Vitreous haemorrhage caused by unusual giant macular tear: a case report

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Abstract
Macular tears rarely occur without trauma. Here, we describe a patient with vitreous haemorrhage, which was caused by an unusual giant macular tear secondary to existing branch retinal vein occlusion. A 60-year-old woman presented with vision loss in the right eye because of vitreous haemorrhage. She had a history of branch retinal vein occlusion and had been treated with retinal photocoagulation 3 years prior. As treatment for vitreous haemorrhage, the patient underwent 23-gauge pars plana vitrectomy combined with silicone oil tamponade. During the operation, a large jagged tear was observed in the macula. We presumed that stretching of the fibrous proliferating membrane secondary to branch retinal vein occlusion was responsible for the macular tear and vitreous haemorrhage. Eventually, the results of pars plana vitrectomy led to anatomical closure of the macular tear and partial restoration of visual acuity.

Keywords
Branch retinal vein occlusion, macular tear, pars plana vitrectomy, fibrous proliferating membrane, vitreous haemorrhage, silicone tamponade, visual acuity, retinal photocoagulation

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Introduction
Vitreous haemorrhage frequently manifests concomitantly with profound vision impairment. The primary causes of vitreous haemorrhage include diabetic retinopathy, branch retinal vein occlusion (BRVO), trauma, retinal tear, rhegmatogenous retinal detachment, and posterior vitreous detachment.¹⁻³ However, vitreous
haemorrhage is rarely caused by a macular tear. To the best of our knowledge, only a few cases of macular tears have been reported in the literature.\textsuperscript{1–3} To facilitate better clinical understanding of macular tears, this report describes the details and corresponding etiological analysis of a patient with a giant retinal tear in the fovea.

**Case report**

In February 2017, a 60-year-old woman presented with vision loss in the right eye, which she had experienced for 14 days. Several days prior, the patient performed a series of movements, including bending, crouching, and quickly standing up; she noticed a fluttering shadow in front of her eyes. Subsequently, she was diagnosed with vitreous haemorrhage and treated with haemostatic agents and Chinese patent medicine. However, her vision did not improve. The patient’s medical history included hypertension for the previous 10 years, as well as retinal photocoagulation treatment for BRVO, 3 years prior.

Initial ocular examination revealed that the patient’s best-corrected visual acuity results were hand movement in the right eye and 120/200 in the left eye; intraocular pressure values were 11.1 mmHg in the right eye and 14.4 mmHg in the left eye. Moreover, both eyes exhibited cataracts (Lens Opacities Classification:\textsuperscript{4} C1N1O0). Fundus examination of the patient revealed fresh bright red vitreous haemorrhage in the right eye; no obvious abnormalities were observed in the left eye (Figure 1a,b). Ultrasound revealed vitreous haemorrhage in the right eye (Figure 1c).

The patient provided informed written consent for surgery under general anaesthesia, then underwent 23-gauge pars plana vitrectomy combined with silicone oil tamponade. During the operation, a large jagged macular tear was observed (Figure 2a). The superior temporal retinal blood vessels exhibited abnormal narrowing into white lines (i.e., atresia). Four days postoperatively, fundus photos revealed that the macular tear had been reattached in the correct anatomical location (Figure 2b). Spectral-domain optical coherence tomography revealed that the inner retinal layer exhibited oedema and the macular tear had completely closed (Figure 2c, d). Fluorescein and indocyanine green angiography revealed that the macular area was torn in both retinal and choroidal layers (Figure 3a–c). One month postoperatively, the patient’s best-corrected visual acuity had improved to 2/200 in the right eye.

![Figure 1](image1.png)

**Figure 1.** Preoperative images. (a) Fundus examination showed dense vitreous haemorrhage in the right eye. (b) No obvious abnormalities were present in the left eye. (c) B-scan ultrasound showed vitreous haemorrhage in the right eye.
Discussion

Macular tears are rare complications that can cause serious visual impairment, regardless of iatrogenic or traumatic aetiology. According to the Gass theory of macular hole formation, vitreous traction is a major cause of macular tears. Here, we propose and discuss other potential causes and mechanisms involved in macular tears.

First, during retrospective analysis of our patient’s records, we definitively
excluded iatrogenic injury, such as the common retrobulbar injection injury. Our patient underwent surgery with general anaesthesia, which did not include retrobulbar injection anaesthesia. Moreover, the surgeon ruled out the possibility of iatrogenic retinal injury. Therefore, iatrogenic injury was not regarded as a potential contributing factor in our patient.

Another frequently reported cause of macular holes is blunt trauma. Karaca et al. described a patient in whom a horseshoe-like macular tear developed after blunt ocular trauma.1 In such

Figure 3. Postoperative fluorescein and indocyanine green angiography images. (a–c) Fluorescein and indocyanine green angiography revealed that the macular area was torn in both retina and choroid layers (a, early stage; b, middle stage; c, late stage).
instances, sudden compression and expansion of the eye produce tractional stress on the retina at the point of vitreous attachment. Thus, the direct contrecoup effect of the traumatic force, distortion of the vitreous body, and sudden traction of the macula may explain the formation of macular holes. Notably, our patient had no definite history of trauma; therefore, the possibility of a traumatic injury etiology was also excluded.

Thus far, only Kubota et al. have described a patient with idiopathic macular tears, which revealed that such tears can occur in patients without histories of ophthalmic disorder or injury (i.e., patients without BRVO or blunt ocular trauma). Karaca et al. proposed that if vitreous attachment is not equal on all sides of the macula, sudden traction exerted on the macula could result in a macular tear. Similarly, Kubota et al. speculated that the onset of adhesion or sudden traction between the retinal tear and the vitreous could result in an idiopathic horseshoe-like macular tear.

Secondary macular tears have been described in a few patients with BRVO. In 1997, Ikuno et al. reported that BRVO is occasionally complicated by two types of retinal breaks (i.e., retinal holes without vitreous traction or retinal traction tears), which may lead to rhegmatogenous retinal detachment. In 2007, Karim-Zade et al. described a patient with a horseshoe-like macular tear secondary to recurrent BRVO. They suggested that preoperative photocoagulation treatment for BRVO could contribute to extensive vitreoretinal adhesion, thus resulting in retinal traction tears. However, Russell et al. reported that none of four patients with BRVO-related retinal traction tears had received preoperative laser treatments to the midperipheral retina. Therefore, the role of photocoagulation in BRVO-related retinal tears is unclear.

Based on a comprehensive analysis of the above aetiologies and mechanisms, we presume that the macular tear in our patient occurred secondary to BRVO. The patient had a clear medical history of BRVO and had undergone fundus photocoagulation treatment. Intra- and postoperative images showed atresia of the patient’s superior temporal retinal vessels, in combination with the formation of fibrous proliferative membranes. Regarding the mechanism of macular tear formation, we suspect that chronic macular oedema and post-BRVO retinal ischemia led to macular thinning, which contributed to formation of the macular tear. Vitreous traction is another probable cause because a fibrous membrane had formed around the superior temporal vessels. Subsequent photocoagulation resulted in uneven vitreous traction around the macula, which may have contributed to formation of the macular tear. The final precipitating factor might have been the patient’s movement, which presumably promoted vitreous traction.

In conclusion, BRVO might contribute to the formation of macular tears. This condition might also be promoted by vitreous adhesion and traction. Additional investigations are needed to further elucidate the causes of macular tears.

Ethical statement and informed consent
We have completely removed all personal information from this report, so no consent to publish was sought from the patient. This case report was not required to be reviewed by the ethics review committee because it only discusses the aetiology of disease in a single patient and no exploratory treatment was performed.

Declaration of conflicting interest
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