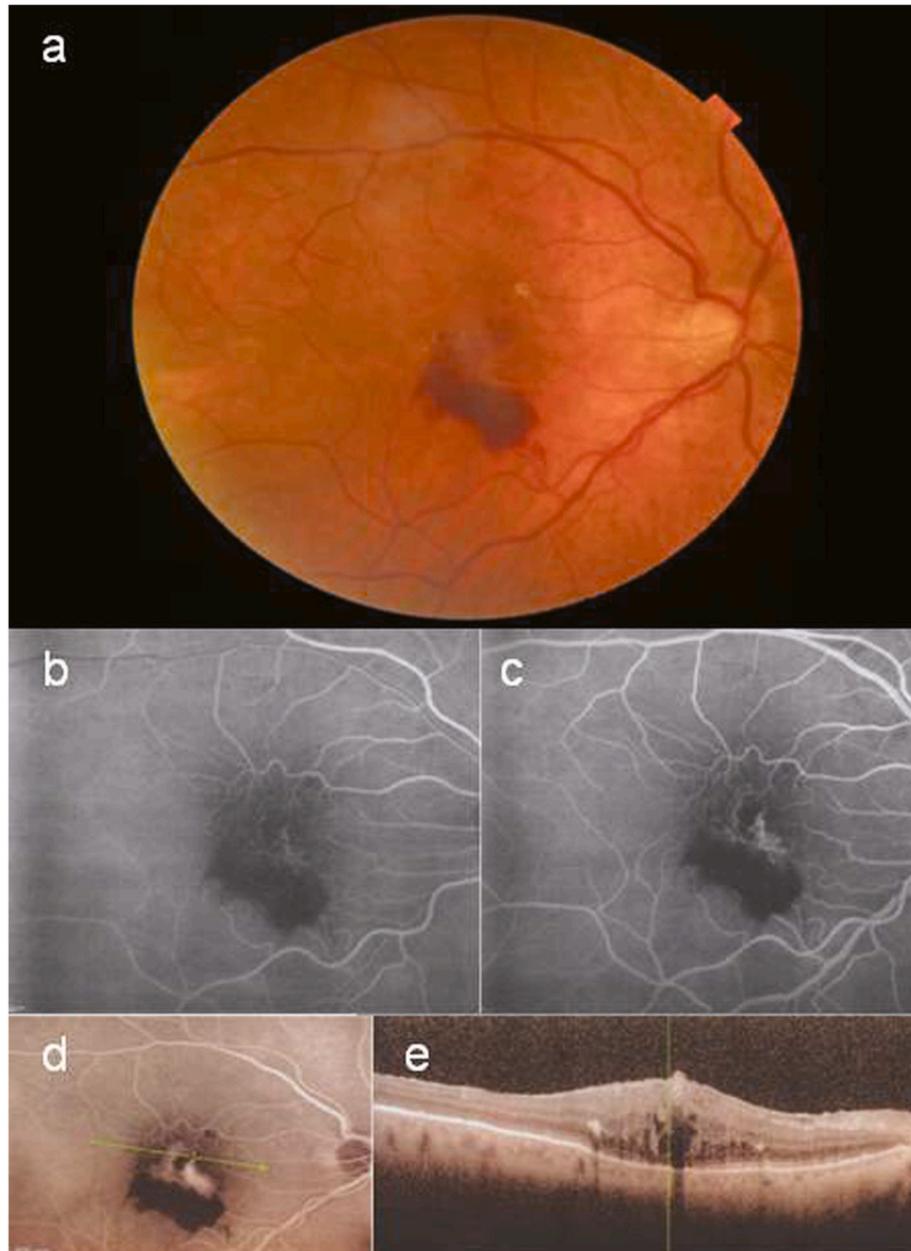
Fig. 1. a. Fundoscopic view at presentation; Subhyaloid and intraretinal hemorrhage inferior to the foveola.
b. Fluorescein angiography showing the intraretinal neovascular membrane, early arteriovenous phase, c. peak phase angiogram. d.-e. OCT scans of the right eye showing ERM and hyperreflective foci in the intraretinal edema in the macula.

no apparent changes in the deep capillary plexus, outer retina and choriocapillaris (Fig. 2). Intravitreal ranibizumab (0.5 mg, Lucentis, Novartis AG) injections were given OD three times at four-week intervals. One month after the third intravitreal injection, BCVA was stable with no intraretinal edema and only remnants of the neovascular tissue remained at the level of the epiretinal membrane in OCT-A (Fig. 3). OCT, OCT-A and clinical examination were repeated monthly for the next four months with no signs of intraretinal fluid. BCVA remained unchanged. Five months after the third injection, OCT revealed intraretinal fluid. OCT-A showed a new branch of the neovascular membrane at the superficial capillary plexus. One month after a fourth intravitreal ranibizumab injection, there was no intraretinal fluid while the OCT-A showed a stable membrane (Fig. 4). BCVA was 20/30 OD and 20/20 OS.
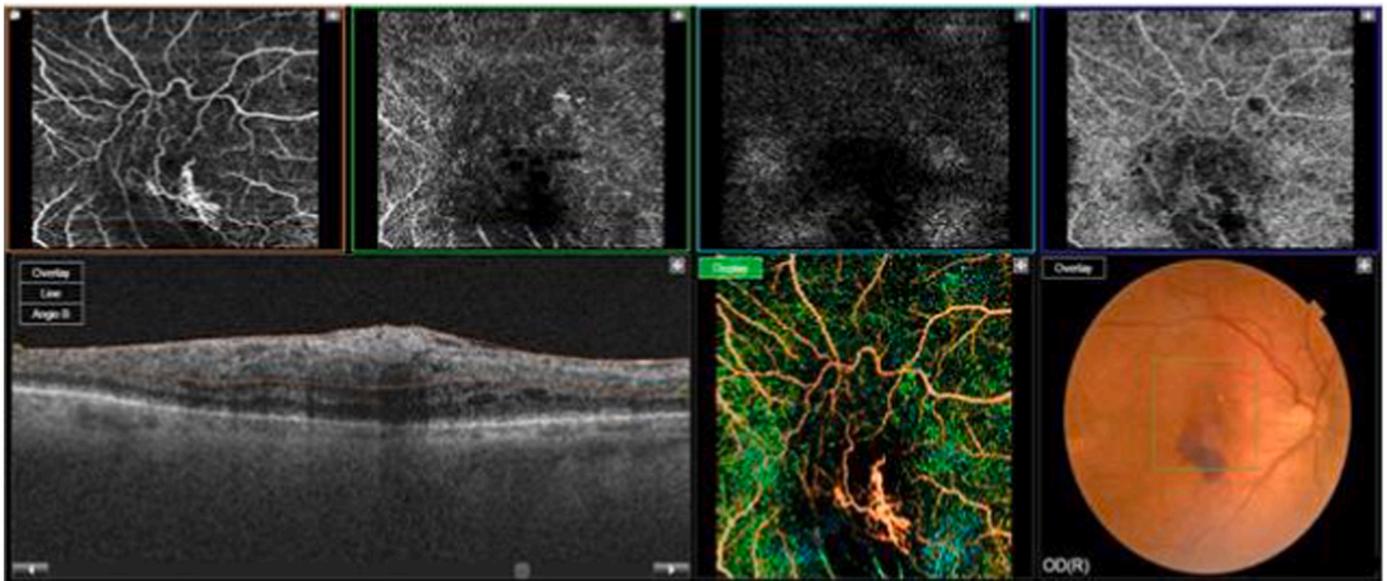
Fig. 2. Baseline visit. Deep capillary plexus, outer retina and choriocapillaris are depicted free of lesions.

3. Discussion

We describe a case of intraretinal neovascularization associated with an epiretinal membrane. To our knowledge there is no clear description of similar lesions in the literature. Epiretinal cellular proliferations can be divided into vascularized and non-vascularized membranes. Vascularized ERMs generally arise in the context of inner retinal ischemia secondary to retinal capillary nonperfusion (e.g. branch retinal vein occlusion (RVO) or diabetic retinopathy). Attached posterior vitreous is required as a scaffold for fibroglial proliferation.[6] In our patient, the posterior vitreous was attached.

Intraretinal neovascularization in our case could be the result of a perimacular branch RVO, a congenital retinal anomaly, an age-related macular degeneration (AMD) related lesion, neovascularization associated with telangiectasia. There was no personal history of diabetes mellitus or intraocular inflammation and no family history of ocular disease.

A macular branch RVO could potentially lead to the clinical manifestations reported here (ERM formation, retinal thickening and/or macular edema and collateral vessel development). Nevertheless, this fails to adequately explain the amount of blood in the sub-hyaloid and the intraretinal hemorrhage observed.

Congenital retinal hamartoma is a rare benign lesion, commonly diagnosed in children, consisting of glial cells, vascular tissue and RPE cells.[7] While OCT findings could be compatible with such a lesion, there were no corroborating findings on the OCT-A (as reported in similar cases).[8,9]

Retinal-retinal anastomosis as a manifestation of AMD crossing the horizontal raphe was another entity rejected due to lack of drusen. Moreover, following anti-VEGF treatment, lesion vessels resembled retinal collaterals and/or vascular remnants.

Macular telangiectasia was also considered. As Yannuzzi described, right angle vessels can form a network of proliferating vessels in the deep capillary plexus that may extend beneath the retina to form a subretinal network.[10] In our case, the existence of neovascularization in the superficial capillary plexus makes this diagnosis unlikely.

Another possible explanation could be epiretinal traction on a preexisting fragile telangiectasia. However, the limited retinal ischemia probably could not trigger a sufficient increase of VEGF production and facilitate formation of the observed neovascular membrane. Moreover, in this case, neovascularization should develop on the inner retinal side, between the ILM and the posterior hyaloid, not in the superficial capillary plexus. Conclusions It is conceivable that neovascularization developed due to or in close conjunction with the epiretinal membrane already in place. This could be attributed to idiopathic neovascularization secondary to the epiretinal membrane.

CRediT author statement

Ioannis Giachos: Investigation, Evangelia Chalkiadaki: Investigation, Konstantinos Andreanos: Resources, Chrysanthos Symeonidis: Writing - Review & Editing, Alexandros Charonis: Investigation, Ilias Georgalas: Project administration, Tryfon Rotsos: Writing - Original Draft, Writing - Review & Editing.

Patient consent

Consent to publish this case report has been obtained from the patient in writing.

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

Declaration of competing interest

The authors have no financial disclosures.
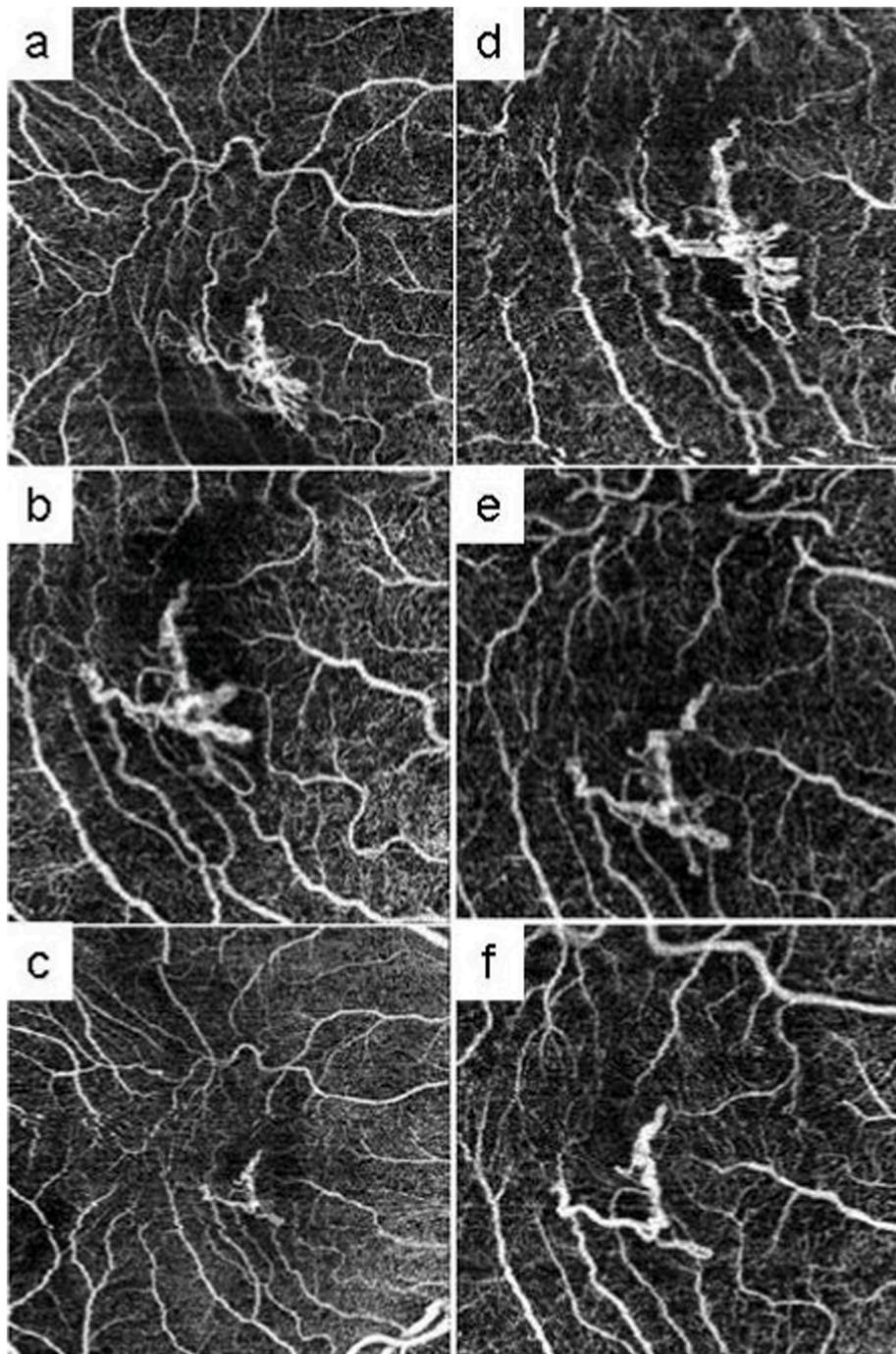
Fig. 3. OCT-Angiography: Neovascular membrane (a.) before the first injection, (b.) immediately after the first injection, (c.) one week after the first injection, (d.) one month after the first injection, (e.) one month after the second injection (f.) one month after the third injection.
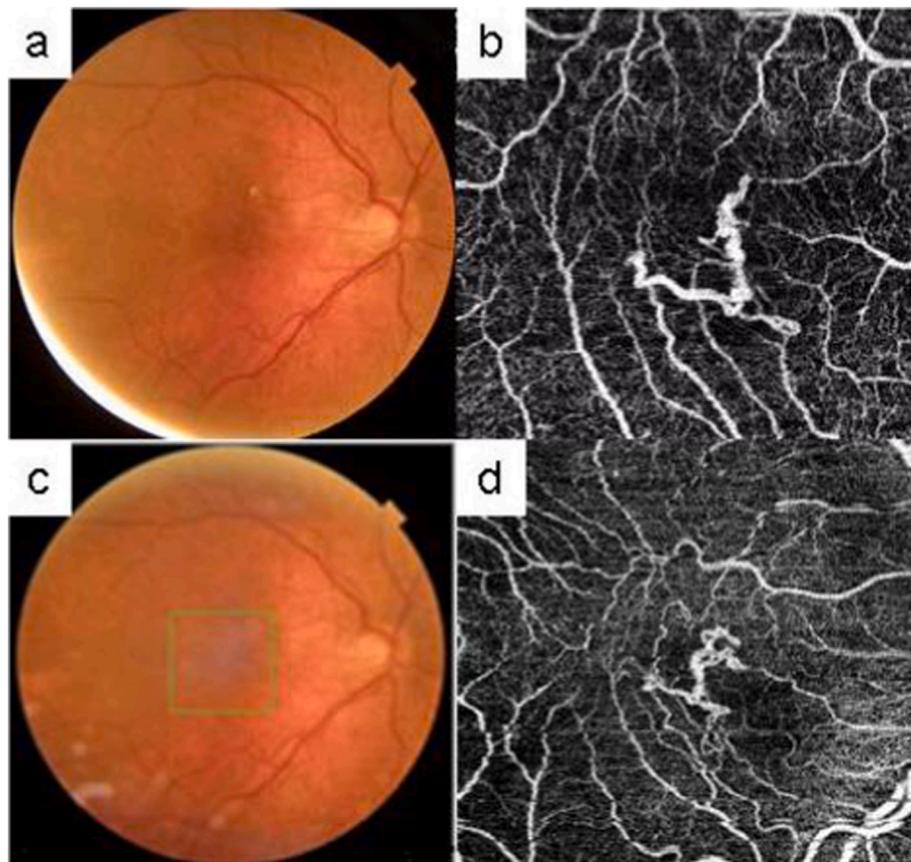
Fig. 4. a. Fundus photo of the right eye one month after the third intravitreal injection, (b.) OCT-Angiography of the lesion one month after the third intravitreal injection showing a vascular remnant configuration.

c.and d. One and a half month after the fourth injection the membrane appears inactive and stable with one of its branches disappearing.

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