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# Macular Branch Retinal Vein Occlusion: A Revisit and Case Report

***Abstract* —** Macular branch retinal vein occlusion (BRVO), a type of retinal vein occlusion, is rarely recognised as a distinct entity. Macular BRVO has unique clinical features and different natural courses than the major BRVO. We report a case of a young patient with macular BRVO with macular oedema who was successfully treated with intravitreal ranibizumab injection. A 43 year-old Chinese man with no underlying medical illness presented with 2 weeks history of left eye painless reduced central vision which was worsening over time. On examination, his left eye visual acuity was 6/30 and Amsler chart drawing showed a lower central scotoma. Dilated fundus examination found marked flame-shaped retinal hemorrhages with cotton wool spot over the superior macular area bounded superiorly by superior arcade and macular thickening. An optical coherence tomography revealed cystoid macular oedema; and fundus fluorescein angiography showed occlusion of a small venous branch draining a superior part of macula to superior temporal venous arcade. A complete medical investigation found that he has hypertriglyceridemia and he was managed accordingly. His vision had improved to 6/6 after receiving 3 injections of intravitreal ranibizumab with no residual central scotoma and complete resolution of macular oedema.

***Keywords* — Macular oedema, Retinal haemorrhage, Vein occlusion**

## 1 INTRODUCTION

Branch retinal vein occlusion (BRVO) is the second most common retinal vascular disorder after diabetic retinopathy [1, 2] with an overall incidence of 0.5 - 1.2% [3]. BRVO is divided into two distinct entities; major BRVO, and macular BRVO. Macular BRVO involves the superior macular region in 81% of cases and the inferior macular region in 19% of cases. Rarely, macular BRVO can develop in both regions and is called double macular BRVO [4]. Many BRVO articles discussed BRVO in general without recognising macular BRVO as a separate entity despite its distinct features. In this article, we present a case of successful treatment of macular oedema caused by macular BRVO and a review of its natural history.

## 2 CASE REPORT

A 43 year-old Chinese man, a teacher with no underlying medical illness presented with complaint of reduced central vision of left eye for 2 weeks. The vision was progressively deteriorating with central scotoma. There was no eye pain or eye redness. He was unaware of any precipitating event. He also denied of having any ocular trauma before. He consumed alcohol occasionally but he never smoke cigarettes. Otherwise he was healthy and never had any medical problem before.

Upon presentation, his visual acuities were 6/6 in the right eye and 6/30 in the left eye. Amsler chart showed a lower central visual field defect. There was no relative afferent pupillary defect elicited. The pupil, extraocular muscle, and anterior segment examinations of both eyes were unremarkable. Dilated fundus examination of left eye revealed multiple extensive flame-shaped retinal hemorrhages with cotton wool spot over the superior part of macula and bordered superiorly by the superior venous arcade (Figure 1A). The size of the affected area was around 5 to 6 disc diameter. The central macula was oedematous and thickened. There were no features suggestive of vasculitis or retinitis. The optic disc was pink and there was no signs of optic disc swelling. Optical coherence

tomography (OCT) examination showed thickening of macula with presence of cystoid macular oedema (Figure 2A). The central macular thickness was 690 μm.

A fundus fluorescein angiography (FFA) was performed at day 2 of presentation and revealed occlusion of a small venous branch draining a superior part of macula to supero-temporal venous arcade (Figure 3A). However, there were no signs of retinal vasculitis or choroiditis. Only a marked hypofluorescence area was noted which corresponded with the area of retinal hemorrhages due to masking effect.

A complete medical work up found that the blood pressure was normal with normal cardiovascular system. Baseline blood profile showed normal full blood count, renal profile and liver profile. Serum lipid profile revealed hypertriglyceridemia with the value of 2.22 mmol/L (normal range for male: 0.68 – 1.88 mmol/L) with normal value of high-density lipoprotein (HDL), low-density lipoprotein (LDL) and cholesterol (HDL: 1.35 mmol/L, normal range for male: 0.78 – 1.81 mmol/L; LDL: 2.67 mmol/L, normal range for male: 2.33 – 4.50 mmol/L; cholesterol: 5.03 mmol/L, normal range: 3.6 – 6.3 mmol/L respectively). Further blood screening for connective tissue disorder including anticardiolipin antibody were negative; as well as blood clotting disorders.

A diagnosis of right superior macular BRVO secondary to hypertriglyceridemia was made based on clinical findings and blood test. He was referred to the nearest general practitioner for the treatment of hypertriglyceridemia. Due to the significant macular oedema with marked central scotoma, the right eye was treated with 3 intravitreal injections of ranibizumab 0.5 mg given 1 month apart which started at the third week of presentation. Before completing the 3rd injection, his left eye visual acuity had improved from 6/30 to 6/6 with resolution of central scotoma. Nonetheless, the patient was planned to complete the total of 3 intravitreal ranibizumab 0.5 mg injections in order to stabilise the macular oedema. The vision remained stable after the third injection. A repeated OCT at 1 month after completing the third injection of intravitreal ranibizumab showed resolved macular oedema with central macular thickness of 255 μm (Figure 2B).

During the follow-up period, there was no fluctuation in his vision. At one year follow-up, the vision was 6/6 in both eyes. Right fundus examination showed a completely resolved retinal hemorrhages with presence of collateral vessels at the superior macular arcade (Figure 1B). There was no sign of retinal new vessel formation.

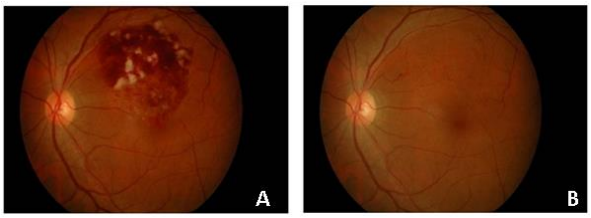


**Figure 1:** Left eye fundus photo showing multiple flame shaped retinal hemorrhages with cotton wool spots over superior part of macular at first presentation (A) and completely resolved retinal hemorrhages with formation of collateral vessels at 1 year follow-up (B).

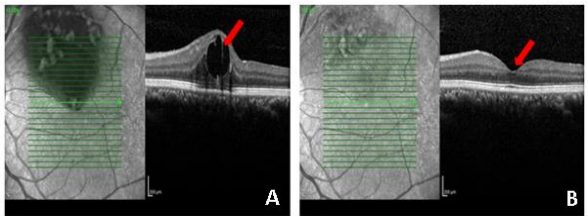


**Figure 2:** Optical computed tomography (OCT) showing cystoid macula oedema at first presentation (red arrow) (A) and completely resolved macular oedema (red arrow) at 1 month after completing the third injection of intravitreal ranibizumab (B).

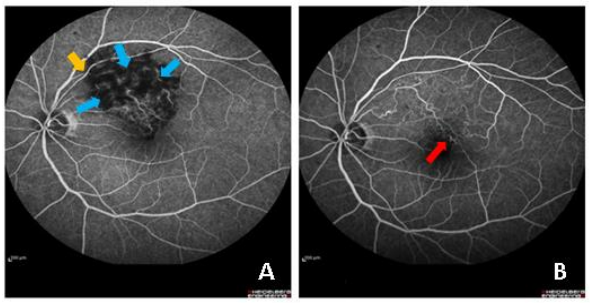


**Figure 3:** Fundus fluorescein angiography (FFA) showing occlusion of a small venous branch draining a superior part of macula (yellow arrow) to supero-temporal venous arcade with hypofluoresence (blue arrow) which corresponded with the area of retinal hemorrhages due to masking effect at first presentation (A) and a repeated FFA at 1 year follow up showing formation of collateral vessels (red arrow) (B).

OCT examination revealed normal macular contour with no macular oedema. Repeated FFA examination showed presence of collateral vessels with no leaking vessels and no new vessel formation seen (Figure 3B). The foveolar avascular zone (FAZ) appeared normal with no increased in size and no features suggestive of cystoid macular oedema. In view of stable vision and no complication of cystoid macular oedema or macular ischemia, the patient was discharged after 2 years follow-up.

## 3 DISCUSSIONS

Macular BRVO is an occlusion of a small venous branch draining a part of macula to either superior or inferior temporal venous arcade [5]. It is a subgroup of BRVO but rarely analysed as a separate entity. As in major BRVO, the etiology of this disease is due to vasculopathy.

Hypertensive, atherosclerotic, inflammatory, or thrombophilic conditions may lead to retinal endothelial vascular damage [6]. Arterial compression of the vein at the intersection is believed to be the main cause of BRVO. Compression of the vein may lead to a turbulent flow. The turbulent flow in combination with preexisting endothelial vascular damage from the different conditions creates a local environment favorable to intravascular thrombus formation [6]. When the venous flow is interrupted, retinal ischemia ensues downstream from the site of occlusion. Retinal ischemia is the most important factor for vascular endothelial growth factor (VEGF) expression [6].

Macular BRVO, as in major BRVO, typically presents as an acute and painless decrease in visual acuity, visual field defect, or monocular visual distortions with blurred or gray vision. Some patients may present with floaters due to a vitreous hemorrhage. Macular BRVO may result in significant morbidity, with visual acuity decreased to counting fingers [7]. As mentioned, the majority of macular BRVO involves the superior macular region as compared to the inferior macular region. The reason may be due to increase in arterial venous crossing at the area. In an acute stage, a dilated fundus examination may find retinal hemorrhages, nerve fibre layer infarct, hard exudate and macular oedema [5]. In late stage or chronic stage, the fundus examination findings may be very subtle [5]. Collateral vessels may form after 6 to 24 months which indicate good clinical prognosis [8].

Macular BRVO is diagnosed clinically. Two types of retinal tests aid the diagnosis of macular BRVO; FFA and OCT [9]. FFA in particular gives information on the presence of macular ischemia by showing enlargement of FAZ as well as the presence of collateral vessels. This information helps clinician to determine prognosis and to devise a treatment strategy. OCT is also a useful tool to monitor the disease progression.

The major complications of macular BRVO are the development of cystoid macular oedema and the presence of central macular ischemia which causes severe visual disturbances and vision loss. Macular oedema can resolve spontaneously within 1 year in about 50% of patients [9]. In a study by Hayreh et. al., he observed that after the resolution of macular oedema, visual acuity had improved in 58% of eyes with macular BRVO and worsened in 42% [9]. As a comparison, 76% of eyes with major BRVO had improved visual acuity and only 9% had worsened visual acuity after the resolution of macular oedema [9]. Majority of eyes with macular BRVO show enlargement of FAZ secondary to the break of the perifoveal capillary arcade which carries a poor visual prognosis [10]. It was also found that visual acuity impairment of patients with macular BRVO seemed to be related to FAZ enlargement [10].

Non-ischemic macular oedema therapeutically can be addressed with intravitreal anti-VEGF, corticosteroid, or focal laser treatment [6]. However, even with treatment there was lack of vision improvement in some eyes. This may be due to ischemic damage to macular retinal ganglion cells, pigmentary degeneration or epiretinal membrane that develop secondary to chronic macular oedema [9].

The rationale of using anti-VEGF (ranibizumab belongs to the group) in the treatment of our patient was based on evidence provided by many reliable clinical trials [2, 3]. We have also learned from our review that anti-VEGF was beneficial in the treatment of macular ischemia [11]. It has no effect on the formation of collateral vessels and we belief that the anti-VEGF possibly may contribute to the early reperfusion of the macula [8, 12].

## 4 CONCLUSION

In patients with macular BRVO, visual improvement following macular oedema resolution is poor as compared to major BRVO due to higher risk of macular ischemia. Although some patients may recover spontaneously, treatment with intravitreal anti-VEGF should be considered especially in patients who presented with poor visual acuity and those with high visual requirement.

## CONFLICT OF INTEREST

The authors declare no conflict of interests. The authors alone are responsible for the content and writing of the paper.

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