Resolution Of Vitreomacular Traction After a Single Intravitreal Injection of Ranibizumab In a Patient with Diabetic Macular Edema

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ABSTRACT

A 63-year-old Caucasian woman presented with the complaint of decreased visual acuity and metamorphopsia in her right eye. She was diagnosed with vitreomacular traction (VMT) and concurrent diabetic macular edema (DME). The patient's best corrected visual acuity was 3/10 and central macular thickness (CMT) was 387 µm in the right eye. Intravitreal anti-VEGF injection was recommended for the treatment of symptomatic VMT with DME which did not show spontaneous improvement. One month later, her BCVA increased to 7/10 and VMT resolved (CMT decreased to 247 µm). Visual acuity and fundus findings remained stable with no additional treatment during the one-year follow-up.

Keywords: Diabetic macular edema, Intravitreal injection, Vitreomacular traction.

INTRODUCTION

Vitreomacular traction (VMT) syndrome is a disorder of vitreomacular interface in which incomplete detachment of the vitreous results in macular traction.[1] VMT frequently leads to decreased visual acuity (VA) and symptomatic metamorphopsia. VMT may be found with increased incidence in disorders such as diabetic retinopathy.[2] This report describes the resolution of VMT and concurrent diabetic macular edema (DME) after a single intravitreal injection of Ranibizumab documented by spectral domain-optic coherence tomography (SD-OCT).

CASE PRESENTATION

A 63-year-old Caucasian woman with type 2 diabetes mellitus presented with decreased vision and metamorphopsia in the right eye. The patient was treated in our clinic a few years ago for proliferative diabetic retinopathy with panretinal laser photocoagulation and intravitreal anti-VEGF injection. Her left eye had undergone vitrectomy a year ago for VMT with DME at another clinic. The fundus examination revealed bilateral panretinal laser scars, macular edema in the right eye and a dull foveal reflex in the left eye. Best corrected visual acuity (BCVA) was 3/10 in the right eye and 1/10 in the left eye. Spectral domain optic coherence tomography (SD-OCT) (RTVue, OptovueInc.,Fremont, CA, USA) demonstrated macular cystoid edema and incomplete posterior vitreous detachment with macular traction in the right eye [central macular thickness (CMT) was 387 µm (Figure 1)] and foveal thinning in the left eye (CMT was 223 µm). She was observed for a month but VMT did not resolve spontaneously. Since the patient was symptomatic, intravitreal anti-VEGF injection was recommended. After informed consent was obtained, she was treated with a single intravitreal injection of Ranibizumab (0.5 mg, Lucentis; Genentech, Inc., South San Francisco, CA).

One month later, her BCVA increased to 7/10 in the right eye, and SD-OCT (figure 2) revealed resolution of the macular cysts and macular traction (CMT decreased to 247 µm). Visual acuity and fundus findings in the right eye remained stable with no additional treatment during the one-year follow-up (figure 3).

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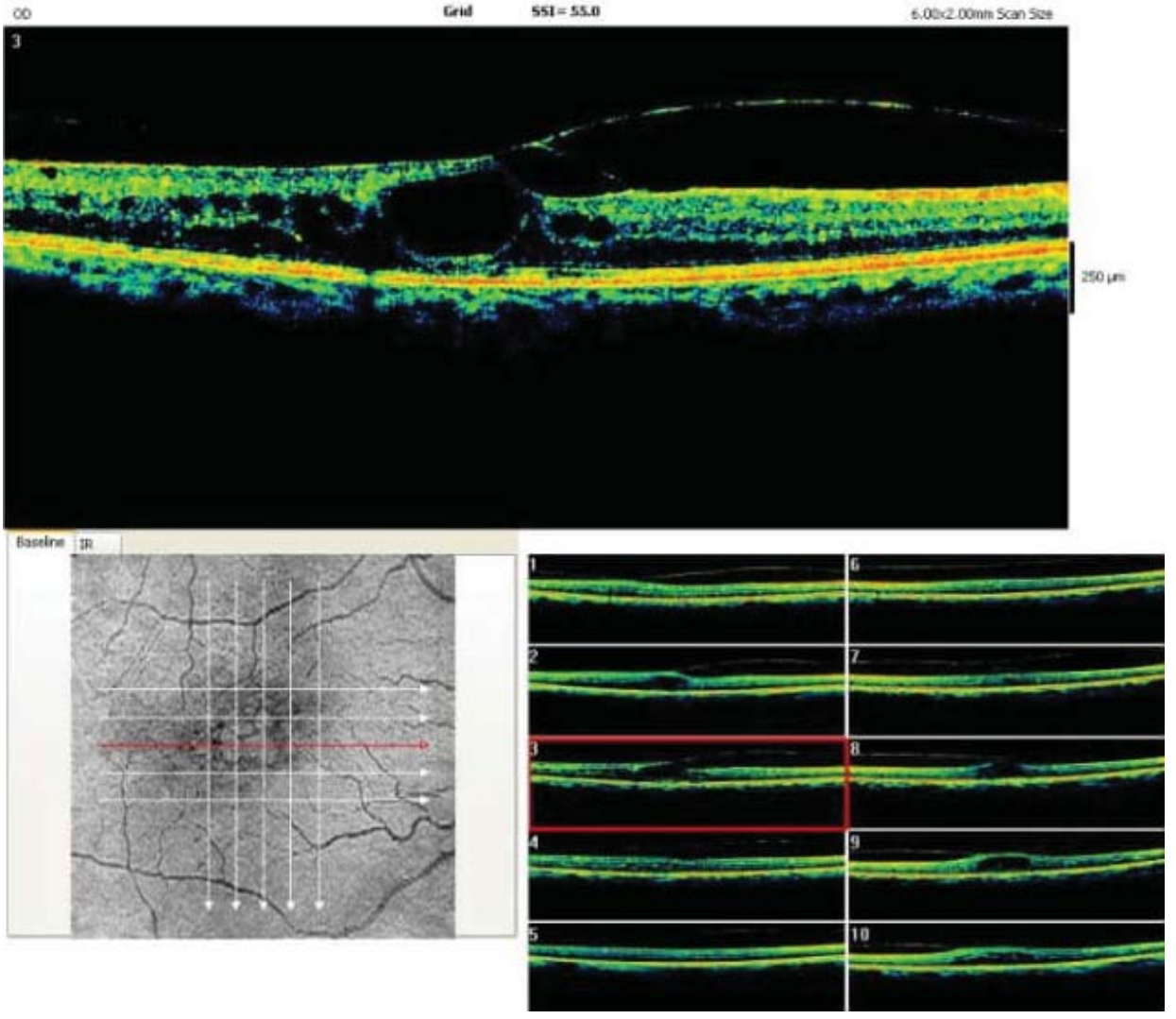

Figure 1. *SD-OCT of the right eye showed vitreomacular traction and cystoid macular edema with increased macular thickness.*

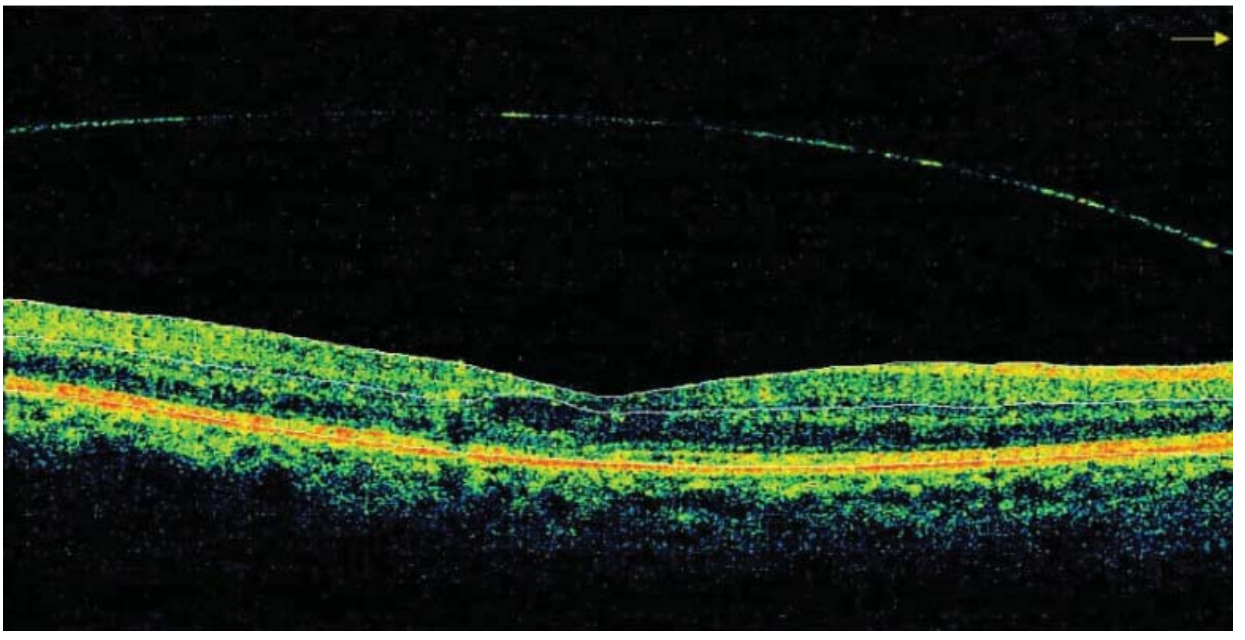

Figure 2. *At one month after anti-VEGF treatment, SD-OCT revealed resolution of vitreomacular traction and macular edema (CMT was 247 µm).*

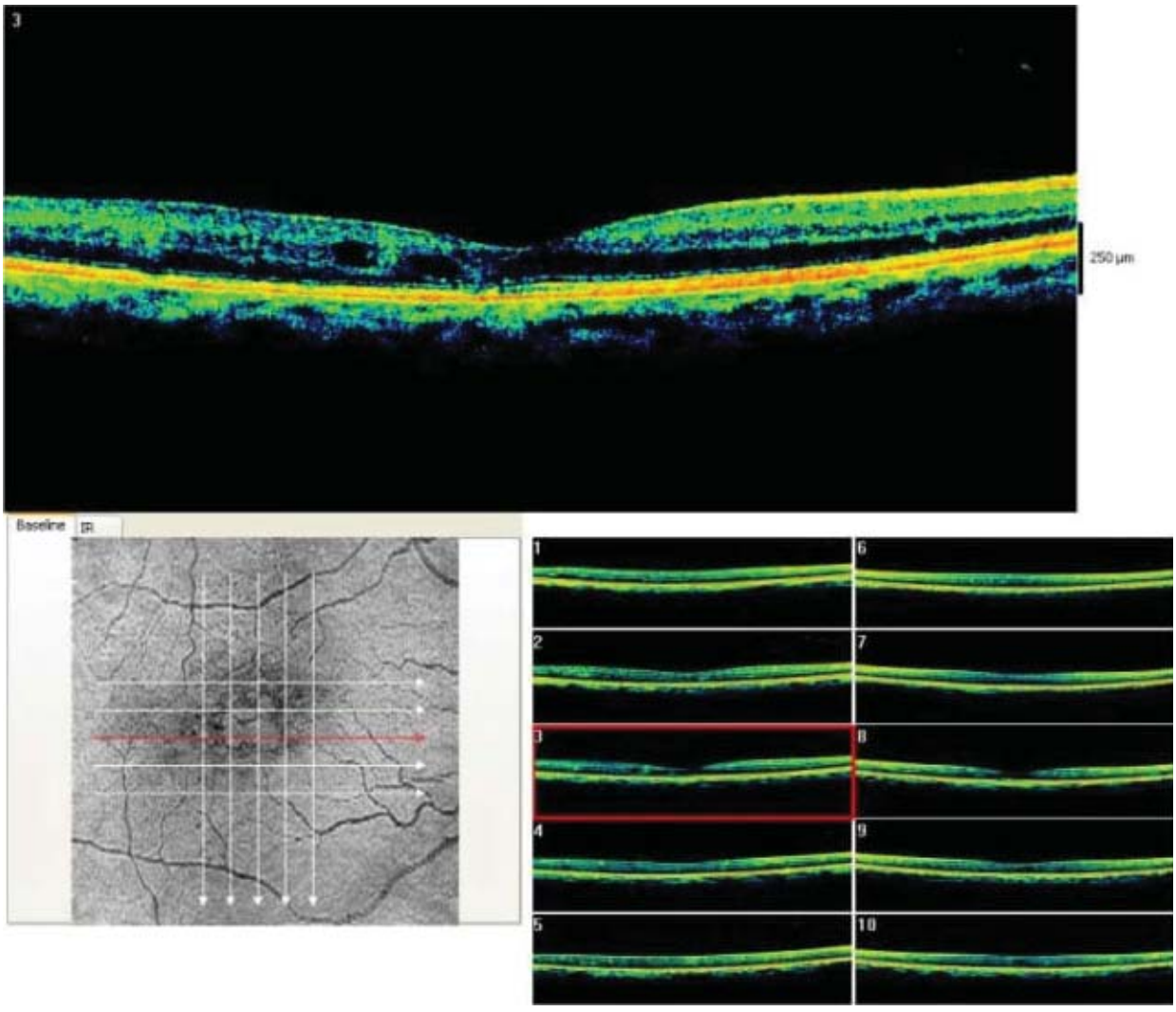

Figure 3. *At 12 months' follow-up, OCT showed a maintained foveal contour and CMT was 237 µm.*

DISCUSSION

DME is the most common cause of vision loss in the patients with diabetic retinopathy. The pathophysiology of DME is complex and multifactorial. DME may be exacerbated by vitreomacular traction effects of incomplete posterior vitreous detachment (PVD).[3] VMT syndrome is a disorder caused by incomplete detachment or separation of the posterior vitreous with persistent macular traction or adhesions causing decreased visual acuity, metamorphopsia, and monocular diplopia. [1] Studies show that prolonged VMT may lead to progressive vision loss. [4] The natural history of VMT with DME is unclear, and the literature includes inadequate data which treatment modality is to be chosen. Some authors advise observation of the patients for a period as an option because of the probability of spontaneous resolution.[5] The incidence of spontaneous VMT resolution was reported to be 11% by Hikichi et al.[6] In a study by Errera et al., spontaneous improvement was observed in 20% of 183 eyes with VMT. Despite spontaneous resolution of VMT without any treatment, resolution may take longer time in some cases.[7] The options for clinicians for the treatment of symptomatic vitreomacular traction include vitreous surgery, pharmacologic vitreolysis with ocriplasmin, or pneumatic vitreolysis.[8] Studies suggest that vitreomacular separation may increase the resolution of DME in some cases. [9] This report presents the case of symptomatic VMT and concurrent DME that treated with a single-dose intravitreal injection of ranibizumab. Several randomized clinical trials such as RISE and RIDE and RESOLVE have demonstrated the efficacy and safety of ranibizumab injections for the treatment of DME.[10,11] Anti-VEGF therapy characterized by anti-inflammatory and anti-angiogenic effects. However, there are few reports about the resolution of VMT with DME after iv injection of anti VEGF in the literature.[12]

Geck et al.[13] followed 61 patients with attached posterior hyaloid after one or more intravitreal injections over an

11.1-week period, during which 15 eyes (24 %) presented a PVD. They suggested that intravitreal injection itself may induce a PVD in patients with macular disease. The precise mechanism of the induction of PVD or resolution of VMT by means of intravitreal injection is unknown.[13] The possible mechanisms that might have played a role in the resolution of vitreomacular traction are the liquefaction of the vitreous gel and mechanical increase of vitreous volume caused by intravitreal injection, which may accelerate the development of complete PVD. [12,13] In addition to these effects, in the present case, it is thought that there was also a functional effect due to anti-VEGF-induced retinal thickness reduction.

In this report, it was demonstrated that rapidly improving and sustaining BCVA was obtained with single-dose ranibizumab therapy. CMT was reduced significantly from baseline (−164 μm) and VMT resolved completely.

CONCLUSION

Intravitreal injection of anti-VEGF may be a reasonable alternative therapy for selected patients with vitreomacular traction and concurrent DME. However, further studies are needed on the management for VMT with DME.

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