

## CASE REPORT

# Optical Coherence Tomography in Spontaneous Resolution of Vitreomacular Traction Syndrome

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Vitreomacular traction syndrome (VTS) is a vitreoretinal interface abnormality. The disorder is caused by incomplete posterior vitreous detachment with persistent traction on the macula that produces symptoms and decreased vision. Most symptomatic eyes with VTS undergo a further decrease in visual acuity. Spontaneous complete vitreomacular separation occurs infrequently in eyes with VTS. Surgical intervention may be considered if severe metamorphopsia and decreased visual quality occur. Herein, we report 2 typical cases of idiopathic VTS with spontaneous resolution of vitreoretinal traction demonstrated by optical coherence tomography. Optical coherence tomography is a sensitive and useful tool for the confirmation of diagnosis and for the serial anatomical evaluation of patients with VTS. [*J Chin Med Assoc* 2010;73(6):334–337]

**Key Words:** optical coherence tomography, posterior vitreous detachment, vitreomacular traction syndrome

## Introduction

Vitreomacular traction syndrome (VTS) is an idiopathic disorder associated with incomplete posterior vitreous detachment (PVD). PVD is a degenerative condition in which posterior hyaloid membrane of vitreous separates from the retina surface. It can be a spontaneous or traumatic process. VTS has persistent vitreous traction and cystoid changes at the macula causing clinical symptoms such as metamorphopsia, micropsia, photopsia, and decreased visual acuity.<sup>1</sup> Traditionally, slit-lamp biomicroscopy has been used to detect the macular vitreoretinal adhesions. However, this technique may underestimate or miss the subtle changes of vitreoretinal interface abnormalities. Recently, optical coherence tomography (OCT) has provided a facilitative method for the diagnosis and image assessment of the vitreoretinal interface. In the natural course of VTS, only about 11% of patients developed spontaneous complete PVD during follow-up.<sup>1</sup> Herein, we report 2 typical cases of idiopathic VTS, and describe the spontaneous resolution of vitreomacular traction without surgical intervention, demonstrated by OCT.

## Case Reports

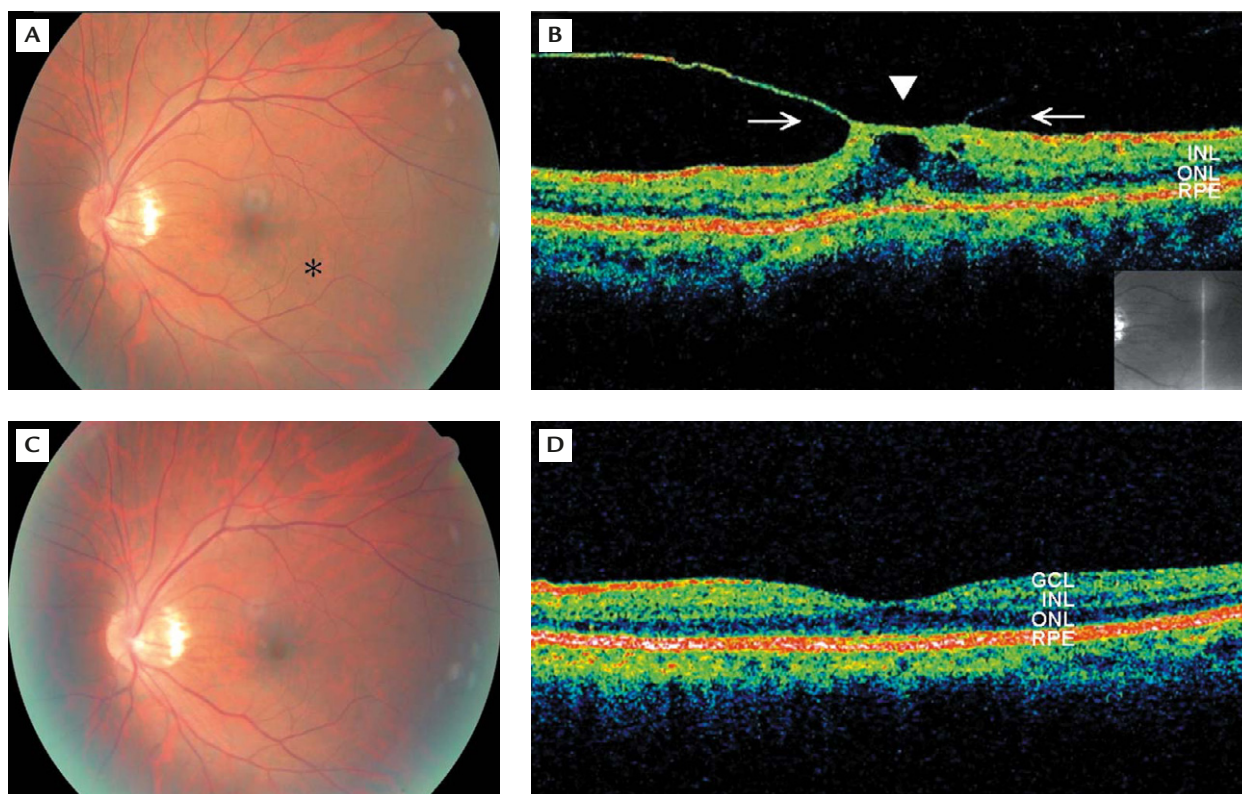
### Case 1

A 53-year-old male suffered from metamorphopsia in his left eye for 1 month. He denied any ocular trauma. Myopia with refraction of  $-5.75$  OD and  $-5.50$  OS was recorded. The visual acuity was 6/6 OU with glasses correction. The anterior segment was unremarkable. Fundus examination showed vitreous tractional maculopathy with retinal striae in the left eye. No Weiss ring was observed (Figure 1A). OCT (Stratus OCT; Carl Zeiss Inc., Dublin, CA, USA) revealed cystoid change, partial posterior hyaloid separation with vitreous attachment around the foveal center (shown as 2 highly reflective bands), upward tenting outer retina tissue and fovea retinal detachment (Figure 1B). Central retinal thickness (CRT) was  $402\ \mu\text{m}$ . VTS was impressed, and surgical intervention was suggested, but the patient declined. One month later, follow-up OCT examination revealed resumed foveal pit contour, no more vitreoretinal adhesion at the fovea, and subsided subretinal fluid with decreased CRT ( $216\ \mu\text{m}$ ). The patient subjectively felt resolution of visual distortion.



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**Figure 1.** (A) Fundus photograph showed tessellated fundus and vitreous tractional maculopathy with retinal striae (\*) in the left eye. (B) Optical coherence tomography showed partial posterior hyaloid separation with vitreous attachment around the foveal center (shown as 2 highly reflective bands; arrows), upward tenting outer retina, and foveal retinal detachment (arrowhead). (C) Four months later, fundus photograph revealed spontaneous resolution of vitreomacular traction. (D) Optical coherence tomography showed separation of vitreous traction with recovery of normal retinal architecture, normal foveal pit and absence of subretinal fluid. GCL = ganglion cell layer; INL = inner nuclear layer; ONL = outer nuclear layer; RPE = retinal pigment epithelium.

Three months later, visual acuity remained 6/6 and in stable condition. There were no retinal striae or vascular traction at the macula (Figure 1C). Normal foveal pit with macular contour recovery was noted (CRT, 210  $\mu$ m) by OCT (Figure 1D).

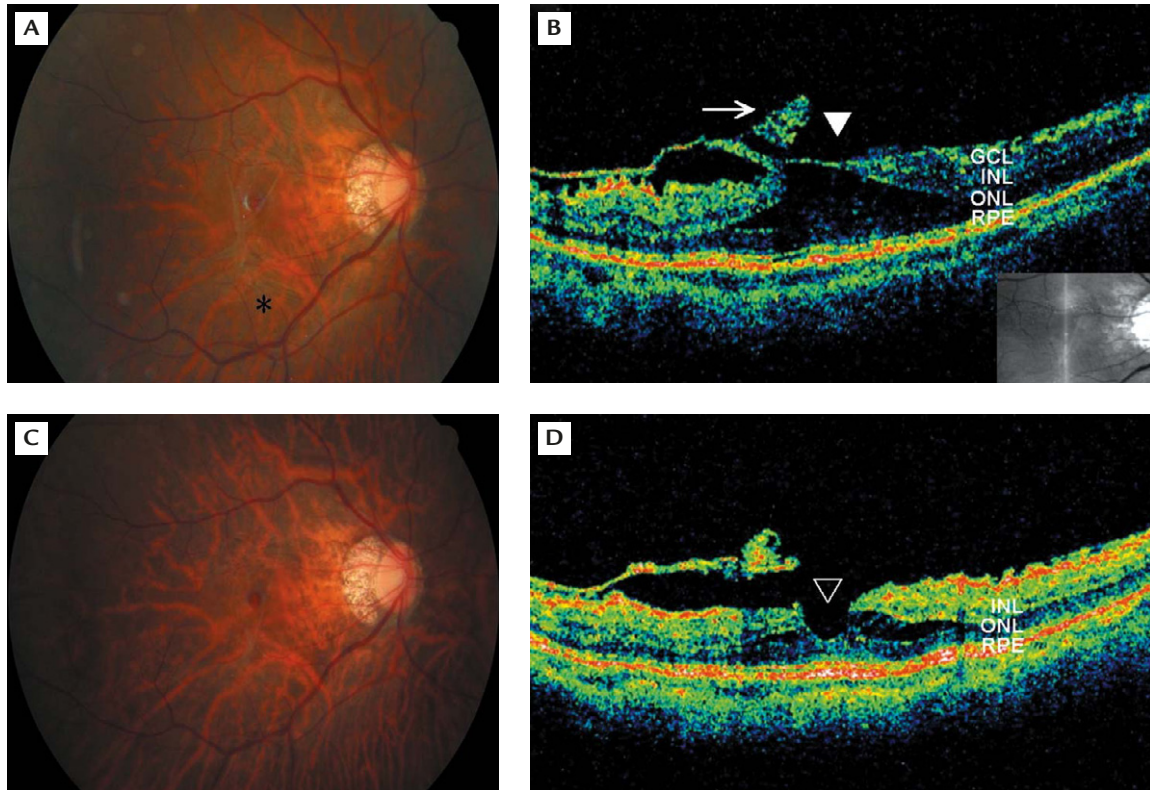
### Case 2

A 48-year-old woman suffered from metamorphopsia in her right eye for several months. High myopia with refraction of  $-8.75-0.5 \times 175$  OD and  $-7.5-2.0 \times 165$  OS was recorded. Visual acuity was 6/7.5 OD and 6/6.7 OS with glasses correction. The anterior segment was normal. However, preretinal vitreous traction with retinal striae in the right eye and cellophane maculopathy in the left eye were noted by indirect ophthalmoscope. Fluorescein angiography showed no peripapillary dye leakage in the late phase of the right eye. The patient was lost to follow-up for 1.5 years. When she returned to our clinic, her visual acuity had dropped to 6/8.6 in the right eye. More prominent preretinal vitreous tractional band at the macula with retinal striae in the right eye were noted (Figure 2A).

OCT revealed highly elevated, cystic change of the inner retina (CRT, 345  $\mu$ m) and vitreous traction band attached at the perifoveal area (Figure 2B) rather than a more generalized thickening of retina appearance with complete separation of vitreous in macular pucker. Three months later, fundus examination showed spontaneous partial resolution of vitreous traction at the superior aspect and margin of the fovea with residual retinal striae (Figure 2C). On OCT image, partial detachment of previous posterior cortical vitreous adhesion at the inferior border of the fovea with appearance of lamellar macular hole was noted in her right eye (Figure 2D).

### Discussion

Vitreous liquefaction and posterior vitreous separation are common phenomena in normal aging eyes. Incomplete vitreous separation and vitreoretinal adhesion may play a role in the development of macular diseases such as VTS, idiopathic macular hole and



**Figure 2.** (A) Fundus photograph showed preretinal vitreous tractional band with retinal striae (\*) in the right eye. (B) Optical coherence tomography showed posterior hyaloid vitreous tissue attachment at the macula (arrow) resulting in tenting, cystic change of the inner retina and foveal retinal detachment (arrowhead). (C) Three months later, fundus examination showed spontaneous partial resolution of vitreous traction at the superior aspect and margin of the fovea with residual retinal striae. (D) Optical coherence tomography showed focal separation and detachment of the previous cortical vitreous adhesion at the inferior foveal margin with impending lamellar hole (arrowhead). The outer retinal layer was still retained. GCL=ganglion cell layer; INL=inner nuclear layer; ONL=outer nuclear layer; RPE=retinal pigment epithelium.

macular pucker. All 3 conditions may affect visual acuity. On the differential diagnosis by OCT finding, elevated fovea and tented outer retina with vitreous tractional band attached on retina is noted in VTS. Complete separation of posterior cortical vitreous with retinal thickening caused by epiretinal membrane is found in macular pucker. Partially or fully dehiscent retinal tissue at the macula with different degrees of vitreous detachment can be seen in idiopathic macular hole. VTS is one of the vitreoretinal interface disorders. The disorder is caused by incomplete PVD with persistent traction on the macula. The natural course of VTS has been described by Hikichi and associates;<sup>1</sup> 81% of patients had cystoid macular changes, of which 67% had persistent condition during a follow-up period of 60 months. The visual acuities at final examination decreased 2 Snellen lines or more from baseline in 34% of cases with VTS. Only 11% of patients with VTS developed complete PVD. In our Case 1, although foveal retinal detachment with focal adherent vitreous traction attachment at the perifoveal area was initially

noted, spontaneous resolution of vitreoretinal traction with PVD formation occurred later. The subjective visual symptoms improved subsequent to anatomical resolution on OCT evaluation.

Gallemore et al reported that OCT is more sensitive than biomicroscopy in identifying vitreoretinal adhesions associated with macular disease.<sup>2</sup> Two patterns of vitreoretinal adhesions were identified with OCT, focal or multifocal, in their study. In this report, we have also demonstrated that OCT is a sensitive, non-invasive and useful tool to diagnose and follow-up the spontaneous regression of VTS.

Yamada and Kishi used OCT to study the surgical outcome of VTS in relation to preoperative macular tractional status.<sup>3</sup> There are 2 types of vitreous traction development in VTS. In the first type of VTS, the vitreous is detached temporally and nasally to the fovea but remains attached at the fovea in the horizontal plane, forming an incomplete V-shaped PVD. In the other type, uneven detachment of vitreous is noted at the contrary side of the fovea, causing an

irregular pattern of vitreoretinal interface. The authors considered that patients with incomplete V-shaped PVD had the more favorable surgical outcome.<sup>3</sup> However, the other type of partial PVD temporal to the fovea may result in sequelae of macular hole or macular atrophy after surgery. The authors hypothesized that the former type had foveal detachment to relieve damage to inner retinal tissue without cystoid macular edema (CME) and no chronic uneven traction of retinal tissue, so the outcome was favorable. The latter type had weaker anterior traction force not sufficient to cause foveal detachment. The uneven and chronic traction force damaged the retinal tissue, leading to subsequent development of CME, so postoperative macular hole or atrophy may occur. Our Case 1, who had incomplete V-shaped vitreous detachment, eventually developed spontaneous complete resolution of vitreoretinal traction and had a favorable outcome. However, our Case 2, who had partial PVD adjacent to the fovea on OCT, developed partial resolution of vitreous traction with residual symptom and impending lamellar macular hole.

Smiddy et al also classified VTS into 3 patterns during vitrectomy according to the vitreomacular anatomy.<sup>4,5</sup> In the serial anatomical evaluation of our patients with VTS, we also observed that there was either complete (Case 1) or incomplete (Case 2) PVD occurrence demonstrated by OCT. Different types of VTS may have inherent different visual outcomes. We hypothesized that different VTS may exert different vectors of traction force in the process of PVD development and vitreous separation. Moreover, another factor affecting the visual outcome is the duration of symptoms. When the inner retinal structure is dragged and distorted for a long period of time, the visual acuity may deteriorate. In our Case 2, the patient was lost

to follow-up for 1.5 years. Chronic traction damage of the macula may lead to retinal thinning and impending lamellar macular hole, and cause irreversible retinal dysfunction even after spontaneous vitreous separation.

In conclusion, we have reported 2 cases of VTS with spontaneous separation of posterior hyaloid vitreomacular traction. We have demonstrated that OCT is a sensitive and useful tool for the confirmation of diagnosis, understanding of pathogenesis, and serial anatomical evaluation of patients with VTS.

## Acknowledgments

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## References

1. Hikichi T, Yoshida A, Trempe CL. Course of vitreomacular traction syndrome. *Am J Ophthalmol* 1995;119:55-61.
2. Gallemore RP, Jumper JM, McCuen BW 2nd, Jaffe GJ, Postel EA, Toth CA. Diagnosis of vitreoretinal adhesions in macular disease with optical coherence tomography. *Retina* 2000;20:115-20.
3. Yamada N, Kishi S. Tomographic features and surgical outcome of vitreomacular traction syndrome. *Am J Ophthalmol* 2005;139:112-7.
4. Smiddy WE, Michels RG, Glaser BM, deBustros S. Vitrectomy for macular traction caused by incomplete vitreous separation. *Arch Ophthalmol* 1988;106:624-8.
5. Smiddy WE. Vitreomacular traction syndrome. In: Yanoff M, Duker JS, eds. *Ophthalmology*, 2<sup>nd</sup> edition. St Louis: CV Mosby, 2004:951-5.