Macular hole after intravitreal ranibizumab injection for diabetic macular edema

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Background

Pathogenesis of macular hole, which remains incompletely elucidated, is suggested that it develops due to anterior-posterior traction and tangential traction on macula. Anterior-posterior traction is caused by incomplete posterior vitreous detachment and abnormally remained vitreomacular traction. Tangential traction comes from a contraction of vitreous cortex caused by proliferation and migration of Muller cells. In eyes with diabetic retinopathy, pathogenesis of macular hole is similar to that of idiopathic macular hole. Some cases of full thickness macular hole associated macular edema have been reported, but its pathogenesis is not completely understood.

Ranibizumab (Lucentis; Genentech, Inc., San Francisco, CA; co-developed by Genentech, Inc., and Novartis) is a useful drug for many intraocular disorders such as age-related macular degeneration, macular edema secondary to diabetic retinopathy and retinal vascular disease. Some cases of full thickness macular hole after intravitreal ranibizumab has been reported, but there was no case of full thickness macular hole after intravitreal ranibizumab for treatment of diabetic macular edema.

Purpose

To report a case of macular hole after treatment with intravitreal ranibizumab injection for diabetic macular edema.

Case report

A 76-year-old diabetic women came to our clinic with decreased vision of right eye. She had a history of diabetes over 30 years and did not have any history of ocular surgery. Best corrected vision were 20/50 on both eyes. She had non-proliferative diabetic retinopathy. Macular edema was noted on fundus examination and optical coherence tomography (OCT). In addition to macular edema, thinning of fovea and vitreofoveal detachment was noted on OCT. External limiting membrane was intact. (Fig 1).

Intravitreal injection of ranibizumab was performed to treat macular edema. One month after intravitreal injection of ranibizumab, a full thickness macular hole was present on OCT (Fig 2). Vitrectomy was planned for treatment of the macular hole. Before vitrectomy, with informed consent, intravitreal injection of bevacizumab (Avastin; Genentech, Inc., San Francisco, CA) was performed. One week after intravitreal injection of bevacizumab, macular hole was still present and size of macular hole was enlarged. (Fig 3). Total vitrectomy, internal limiting membrane peeling and intraocular gas tamponade were performed. Six months after surgery, the macular hole was closed on OCT (Fig 4).

Discussion

In this case, as tissues above the external limiting membrane in the fovea were thin on OCT before intravitreal injection of ranibizumab, we thought it became weakened due to posterior vitreous detachment. As complete vitreofoveal detachment was noted on macula, we thought that intravitreal injection would not be harmful in this case. So, we performed intravitreal injection of ranibizumab to resolve macular edema.

One month after intravitreal injection of ranibizumab, full thickness macular hole was noted on OCT. Intravitreal injection might cause formation of macular hole, but we thought that intravitreal injection was not a major factor because complete vitreofoveal detachment was present. With assumption that resolution of macular edema might induce a closer fit of macular hole, we performed intravitreal injection of bevacizumab before vitrectomy with informed consent. But this was not effective. This may be due to insufficient effect of bevacizumab on the macular edema. Also intravitreal injection of anti-VEGF did not resolve tangential traction caused by remained vitreous cortex.

Pathogenesis of macular hole associated with macular edema is not clearly elucidated. But macular edema has been suggested to be one of the factors inducing a full thickness macular hole. Some cases about closure of macular hole after treatment with intravitreal triamcinolone were reported. But in our case, macular hole associated diabetic macular edema was not closed after intravitreal bevacizumab injection.

In this case, macular hole might have been induced by diabetic macular edema in eyes with weak foveal center. And injection of ranibizumab and subsequent bevacizumab might have been ineffective on the macular edema. We don't know whether macular hole is closed if it is treated with intravitreal triamcinolone.

In conclusion, we report a case of macular hole formation after intravitreal ranibizumab injection for treatment of diabetic macular edema. The exact contribution of ranibizumab injection to the occurrence of the full-thickness macular hole remains unclear. For macular hole closure, intravitreal injection of bevacizumab was ineffective and surgery was needed finally.

References