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SHORT COMMUNICATION

Spontaneous closure of a macular hole caused by a ruptured retinal arterial macroaneurysm

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PURPOSE. *To report a spontaneous closure of a macular hole (MH) that was caused by a ruptured retinal arterial macroaneurysm (RAM).*
METHODS. *Observational case report. Clinical examinations and optical coherence tomographic (OCT) evaluations of the retina of a 73-year-old woman who developed a MH secondary to a ruptured RAM.*
RESULTS. *The first sign of a closure of the MH was the appearance of tissue bridging the MH in the OCT images. Later, OCT images showed a hyperreflective tissue, probably glial cells, that connected the bridging tissue to the RPE. Seven months after the first examination, the hyperreflective tissue was smaller and the shape of the foveal pit had recovered.*
CONCLUSIONS. *A spontaneous closure of a MH caused by a ruptured RAM can occur and surgical intervention was not necessary. The tissue bridging over the MH and the hyperreflective tissue connecting the bridging tissue to the RPE most likely were involved in the spontaneous MH closure. (Eur J Ophthalmol 2008; 18: 462-5)*

KEY WORDS. *Macular hole, Optical coherence tomography, Ruptured retinal arterial macroaneurysm, Spontaneous closure of secondary macular hole*

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INTRODUCTION

We are unaware of any reports describing a spontaneous closure of a macular hole (MH) caused by a ruptured retinal arterial macroaneurysm (RAM). We had the opportunity to follow the process of spontaneous MH closure by optical coherence tomographic (OCT) examinations.

Case report

A 73-year-old woman complained of a sudden visual decrease in her right eye. Her visual acuity (VA) was 0.02, ophthalmoscopy showed subretinal and subhyaloid hemorrhages around the posterior pole, and the macula could not be examined.
Two weeks later, the subhyaloidal hemorrhage had settled into the inferior vitreous following a posterior vitreous detachment (PVD), and a MH was detected. A ruptured RAM was seen superior to the macula (Fig. 1A). Fluorescein angiography (FA; Fig. 1B) and indocyanine green angiography (Fig.1C) revealed hyperfluorescence and arterial telangiectatic changes.
OCT showed that the MH that was surrounded by a fluid cuff with perifoveal cysts (Fig. 2A). The patient refused any treatment.
Ten weeks later, the MH appeared to be closed (Fig. 1D). Evidence of epiretinal membranes and ischemic changes were not present in the macular area (Fig. 1D) in the FA (Fig. 1E) image. OCT demonstrated a foveal detachment and tissue bridging the MH (Fig. 2B).
One month later, the degree of retinal detachment had decreased. OCT demonstrated hyperreflective tissue connecting the bridging tissue across the MH to the retinal pigment epithelium (RPE; Fig. 2C). Her VA had recovered to 0.15. After another month, the foveal detachment was minimal, and OCT showed the hyperreflective tissue was smaller (Fig. 2D).

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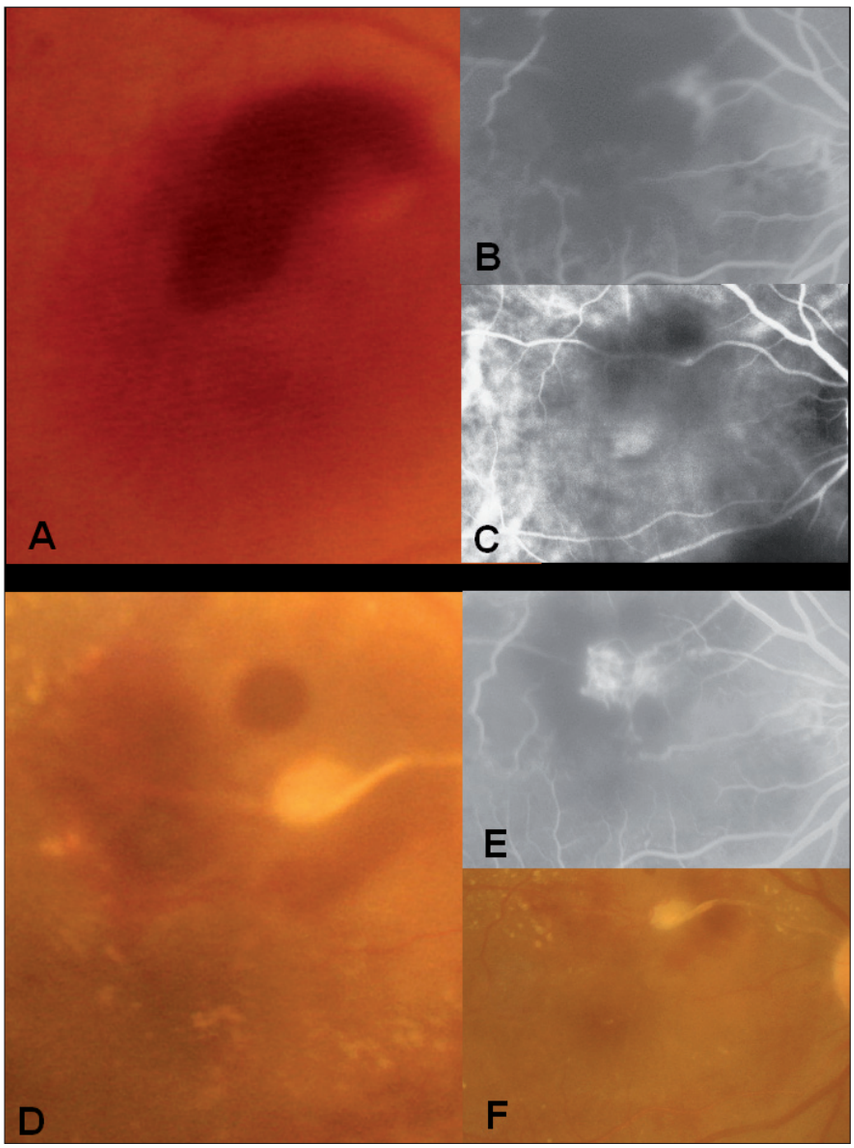

Fig. 1 - *Fundus photographs, fluorescein angiographic images (FA), and indocyanine green angiographic images (ICGA).* **(A)** *The ruptured retinal arterial aneurysm (RAM) causing the preretinal and subretinal hemorrhages is seen superior to a macular hole (MH) and within the nasal-superior arcade.* **(B)** *FA shows weak hyperfluorescence because of a blockage by the thick hemorrhage corresponding to the ruptured RAM. The timing of the angiography sequence is 46 sec (see FA features in A).* **(C)** *ICGA reveals hyperfluorescence and telangiectatic arterial change. The timing of the angiography sequence is 44 sec (see ICGA features in A).* **(D)** *The subretinal hemorrhage is thinner and has enlarged over the posterior pole. The MH appears to be closed ophthalmoscopically.* **(E)** *The collateral vessels can be seen around the ruptured RAM and peripheral arteries. The timing of the angiography sequence is 37 sec (see FA features in D).* **(F)** *The foveal detachment is not present.*

Two months later, about 7 months after the first examination, the foveal detachment was not present (Fig. 1F), and the configuration of the fovea was normal (Fig. 2E). The VA had improved to 0.2.

DISCUSSION

Several mechanisms can be proposed to have caused the MHs following a ruptured RAM. The first mechanism is a vitreal traction on the fovea from a contraction of the vitreous cortex which, in our case, could have been induced by the vitreous hemorrhage (1). A second mechanism is mechanical and metabolic damage to macula caused by the fibrin and iron from the subretinal hemorrhage which can occur within 24 hours after the hemorrhage (2). A third mechanism is a rupturing of intraretinal cysts following the ruptured RAM as has been proposed following branch retinal vein occlusion (3). A fourth mechanism is the increased hydrodynamic pressure in the subretinal space caused by the submacular hemorrhage (1, 4). We suggest all mechanisms probably played a role in our case.

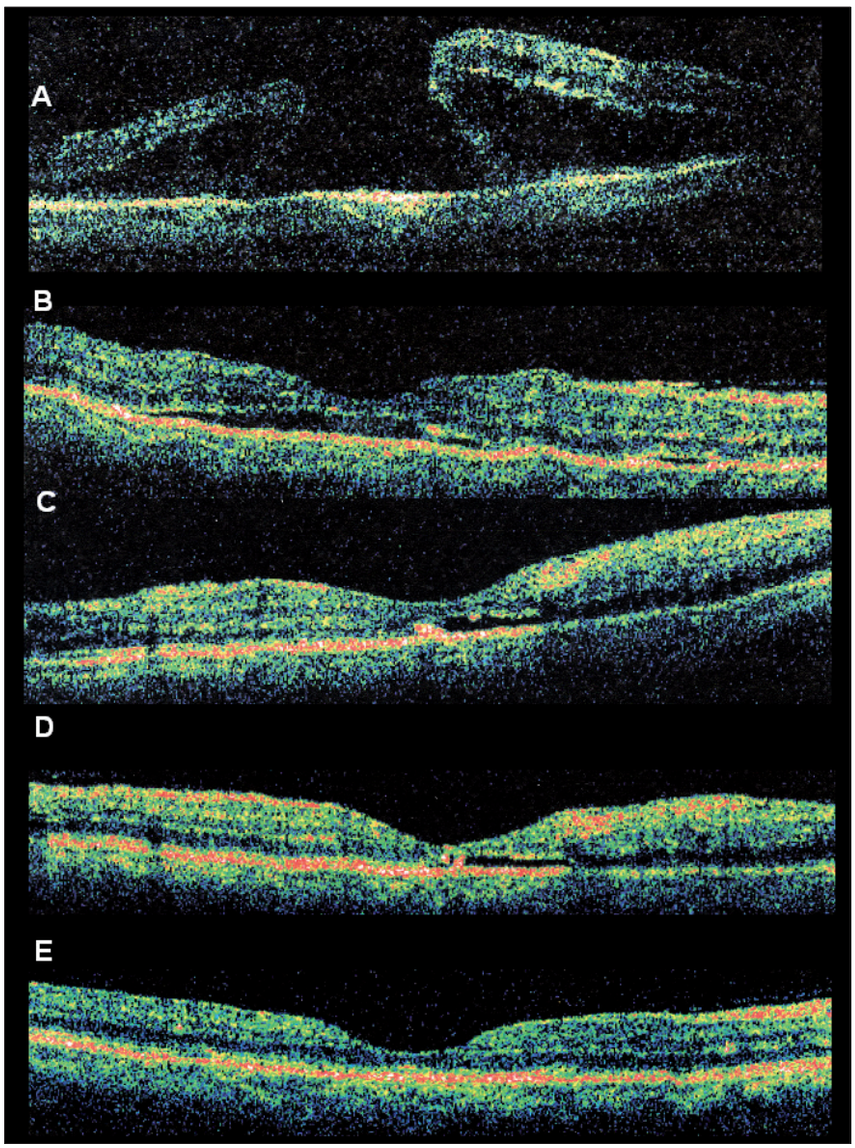

Fig. 2 - *Optical coherence tomograms (OCT, 5 mm horizontal scans).* **(A)** *OCT shows a macular hole (MH) surrounded by a fluid cuff with perifoveal cysts (see OCT images in Fig. 1A).* **(B)** *OCT showing tissue bridging the hole with subretinal fluid. Foveal cysts are not present (see tomographic image in Fig. 1D).* **(C)** *OCT demonstrating a band of hyperreflective tissue connecting the bridging tissue across the MH to the foveal retinal pigment epithelium (RPE).* **(D)** *OCT showing that the hyperreflective tissue is present but smaller at the site of the macula.* **(E)** *OCT showing that the bridging tissue attached to the RPE, and a recovery of the shape of the foveal pit. The hyperreflective tissue is not detectable at the site of the macula (see tomographic image in Fig. 1F).*

The time when a MH forms following a ruptured RAM varies (1, 4). In our case, the MH was detected 14 days after the rupture, but we suggest that the MH developed at the time or early after the rupture.

Two hypotheses may be proposed for closures of a secondary MH. First, the release from vitreous traction following a PVD, and second, a proliferation of glial or RPE-derived cells as has been suggested for idiopathic (5) and traumatic MHs (6). In our case, OCT showed tissue bridging the hole as the first step in the closure (3). The second step was the development of hyperreflective tissue, possibly glial cells, that connected the tissue bridging the MH to the RPE. These findings resemble those reported by Parmar et al (6). Then, a decrease of the hyperreflective tissue would lead to the recovery of the foveal pit.

Conventional MH surgery can lead to morphologic success in a high percentage of eyes, although the visual outcome after the MH surgery is mixed (4). It is difficult to decide when to do MH surgery, because the longer the delay, the more likely it is that the photoreceptor will be damaged. In addition, the macula damage (2) caused by the ruptured RAM may also affect the postoperative VA.

In conclusion, although we do not definitely know whether MH surgery would have altered the VA, our case demonstrates that a secondary MH following ruptured RAM can close spontaneously.

Proprietary interest: None.

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