Ankyra Medical Journal

Case Report

DOI: 10.51271/ANKMJ-0038

Enhanced diagnosis of superior branch retinal vein occlusion with collateral vessels using optical coherence tomography angiography: a case study

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Cite this article: Havan C, Teke MY, Çıtırık M. Enhanced diagnosis of superior branch retinal vein occlusion with collateral vessels using optical coherence tomography angiography: a case study. *Ank Med J.* 2025;4(1):14-16.

Received: 01/12/2024 ◆ **Accepted**: 15/12/2024 ◆ **Published**: 06/01/2025

ABSTRACT

A 41-year-old woman was referred to our hospital because of decreased vision in the right eye. Fundus examination revealed microaneurysms, venous compression, empty veins, and formation of collateral vessels distal to the occlusion, which were diagnosed as superior branch retinal vein occlusion (BRVO). Collateral vessels, originating from the intermediate and deep capillary plexuses, were determined using optical coherence tomography angiography (OCTA) imaging. OCTA can play an important role in BRVO diagnosis and the evaluation of microvasculature architecture. This case emphasizes the important diagnostic capabilities of OCTA in visualizing microvascular changes, such as collateral vessels, foveal avascular zone (FAZ) changes, and capillary nonperfusion.

Keywords: Branch retinal vein occlusion, collateral vessels, optical coherence tomography angiography

INTRODUCTION

BRVO is a common retinal vascular disease. The major causes of visual impairment include varying degrees of retinal hemorrhage, retinal ischemia, and macular edema. The pathophysiology of BRVO involves arteriovenous crossing-related venous compression and subsequent occlusion. Capillary nonperfusion, vascular leakage, and macular edema eventually occur. Collateral vessel formation is an adaptive response observed in response to vascular blockage; its purpose is to restore blood flow and reduce ischemia.[1] We present a case of superior BRVO, focusing on collateral vessel formation and retinal microvasculature changes, using OCTA imaging.

CASE

A 41-year-old female patient was admitted to our clinic with a history of decreased vision in her right eye for more than 6 months. She denied any systemic comorbidities except for hypertension. On examination, the best-corrected visual acuity (BCVA) was 20/200 in the right eye (OD) and 20/20 in the left eye (OS). Intraocular pressure (IOP) was 14 mmHg on the right and 15 mmHg on the left. The anterior segment examination via slit-lamp biomicroscopy was normal. Dilated fundus examination of the right eye revealed arteriovenous crossings with venous compression and microaneurysms. Additionally, an empty vessel distal to the occlusion site was observed in the superior vascular arcade, which is indicative of vascular stasis (Figure 1a). These findings were compatible with a possible diagnosis of BRVO. Further diagnostic evaluations, including optical coherence tomography (OCT) and fluorescein angiography (FA), were planned to confirm the diagnosis and assess the extent of retinal involvement. OCT revealed a cystic appearance temporal to the fovea and macular thickening, consistent with macular edema secondary to vascular occlusion. FA of the right eye showed filling defects in the superior branch retinal vein, with collateral vessel formation distal to the occlusion site (Figure 1b, 1c). Additionally, mild FAZ enlargement was noted. OCTA demonstrated the collateral vessels very well, clearly showing their origin from the deep and intermediate capillary plexuses (Figure 1d). In the deep capillary plexus, shunt vessels appeared brighter or more prominent on OCTA images. This appearance was different from that of neovascularization, which has a more irregular and diffuse appearance.

DISCUSSION

Retinal vein occlusion is an important cause of vision loss. Macular edema is the main cause of deterioration in patients with BRVO.[1] Acute phase retinal vein occlusion causes macular edema, rupture of vessel walls, and intraretinal hemorrhages. Chronic phase microvascular changes include capillary dilation, nonperfusion, microvascularization, collateral formation, and retinal vascularization.[2] Studies have shown that the size of the FAZ, retinal vessel density, and capillary perfusion are

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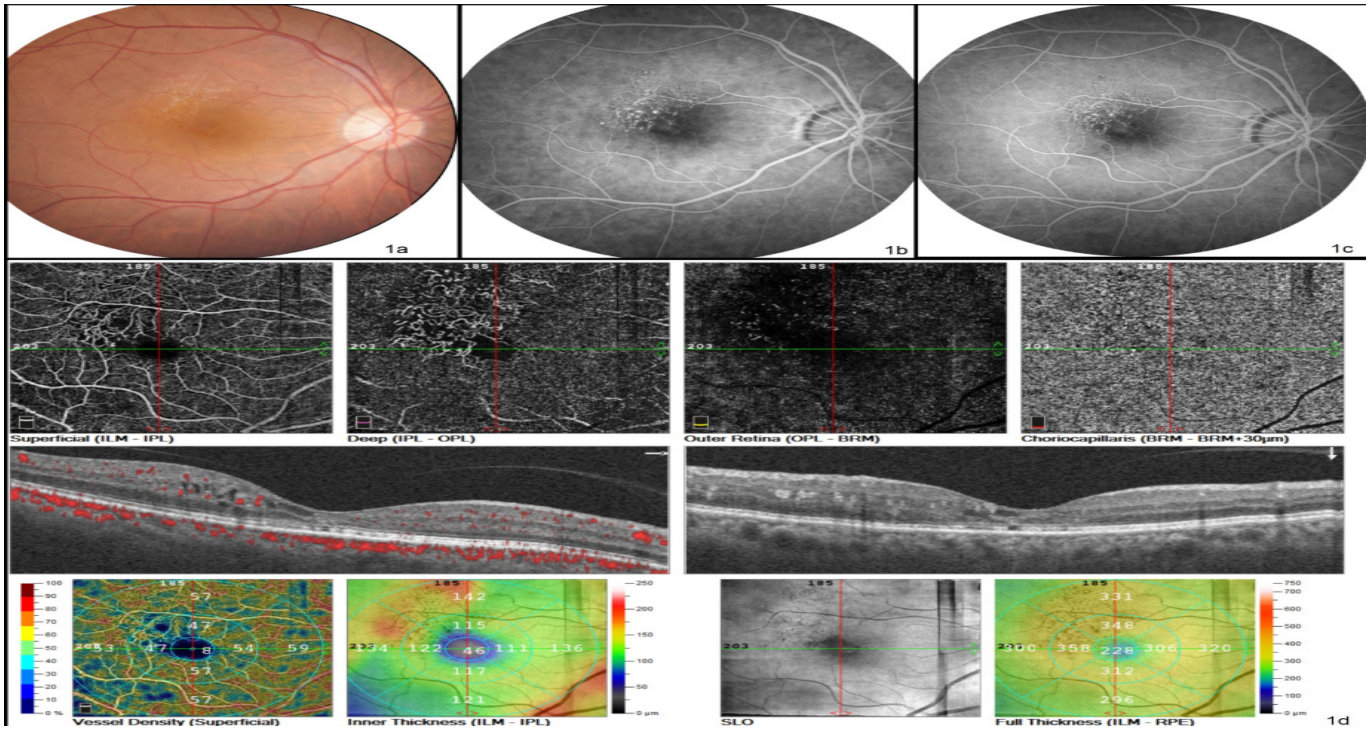

Figure 1a. Colored fundus photograph of the right eye; arteriovenous crossings with venous compression, microaneurysms, and an empty vessel distal to the superior vascular arcade. **1b-c.** Fluorescein angiography of the right eye; filling defects in the superior branch retinal vein with collateral vessel formation distal to the occlusion site. **1d.** Optical coherence tomography angiography of the right eye; the collateral vessels very well and clearly showing their origin from the deep and intermediate capillary plexuses

important determinants of visual outcomes. FAZ enlargement and decreased vascular density in the deep capillary plexus are major predictors of vision disability in BRVO.[3] FA is a gold standard for the diagnosis. However, it has certain limitations; for instance, it is an invasive procedure, carries a risk of allergic reactions to fluorescein, and provides inadequate visualization of deeper retinal layers owing to dye pooling and leakage.[1] OCTA can provide retinal and choroidal microvasculature in a non-invasive manner. In addition, collateral vessel formation can be easily detected with OCTA in contrast to FA.

This case demonstrated retinal microvasculature changes and collateral formation within specific layers, as visualized using OCTA.[2,4] In our case, the mild enlargement of the FAZ and decrease in perfusion correlated with a decrease in visual acuity. In fact, collateral vessels develop as a corrective response to bypass occluded veins, with a predilection for the deep capillary plexus. Vessel formation is associated with the extent of ischemia (non-perfused area) and the amount of congested venous blood.[4] Collateral vessels develop from preexisting capillary networks. In this manner, it reduces venous pressure and allows perfusion. They are generally located around the FAZ and temporal watershed areas.[5] However, the presence of these vessels often correlates with worse anatomical outcomes, such as persistent macular edema and greater central macular thickness (CMT). Venous collaterals appear as large, well-defined vessels that cross the intermediate and deep layers. These are compensatory and should be distinguished from retinal neovascularization, which appears more irregular and tuft-like. These collaterals form a bypass for venous blood flow to reroute around the occlusion, reducing venous congestion and alleviating ischemic damage. Their presence suggests chronic BRVO, and they generally do not require treatment unless associated with complications, such as macular edema.[2,5]

Previous studies have emphasized that collateral vessels do not leak. However, Suzuki et al.[2] demonstrated that leakage may occur in microaneurysms associated with the collateral vessels. Additionally, Suzuki et al.[2] suggested that collateral vessels may influence the recurrence and persistence of macular edema owing to the presence of collateral-associated microaneurysms. Arrigo et al.[1] suggested that eyes affected by BRVO are more likely to develop OCTA-detectable collateral vessels when macular ischemia is more severe. The treatment choice for BRVO-related macular edema is the intravitreal injection of antivascular endothelial growth factor (anti-VEGF). Leakage related to collateral-associated microaneurysms is refractory to anti-VEGF therapy. Suzuki et al.[2] stated that targeting leaky microaneurysms is an effective treatment option and can significantly reduce macular edema. Therefore, personalized diagnostic and treatment approaches are required. This case emphasizes the prognostic aspects of OCTA parameters, including vessel density, FAZ size, and the presence of collateral vessels. RVO-associated macular edema may be refractory to treatment. In such cases, OCTA may provide valuable diagnostic and prognostic information regarding the microvascular system.

CONCLUSION

In conclusion, this case demonstrates the potential contribution of OCTA in the analysis of patients with BRVO. These results have important implications for understanding disease severity and visual prognosis, including enhanced collateral vessel formation, vascular density changes, and altered FAZ size. Hence, the BRVO study should incorporate advanced imaging into standard care to improve patient outcomes and individualized treatment options.

ETHICAL DECLARATIONS

Informed Consent

The patient signed and free and informed consent form.

Referee Evaluation Process

Externally peer-reviewed.

Conflict of Interest Statement

The authors have no conflicts of interest to declare.

Financial Disclosure

The authors declared that this study has received no financial support.

Author Contributions

All of the authors declare that they have all participated in the design, execution, and analysis of the paper, and that they have approved the final version.

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