CASE REPORT

Macular hole formation after toxoplasmic retinochoroiditis

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SUMMARY

A 56-year-old female patient presented with toxoplasmic retinochoroiditis (TR) in the right eye. Optical coherence tomography revealed a full-thickness macular hole (MH) in the affected eye. Fluorescence angiography and indocyanine green-angiography disclosed focal choroidal ischaemia in the area of inflammation. Heidelberg retinal flowmetry confirmed the significant hypoperfusion in this area. Proper medication was administered. Ophthalmological examination 4 weeks later revealed an improvement of the clinical findings without visual restoration. This case supports the clinical hypothesis that retinochoroidal ischaemia due to TR may induce the development of MH, indicating that patients with TR may have a certain risk for MH formation.

BACKGROUND

Toxoplasmosis is an infectious disease caused by the parasite *Toxoplasma gondii*. A typical manifestation of ocular toxoplasmosis consists of focal necrotising retinochoroiditis accompanied by vitreous inflammatory reaction, frequently associated with adjacent retinochoroidal scars.¹

In our report we present for the first time a patient with ocular toxoplasmosis who developed macular hole (MH) in the affected eye and describe the potential aetiopathological association between toxoplasmic retinochoroiditis (TR) and MH formation.

CASE PRESENTATION

A 56-year-old female patient was referred to our department with photophobia and floaters in the right eye. She had experienced one episode of TR in the same eye 1 year ago, treated successfully in another hospital. The patient experienced a gradual decrease of visual acuity in the right eye for the last 3 months, but did not ask for medical advice.

Best corrected visual acuity (BCVA) was 2/20 in the right eye (OD, oculus dexter) and 20/20 in the left eye (OS, oculus sinister). A slit-lamp examination revealed signs of inflammation in the anterior segment; anterior chamber cells 2+, anterior chamber flare 2+ and vitreous humour cells 3+. Intraocular pressure was 14 mm Hg in both eyes. Fundus examination revealed a parapapillary retinochoroidal lesion with a size of <1 disc area as well as MH grade IV (Gass classification) (figure 1). There were no signs of proliferative vitreoretinopathy, but posterior vitreous was detached. Optical coherence tomography (OCT) confirmed the presence of a full-thickness MH (figure 1). Fluorescence angiography and indocyanine green (ICG) angiography disclosed

focal ischaemia of the choroid in the area of inflammation (figure 2). Consequently, we measured the retinal perfusion in the area of inflammation by the Heidelberg retina flowmeter using a technique described elsewhere.² Briefly, we evaluated blood flow in 10 different horizontal levels above as well as below the area of interest. We were able to document a significantly decreased regional mean blood flow in this area (137.6 ± 29.5 a.u.) compared to the same location OS (452.76 ± 42.4 a.u.) (Student t test, $p < 0.001$).

INVESTIGATIONS

Serological screening was positive for toxoplasmosis (positive IgM and IgG antibodies). Blood sample PCR-test confirmed the diagnosis of toxoplasmosis.

TREATMENT

Oral medication (pyrimethamine 50-mg per day for 4 weeks, sulfadiazine 2-g per day for 4 weeks, folic acid 7.5-mg per week for 4 weeks, methylprednisolone 80 mg tapered within 4 weeks) as well as topical therapy (prednisolone eye drops 3 times per day for 4 weeks) were administered.

OUTCOME AND FOLLOW-UP

Ophthalmological examination 4 weeks later revealed an improvement of the clinical findings without visual restoration (BCVA OD 2/20).

DISCUSSION

MH is a clinical entity that usually occurs idiopathically in elderly individuals. Although many other causes of MH formation, such as trauma, proliferative diabetic retinopathy, epiretinal gliosis, etc, are considered to represent important aetiological factors, the major pathophysiological mechanism implicated in the pathogenesis of MH remains the genesis of vitreoretinal tractions.³

Fundus examination in case of our patient revealed no prominent vitreoretinal tractions, which represent the major predisposing factor for pathogenesis of MH. Nonetheless, the presence of operculum above the MH may indicate that vitreoretinal traction due to inflammatory contraction of the posterior hyaloid face could have possibly contributed, at least to some extent, to the formation of MH.

On the other hand, it is interesting that although the patient had experienced an episode of TR in the same eye 1 year ago, no visible pigmented scar was documented on the fundus. Moreover, both IgG and IgM antibodies were positive for toxoplasmosis and PCR confirmed the diagnosis. Based on

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Figure 1 *Fundus image*: the black arrow shows the macular hole, while the yellow arrows outline the toxoplasmic retinochoroidal (TR) lesion (A). *Optical coherence tomography image*: full-thickness macular hole (grade IV) (B). *Infrared image*: the red arrow points to the macular hole; the large yellow arrows outline the TR lesion and the small yellow arrows point to satellite retinochoroidal lesions (C). *Autofluorescence image*: the red arrow points to the macular hole; the large yellow arrows outline the RT lesion and the small yellow arrows point to satellite retinochoroidal lesions (D).

Figure 2 *Indocyanine green (ICG) angiography (0.09 s after dye injection)*: the large yellow arrows outline the area of toxoplasmic lesion and choroidal 'non-perfusion', and the small yellow arrow shows choroidal vessels projecting to the macular hole (A). *ICG angiography (5.28 s after dye injection)*: the red arrow shows the macular hole; the large yellow arrow outlines the area of the retinochoroidal lesion as seen on fundus image and the small yellow arrow points to small lesions along a 'Bjerrum-like' nerve fibre involvement (B). *Fluorescence angiography (0.13 s after dye injection)*: the red arrow shows the macular hole and the small yellow arrows outlines the area of retinal 'non-perfusion' (C). *Fluorescence angiography (5.30 s after dye injection)*: the small yellow arrows depict the area of the retinochoroidal lesion and the small red arrow hints to a small area of dye leakage directly adjacent to the retinochoroidal lesion (D).

these findings, one may presume that our patient represents a case of acquired toxoplasmosis.

However, this case is consistent with reports which suggest an aetiopathological association between TR and MH formation, thereby supporting the clinical hypothesis that retinochoroidal ischaemia due to TR may induce the development of MH.

Blaise *et al*⁴ were the first to present a patient with a giant MH as an atypical consequence of TR. Auer *et al*⁵ have unravelled, with the aid of ICG, a significant choroidal involvement in TR, consisting predominantly of marked hypofluorescence extending beyond the visible retinochoroidal lesions. Khairallah *et al*⁶ have recently documented that TR is associated with acute choroidal ischaemia.

Moreover, it is well established that retinal ischaemia may lead to retinal hole formation.⁷ MH formation has also been reported due to underlying foveolar ischaemia.⁸

In conclusion, our case report supports the clinical hypothesis that patients with TR may have a certain risk for MH formation and, therefore, it is important that ophthalmologists are aware of this unusual complication.

Competing interests None.

Patient consent Obtained.

Provenance and peer review Not commissioned; externally peer reviewed.

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Learning points

- ▶ Retinal ischaemia may lead to retinal hole formation.
- ▶ Toxoplasmic retinochoroiditis (TR) may have a certain risk for macular hole formation as a result of the induced retinal ischaemia.
- ▶ Ophthalmologists must be aware of this unusual complication of TR.

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