Case Report

Full-thickness macular hole in a patient with diabetic cystoid macular oedema treated by intravitreal triamcinolone injections

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ABSTRACT.

Purpose: **Full-thickness macular hole associated with diabetic macular oedema is a rare feature and its pathogenesis remains incompletely elucidated. We report the occurrence of a full-thickness macular hole, documented with optical coherence tomography (OCT), in a patient with diabetic cystoid macular oedema treated by intravitreal triamcinolone injections.**

Case Report: **A 48-year-old woman with refractory diabetic cystoid macular oedema underwent successive intravitreal triamcinolone injections, which were followed by a progressive thinning of the neurosensory retina at the fovea, and then by a full-thickness macular hole, associated with a perifoveal posterior hyaloid detachment, visible on OCT. During pars plana vitrectomy, a thin epiretinal macular membrane was diagnosed and removed.**

Discussion: **Pathogenesis of the present full-thickness macular hole associated with diabetic macular oedema is different from that of idiopathic macular holes because anteroposterior vitreous tractions were not involved in its formation. Recurrent intravitreal triamcinolone injections may have had an indirect role in the development of the macular hole, by favouring the rupture of distended Muller cells and intraretinal pseudocysts.**

Key words: diabetic macular edema – full-thickness macular hole – intravitreal triamcinolone acetonide injection – retinal thinning

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Introduction

Full-thickness macular holes have been reported in diabetic patients with various degrees of diabetic retinopathy (Brazitikos & Stangos 1999; Ghoraba 2002; Yoon & Seo 2003). When associated with no or non-proliferative diabetic retinopathy without macular oedema, their pathogenesis is similar to that of idiopathic macular holes, resulting from anteroposterior tractions by the vitreous on the fovea (Brazitikos & Stangos 1999). In patients with proliferative diabetic retinopathy, macular holes are associated with tractional retinal detachment or premacular haemorrhage, and are mainly the result of tangential vitreous tractions (Ghoraba 2002).

However, only a few cases of full-thickness macular holes associated with macular oedema have been described in the literature, and their pathogenesis remains incompletely elucidated (Brazitikos & Stangos 1999; Ghoraba 2002; Spaide 2005).

Using optical coherence tomography (OCT), we report the progressive thinning of the neurosensory retina at the fovea followed by the occurrence of a full-thickness macular hole in a patient with diabetic cystoid macular oedema treated by intravitreal triamcinolone injections (Jonas 2005; Sorensen et al. 2005; Sivaprasad et al. 2006; Vinten et al. 2007), and discuss its pathogenesis.

Case Report

A 48-year-old diabetic woman presented with severe non-proliferative diabetic retinopathy and macular oedema refractory to laser photocoagulation

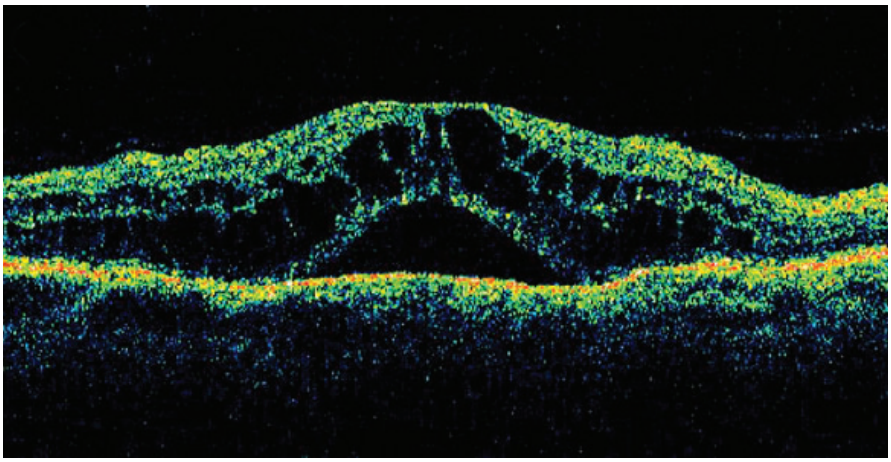

Fig. 1. Optical coherence tomography (OCT) of the right eye of a patient with a diabetic macular oedema refractory to laser photocoagulation. Before triamcinolone injection, OCT showed a cystoid macular oedema with subfoveal retinal serous detachment, and a perifoveal posterior hyaloid detachment (central macular thickness including subfoveal retinal serous detachment: 958 μm; foveal retinal thickness without subfoveal retinal serous detachment: 590 μm).

in the right eye. OCT (StratusOCT, Carl Zeiss Meditec, Dublin, California, USA) showed a cystoid macular oedema with subfoveal retinal serous detachment, and a perifoveal posterior hyaloid detachment (Massin et al. 2006) (Fig. 1). A first intravitreal injection of 4 mg (0.1 mL) triamcinolone acetonide was performed (Audren et al. 2006); this led to a decrease of central macular thickness (1000 μm diameter area) from 958 μm (including the subfoveal detachment) to 187 μm, with a concomitant improvement of visual acuity from 20/250 to 20/63 (Fig. 2). Three additional intravitreal triamcinolone injections were performed because of the recurrence of macular oedema 6 months after each previous injection. They were, respectively, followed by a decrease of central macular thickness to 174 μm after the second injection, 170 μm after the third injection and finally 151 μm after the fouth injection, with the occurrence of a full-thickness macular microhole without raised thickened edges (Fig. 3). The perifoveal posterior hyaloid detachment remained identical on OCT. Concomitant visual acuities were, respectively, 20/60, 20/60 and 20/125. As macular oedema recurred, OCT showed a full-thickness macular hole with raised edges, and the persistence of the perifoveal posterior hyaloid detachment (Fig. 4). Pars plana vitrectomy associated with gas tamponade was performed as management of the full-thickness macular hole.

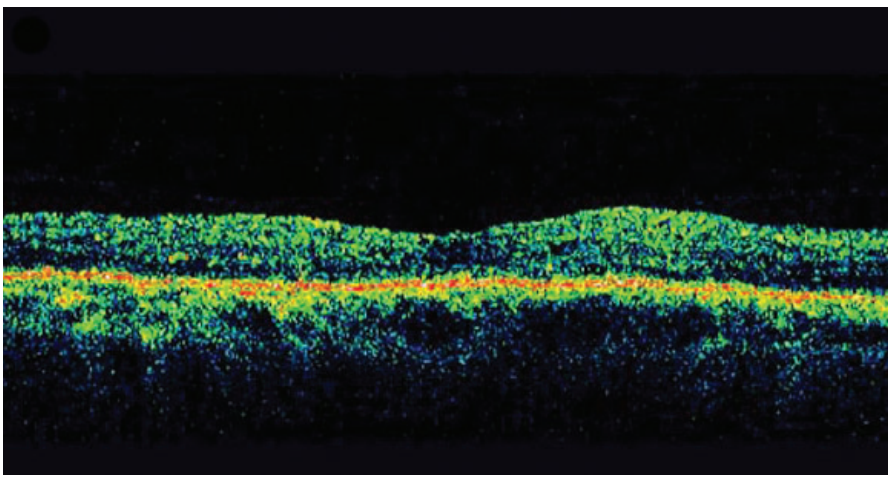

Fig. 2. Three months after the first triamcinolone injection, central macular thickness decreased to 185 μm.

During pars plana vitrectomy, the absence of posterior vitreous detachment was confirmed, and a thin epiretinal macular membrane associated with the full-thickness macular hole was diagnosed and removed. One month postoperatively, OCT demonstrated macular hole closure and recurrence of macular cystoid changes. Three months after surgery, visual acuity was 20/160.

Discussion

We report here a case of progressive thinning of the neurosensory retina at the fovea followed by the occurrence of a full-thickness macular hole, documented by OCT, in a patient with diabetic cystoid macular oedema treated by intravitreal triamcinolone injections.

It is currently accepted that the formation of idiopathic full-thickness macular holes relies on anteroposterior tractions by the vitreous on the fovea, which lead to a retinal operculum as the posterior hyaloid detaches from the fovea (Gaudric et al. 1999; Spaide 2005). Pathogenesis of traumatic macular holes is more uncertain; it may be the result of a break in the foveal inner retina and a cyst formation extending laterally to the outer retinal tissue (Oehrens & Stalmans 2006).

In the present case, the pathogenesis seems to be different from that of idiopathic macular holes because the full-thickness macular hole appeared while the posterior hyaloid remained attached to the macula, far away from the edges of the full-thickness macular hole. Therefore, it seems that anteroposterior tractions were not involved in the formation of the macular hole.

In our case, the full-thickness macular hole occurrence may be explained by the following mechanisms. First, the retinal thinning at the fovea is probably because of the degeneration of retinal cells, particularly the distended Muller cells, caused by prolonged and recurrent cystoid macular oedema (Spaide 2005). Second, the thin epiretinal macular membrane may have led to a tangential vitreous traction on the fovea, accounting for the development of the macular hole (Spaide

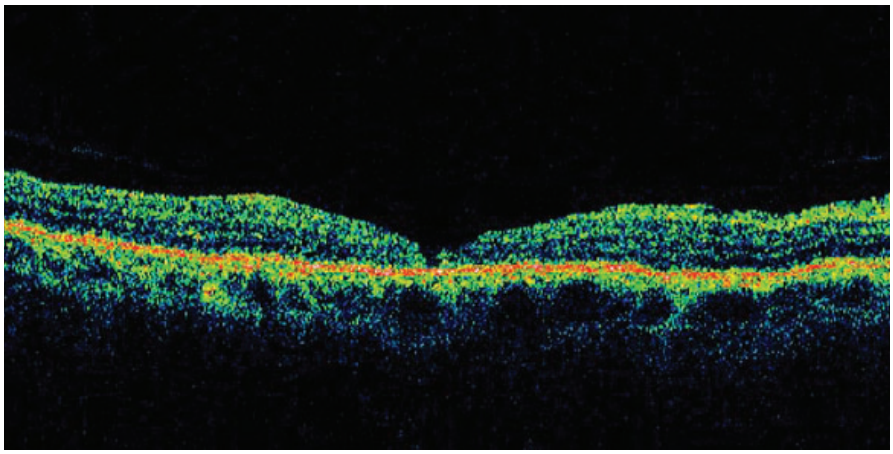

Fig. 3. Three additional intravitreal triamcinolone injections were performed because of the recurrence of macular oedema 6 months after each injection. Three months after the fourth triamcinolone injection, OCT demonstrated macular thinning with a full-thickness macular microhole without raised, thickened edges. The perifoveal posterior hyaloid detachment remained identical.

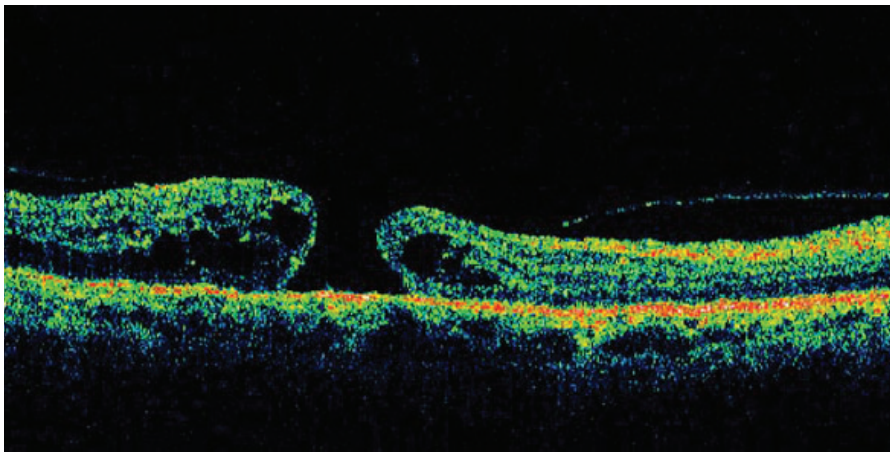

Fig. 4. Four months after the fourth triamcinolone injection, the macular hole enlarged and its edges became raised and thickened. The perifoveal posterior hyaloid detachment remained identical.

2005). Finally, the presence of a vitreoschisis, which is frequently found in patients with severe diabetic retinopathy, may account for some additional degree of tangential vitreous traction (Chu et al. 1996; Spaide 2005).

The role of intravitreal triamcinolone injections in the occurrence of the full-thickness macular hole remains unclear.

Even if corticosterone hypersecretion has been reported to induce hippocampal neuron atrophy in rat models – via excitatory mechanisms involving N-methyl-D-aspartate receptors (Magarinos & McEwen 1995) – there is no sufficient data in the literature in favour of some retinotoxicity of intravitreal corticosteroid injections. Therefore, it is unlikely that intravitreal triamcinolone injections had directly led to the retinal atrophy.

However, recurrent intravitreal triamcinolone injections may have had an indirect role in the development of the macular hole. Successive significant variations of retinal thickness following each triamcinolone injection (Ozdemir et al. 2005; Audren et al. 2006) may have favoured the rupture of distended Muller cells and intraretinal pseudocysts. Moreover, recurrent intravitreal triamcinolone injections might have led to tangential vitreal tractions.

In conclusion, we report here a case of progressive thinning of the neurosensory retina at the fovea followed by the occurrence of a full-thickness macular hole in a patient with diabetic cystoid macular oedema treated by intravitreal triamcinolone injections – a case that, to our knowledge, has not been previously described using OCT. Pathogenesis of the present full-thickness macular hole related to diabetic macular is different from that of idiopathic macular holes. The exact contribution of triamcinolone acetonide injections to the occurrence of the full-thickness macular hole remains unclear, but recurrent intravitreal triamcinolone injections may have had an indirect role in the development of the macular hole, by favouring the rupture of intraretinal cysts after successive significant variations of macular retinal thickness.

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