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SHORT COMMUNICATION

Optical coherence tomography and fundus microperimetry imaging of spontaneous closure of traumatic macular hole: A case report

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PURPOSE. *To report a case of spontaneous closure of traumatic macular hole in a young patient followed using optical coherence tomography (OCT) and fundus microperimetry.*
METHODS/RESULTS. *In the right eye of a 10-year-old child, a traumatic macular hole was observed to spontaneously resolve 18 weeks after blunt trauma. Initially, visual acuity in the right eye was 20/200 and OCT examination showed a 200µm-diameter full-thickness macular hole with perifoveal edema. Fundus microperimetry examination showed an evident decrease in retinal sensitivity within the macular hole and in the upper macular region where an area of commotio retinae was clearly visible. During follow-up OCT demonstrated the appearance of a band of tissue linking the inferior edge of the hole to the foveal retinal pigment epithelium and at the bottom of the hole the presence of hyperreflective (glial) material. Eighteen weeks after trauma right eye visual acuity had improved to 20/25, OCT examination showed a restored foveal depression, and fundus microperimetry demonstrated an increase in foveal sensitivity.*
CONCLUSIONS. *Both OCT and fundus microperimetry were useful tools for following the favorable course in a case of spontaneous closure of traumatic macular hole in a young patient. During follow-up OCT examinations were able to demonstrate the course of macular hole closure. (Eur J Ophthalmol 2005; 15: 165-9)*

KEY WORDS. *Optical coherence tomography, Commotio retinae, Microperimetry, Traumatic macular hole*

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INTRODUCTION

Traumatic macular hole (TMH) is a well-recognized complication of ocular trauma. Spontaneous macular hole closure after blunt trauma is not uncommon. Many authors (1-6) described cases of spontaneous closure of TMH using optical coherence tomography (OCT). Both Parmar and coworkers (2) and Menchini and coworkers (6) presented a case of TMH with spontaneous closure imaged with OCT and confocal laser scanning ophthalmoscopy (CSLO). We report a young patient in whom a TMH spontaneously closed and who was followed using OCT (STRATUS OCT, Carl Zeiss Meditec, Dublin, CA) and recently available fundus microperimetry (MP-1, Nidek Technologies, Vigonza PD, Italy). To our knowledge, this has not been previously described.

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Case report

The right eye of a 10-year-old boy was hit by a soccer ball on October 22, 2002. He immediately noticed a loss of central visual acuity. Two days later, on examination, best-corrected visual acuity (BCVA) was 20/200 in the right eye and 20/20 in the left eye. Biomicroscopy disclosed a full-thickness macular hole with associated commotio retinae and absence of posterior vitreous detachment in his right eye. OCT and MP1 were performed. OCT showed a small full-thickness macular hole (diameter 200 µm) with minimum edema of the borders, without epiretinal membrane or posterior vitreous detachment. In addition, a slight, flat detachment of the neuroretina was evident in the inferior perifoveal region (Fig. 1A). MP1 retinography disclosed macular hole and commotio retinae as a "salt and pepper" degeneration area with pigmentary leakage in the superotemporal side of the posterior retina. MP-1 fundus microperimetry showed an absolute scotoma within the hole and a deep reduction of retinal sensitivity at its borders and at the site of the salt and pepper area; pattern of fixation was classified as predominantly eccentric and relatively unstable (Fig. 2A). One week later (October 31, 2002), BCVA remained unchanged, and OCT examination revealed detachment of TMH margins (especially the inferior one) and consequent enlargement of basal diameter of the hole (700 µm). A thin reflective band links the edges of the hole (Fig. 1B).

On November 7, 2002, BCVA improved to 20/50. OCT examination revealed a minimum increase of inferior border detachment with the appearance of a band of tissue linking the inferior edge of the hole to the foveal retinal pigment epithelium (Fig. 1C).

Twelve days later (November 19, 2003), BCVA remained unchanged (20/50), but OCT examination showed the appearance of cystic changes at the edge of the hole with minimum detachment of the outer wall of the cysts and reduction of the outer diameter of the hole (450 µm); then, at the bottom of the hole the presence of a hyperreflective signal likely corresponding to glial material was evident. The reflective band linking the hole's edges is more evident (Fig. 1D).

On December 10, 2002, BCVA improved to 20/40. OCT examination showed a further reduction of the outer diameter of the hole (350 µm) and the appearance of thin bands linking the edges to the bottom of

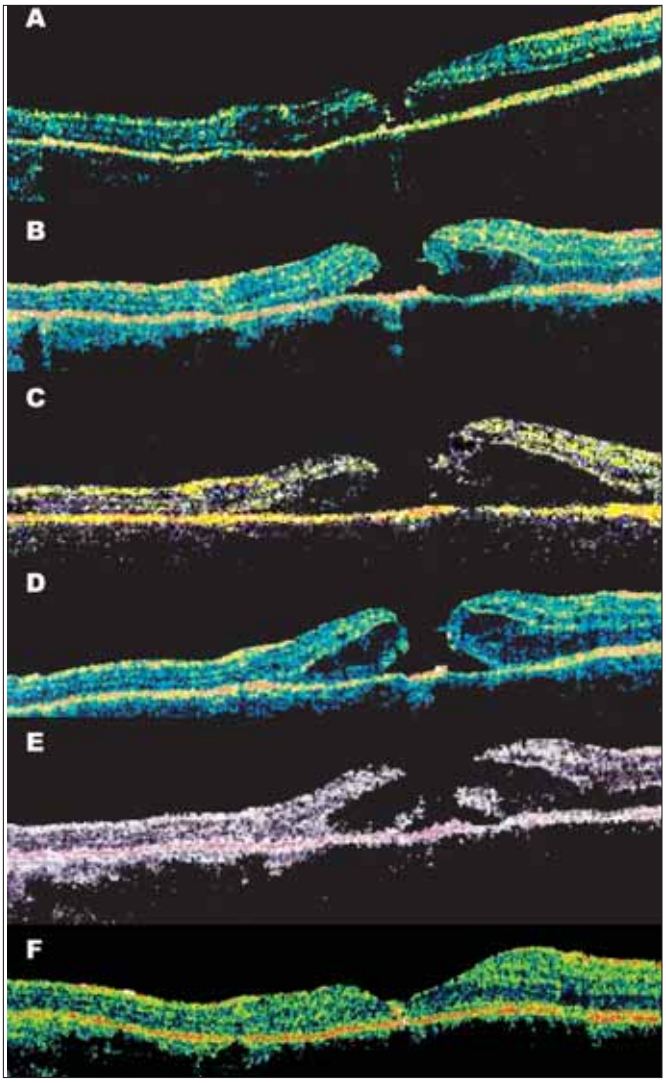

Fig. 1 - *Sequence of optical coherence tomography (OCT) images showing the progressive closure of traumatic macular hole (TMH).* **(A)** *At the time of the injury, OCT image shows a small full-thickness macular hole (diameter 200 mm) with minimum edema of the borders, without epiretinal membrane or posterior vitreous detachment. At the right side of the scan a slight, flat detachment of the neuroretina is evident.* **(B)** *Detachment of TMH margins (especially the inferior one) and consequent enlargement of basal diameter of the hole (700 mm). A thin reflective band links the edges of the hole.* **(C)** *Minimum increase of inferior border detachment with the appearance of a band of tissue linking the inferior edge of the hole to the foveal retinal pigment epithelium.* **(D)** *Appearance of cystic changes at the edge of the hole with minimum detachment of the outer wall of the cysts and reduction of the outer diameter of the hole (450 mm); at the bottom of the hole the presence of hyperreflective signal likely corresponding to glial material is evident. The reflective band linking the edges of the hole is more evident.* **(E)** *Further reduction of the outer diameter of the hole (350 mm). Thin reflective bands link the edges to the bottom of the hole.* **(F)** *OCT image discloses complete closure of the macular hole and restoration of normal foveal configuration with a U-shaped configuration. A hyperreflective retinal signal is detectable at the site of the fovea.*

the hole (Fig. 1E). On March 6, 2003, BCVA improved to 20/25 and OCT examination disclosed complete closure of the macular hole and the restoration of normal foveal configuration with a U-shaped configuration. A hyperreflective retinal signal was detectable at the site of the fovea (Fig. 1F). At MP1 retinography the salt and pepper area in the superior temporal region was still evident, but the macular hole was no longer detectable; MP1 fundus microperimetry showed improvement of the foveal threshold of sensitivity and a predominantly central, stable pattern of fixation (Fig. 2B).

DISCUSSION

The exact mechanism of formation of TMH is uncertain. Evidence from animal studies and biomicroscopic and surgical observations of patients with a TMH strongly suggest that vitreous traction is important in the pathogenesis of TMH. Experimental observation shows that sudden compression of the globe produces an immediate stress on the retina at points of vitreous attachment (7). Johnson et al (8) theorized that the flattening of the posterior pole associated with equatorial expansion and subsequent posterior displacement of the posterior pole of the eye is the mechanism of most cases of TMH formation. In fact, it seems likely that with this trampoline-like movement of the posterior pole, sudden tractional forces, especially tangential ones, exerted on the anatomically thin fovea result in an immediate formation of a macular hole in most cases.

Yamashita et al (9) hypothesized two distinct mechanisms of TMH formation, depending on whether the posterior hyaloid is attached or detached: one type that causes immediate visual loss due to primary dehiscence of the fovea, and the other type that leads to delayed visual loss due to dehiscence of the fovea secondary to persistent vitreofoveal adhesion.

The area of commotio retinae does not seem to influence the pathogenesis of the TMH. Commotio retinae can cause the vacuolization of the inner segments and the loss of the outer segments in the layer of photoreceptors, changes (nuclear pyknosis) in the outer nuclear layer (10-13) and in the retinal pigment epithelium (RPE) (11-13). Commotio retinae can be followed by RPE atrophy and pigment migration in the neuroretina (14, 15). In extreme cases, a pseudoretinitis pigmentosa appearance occurs (8). Substantial photoreceptor damage is typical in this setting. If significant disruption of the RPE is present near or within the center of the macula, this potentially has significant importance for vision recovery after spontaneous or surgically induced TMH closure.

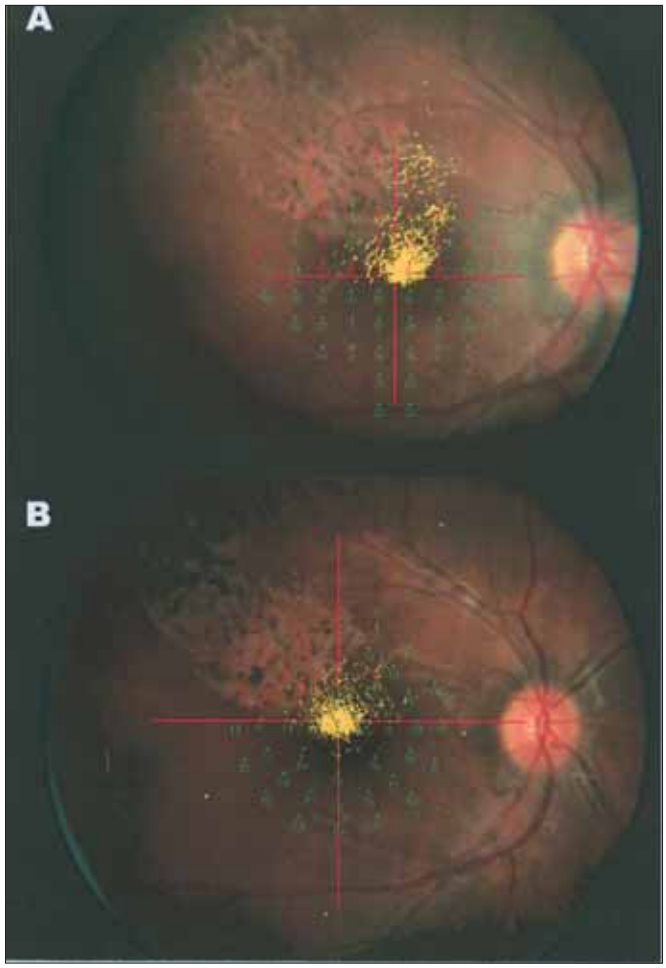

Fig. 2 - (A) *At the time of injury, MP1 fundus microperimetry demonstrates an absolute scotoma within the traumatic macular hole and a deep reduction of retinal sensitivity at its borders. Loss of retinal sensitivity is evident at the site of the "salt and pepper" area; pattern of fixation is classified as predominantly eccentric and relatively unstable.* **(B)** *Eighteen weeks later, MP1 fundus microperimetry shows improvement of the foveal threshold of sensitivity and a predominantly central, stable pattern of fixation. Loss of retinal sensibility is still evident at the site of the salt and pepper area.*

Spontaneous closure of TMH is not uncommon (1-6). Many authors tried to explain the mechanism of traumatic macular hole spontaneous closure (1-6). Using OCT, Mitamura et al (3) and Menchini et al (6) could document bridging of neuroretinal tissue over an optically empty subretinal area as the first step of the

process of spontaneous TMH in three cases. Menchini et al (6) also observed a duplication of the hyperreflective band corresponding with the RPE–Bruch membrane–choriocapillary complex and interpreted this sign as proliferation of glial cells or as a regrowth of RPE-derived cells on the outer surface of the bridging neuroretinal tissue.

In our case, 7 days after the disclosure of TMH, OCT examination showed an initial worsening of findings: detachment of TMH margins and enlargement of basal diameter of the hole. This may be linked to residual tractional forces on the hole from the vitreous. In addition, an extremely thin band of tissue linking the edges of the hole is detectable.

This finding is not similar to the thick bridging tissue visualized by Mitamura et al (3) and Menchini et al (6). Seven days later, OCT documents the appearance of a band of tissue linking the inferior edge of the hole to the foveal RPE.

This finding is very similar to that described by Parmar et al (2) in their case report of a TMH with spontaneous closure and it is likely to be linked to glial proliferation. Twelve days later, OCT examination showed the presence of a hyperreflective signal at the bottom of the hole. This can be interpreted as proliferation of glial cells or RPE-derived cells which fill the hole bottom (5). In addition, the band bridging the hole's edges is more evident and the outer diameter of the hole is evidently smaller. This may help to interpret the bridging band as a contractile tissue. The last phase before the hole closure documented by OCT is characterized by the appearance of further bands of tissue linking the edges to the bottom of the hole. The contractile nature of these bands can be hypothesized.

Our patient was also followed by means of MP1 fundus microperimetry. At the first examination MP1 showed an absolute scotoma within the macular hole and a deep reduction of retinal sensibility at its borders and at the site of the salt and pepper area; pattern of fixation was classified as predominantly eccentric and relatively unstable. At the end of follow-up MP1 examination showed foveal recovery of sensitivity, confirmed a deep reduction of retinal sensitivity at the site of the salt and pepper area, while pattern of fixation was predominantly central and stable.

Some interesting findings emerged from our case report followed with OCT and fundus microperimetry. OCT examination showed macular hole at the time of injury, demonstrating that macular hole was caused by direct contrecoup injury of the eyeball (5).

One week after the trauma OCT showed elevation of the sensory retina surrounding the macular hole's edge, so one may hypothesize that vitreous body changes can continue to exert their effect on the macula for a few days after the trauma. During follow-up, using OCT we could observe the natural progression to closure of the traumatic macular hole.

We could not observe the appearance of the classical bridging tissue visualized by Mitamura et al (3) and Menchini et al (6). In our patient the mechanism of closure was very similar to that described by Parmar et al (2) in their case report.

Bands of tissue bridged the edges of the hole to the foveal RPE and the hole bottom was filled by hyperreflective tissue. These findings support the hypothesis that one of the possible mechanisms of TMH closure may be linked to glial cells or RPE cells proliferation from each bank of the hole's edge in order to fill the hole bottom and close the hole by means of contractile tissue. In addition, our patient was very young and the glial proliferation is likely to be vigorous in younger patients (2, 5). Finally, when visual acuity improved due to TMH closure MP1 fundus microperimetry well documented the recovery of foveal sensitivity and the improved stability and location of fixation.

The good improvement in visual acuity observed in this case may be explained by the superotemporal dislocation of the area of retinal pigment damage. In fact, MP1 was able to show an irreversible functional loss in the salt and pepper area.

In conclusion, although surgery has been advocated in cases of TMH, our case report supports the opinion that observation in conjunction with OCT and MP1 examination is wise in following TMH, because of the possibility that the macular hole may close spontaneously within a few months. Together with the macular hole closure the position of the area of RPE atrophy may have a strong influence on visual recovery.

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REFERENCES

1. Kusaka S, Fujikado T, Ikeda T, Tano Y. Spontaneous disappearance of traumatic macular holes in young patients. Am J Ophtalmol 1997; 123: 837-9.
2. Parmar DN, Stanga PE, Reck AC, Vingerling J, Sullivan P. Imaging of a traumatic macular hole with spontaneous closure. Retina 1999; 19: 470-2.
3. Mitamura Y, Saito W, Ishida W, Yamamoto S, Takeuchi S. Spontaneous closure of traumatic macular hole. Retina 2001; 21: 385-92.
4. Yamashita T, Uemara A, Uchino E, Doi N, Ohba N. Spontaneous closure of traumatic macular hole. Am J Ophtalmol 2002; 133: 230-5.
5. Yamada H, Sakai A, Yamada E, Nishimura T, Matsumura M. Spontaneous closure of traumatic macular hole. Am J Ophtalmol 2002; 134: 340-7.
6. Menchini U, Virgili G, Giacomelli G, Cappelli S, Giansanti F. Mechanism of spontaneous closure of traumatic macular hole: OCT. Retina 2003; 23: 104-6.
7. Delori F, Pomerantzeff O, Cox MS. Deformation of the globe under high-speed impact: its relation to contusion injuries. Invest Ophthalmol 1969; 8: 290-301.
8. Johnson RN, McDonald HR, Lewis H, et al. Traumatic macular hole. Observations, pathogenesis, and results of vitrectomy surgery. Ophthalmology 2001; 108: 853-7.
9. Yamashita T, Uemara A, Uchino E, Doi N, Ohba N. Spontaneous closure of traumatic macular hole. Am J Ophtalmol 2002; 133: 230-5.
10. Sipperley JO, Quigley HA, Gass DM. Traumatic retinopathy in primates. The explanation of commotio retinae. Arch Ophthalmol 1978; 96: 2267-73.
11. Liem AT, Keunen JE, Van Norren D. Reversible cone photoreceptor injury in commotio retinae of the macula. Retina 1995; 15: 58-61.
12. Meyer CH, Rodrigues EB, Mennel S. Acute commotio retinae determined by cross-sectional optical coherence tomography. Eur J Ophthalmol 2003; 13: 816-8.
13. Mansour AM, Green WR, Hogge C. Histopathology of commotio retinae. Retina 1992; 12: 24-8.
14. Friberg TR. Traumatic retinal pigment epithelial edema. Am J Ophthalmol 1979; 88: 18-21.
15. Bastek JV, Foos RY, Heckenliveley J. Traumatic pigmentary retinopathy. Am J Ophthalmol 1981; 92: 621-4.