## SHORT COMMUNICATION

#### Case report

# Subfoveal choroidal neovascularization in a patient with pre-existing pseudomacular hole

L. BORGIA, F. BADALÀ

Istituto di Medicina Oftalmica, Genova - Italy

PURPOSE. To report concomitant use of fluorescein angiography (FA) and optical coherence tomography (OCT) in a case of pre-existing pseudomacular hole (PMH) and macular choroidal neovascularization (CNV) treated with photodynamic therapy (PDT). METHODS. Case report.

RESULTS. A 63-year-old man who received laser treatment for branch retinal vein occlusion in 1993 developed an asymptomatic macular epiretinal membrane 2 years later. In 2001, the patient complained of metamorphopsia. Biomicroscopy showed a PMH, confirmed by OCT. One year later, metamorphopsia suddenly increased and visual acuity dropped from 20/20 to 20/70. FA and OCT revealed subfoveal occult CNV. PDT was successfully performed. FA and OCT were essential diagnostic tools during the follow-up.

CONCLUSIONS. The case presented stresses the importance of different tools for accurate diagnosis and proper follow-up of macular diseases. (Eur J Ophthalmol 2003; 13: 718-21)

KEY WORDS. Pseudomacular hole, Choroidal neovascularization, Fluorescein angiography, Optical coherence tomography, Photodynamic therapy

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### INTRODUCTION

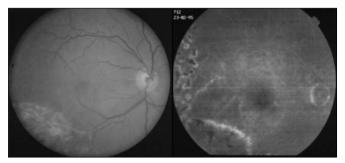
Epiretinal membrane (ERM) may occasionally occur soon after retinal photocoagulation (PC), particularly when the treatment is done in the paramacular area (1).

Age-related macular degeneration (ARMD) with choroidal neovascularization (CNV) affects 5.6% of people over 65 years of age (2). Photodynamic therapy (PDT) is the recommended treatment for classic and selected occult subfoveal CNV in ARMD (3).

We report a patient with branch retinal vein occlusion (BRVO) treated with PC who developed ERM. Afterwards, he developed wet ARMD, which was successfully treated with PDT.

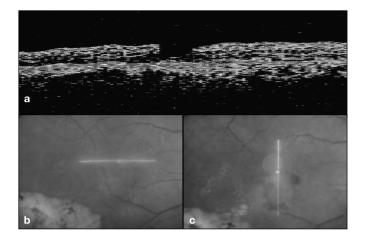
#### **Case report**

In 1993, a 63-year-old man with inferotemporal BR-VO in the right eye (RE) received laser treatment of



**Fig. 1** - February 1995: Color fundus photograph and fluorescein angiography (FA). FA shows a capillary nonperfusion area temporally and inferiorly to the macula with adjacent laser scars. No angiographic sign of macular edema is present. Note the faint, focal area of hyperfluorescence in the fovea consistent with a window defect.

the ischemic retina. The area of capillary nonperfusion involved the inferotemporal quadrant extending from the paramacular area to the extreme periphery. One year later the left eye (LE) was diagnosed with juxtafoveal CNV and underwent photocoagulation, which resulted in poor visual acuity (VA) (hand motions).



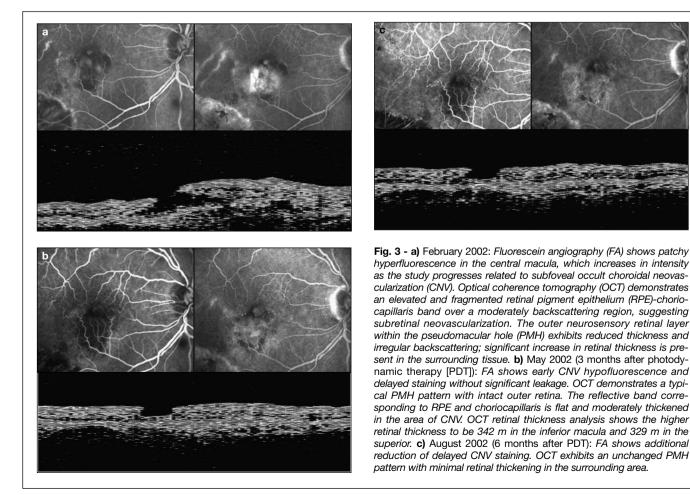
**Fig. 2 - a)** March 2001: Optical coherence tomography (OCT) image shows an abnormally steep foveal contour. In the foveal area, the presence of a reflective outer retinal layer corresponding to the retinal photoreceptors suggests the diagnosis of pseudomacular hole. The epiretinal membrane was not clearly distinct from the neurosensory retina presumably because of its tight apposition with the inner retina. b) Red-free indicating orientation of Figures **3** and **4** scansion.

The patient came to our observation in 1995. Biomicroscopically, the RE showed cellophane maculopathy. Fluorescein angiography (FA) revealed laser scars temporally to the macula and a small, mild hyperfluorescent area involving the foveal avascular zone (Fig. 1). VA was 20/20. The patient was given an Amsler-grid test and asked to check visual function periodically and return for follow-up twice a year.

In March 2001, he complained of metamorphopsia in the RE. VA was still 20/20 but biomicroscopy showed pseudomacular hole (PMH) confirmed by optical coherence tomography (OCT) (Fig. 2).

Symptoms were stable until February 2002, when the patient reported a sudden increase in metamorphopsia and VA dropped to 20/70. FA and OCT revealed CNV involving the subfoveal area (Fig. 3a). PDT was performed 2 days later, following current guidelines (3).

Three months after PDT, VA was 20/32 and a marked reduction in metamorphopsia was noted. FA showed



an important decrease in dye leakage and staining from CNV. Macular thickness, examined with OCT, was significantly reduced (Fig. 3b).

Six months after PDT, VA was 20/26. The patient still reported a slight metamorphopsia. FA revealed an additional reduction of CNV leakage. OCT showed a PMH pattern similar to the one preceding CNV occurrence (Fig. 3c).

One year after PDT, VA was unchanged (20/26) but the patient complained of a slight increase in metamorphopsia. FA did not show any signs of CNV reactivation. OCT confirmed a stable pattern of the deeper retina and demonstrated thickening of PMH surrounding retina (Fig. 4).

### DISCUSSION

The role of FA and indocyanine green (ICG) angiography in diagnosis of occult CNV has been described extensively (4, 5). The patient refused ICG angiography because he experienced ICG intolerance in 1994, when his LE was studied for ARMD. OCT, a recently introduced technique, improved our ability to detect vitreoretinal interface abnormalities and define intra- and subretinal pathology (6-8).

PDT, originally proposed for classic CNV, has also been used for occult neovascular membranes (9). The

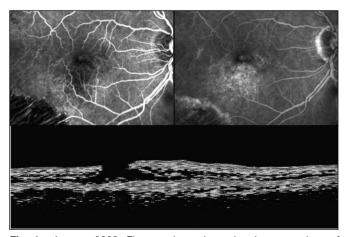
patient described is the first documented case of concurrent PMH and wet ARMD treated with PDT.

This present case stresses the importance of different tools for accurate diagnosis and proper followup. The concomitant use of FA and OCT gave us the opportunity to document occurrence of CNV on a preexisting pattern of PMH.

Following PDT, FA showed signs of progressive CNV regression; OCT delineated a flat retinal pigment epithelium and supported the angiographic features. Furthermore, later in the follow-up, metamorphopsia increase was not related to angiographic evidence of CNV reactivation. At the same time, OCT demonstrated thickening of PMH surrounding retina.

The clinical course of our patient raises the question whether PDT could result in worsening of pre-existing vitreoretinal interface pathology.

Reprint requests to: MD, Luigi Borgia is.pre. Istituto di Medicina Oftalmica Via Antiochia, 29 rosso 16129 Genova, Italy I.borgia@wind.it.net



**Fig. 4** - January 2003: Fluorescein angiography shows no signs of choroidal neovascularization reactivation. The foveal hyperfluorescence (consistent with a window defect) is enlarged. Optical coherence tomography demonstrates thickening of pseudomacular hole surrounding retina.

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