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Left Eye Superotemporal Branch Retinal Vein Occlusion With Cystoid Macular Edema Associated With Hyperhomocysteinemia In A Young Adult: A Case Report

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Abstract
Purpose: *Report a young adult with branch retinal vein occlusion associated with hyperhomocysteinemia.*
Methods: *Case report of a 35-year-old male with clinical examination, OCT, laboratory evaluation, treatment, and follow-up.*
Results: *Left superotemporal BRVO with cystoid macular edema (CMT 528 µm) and elevated homocysteine (37.04 µmol/L). Following intravitreal ranibizumab, OCT showed resolution of cystoid edema and CMT decreased to 352 µm.*
Conclusions: *Young patients with BRVO should be evaluated for thrombophilia, including hyperhomocysteinemia. Early anti-VEGF therapy provides favorable anatomical outcomes.*
Keywords: *Branch retinal vein occlusion; BRVO; Hyperhomocysteinemia; Cystoid macular edema; Ranibizumab; Young adult.*
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I. Introduction

Retinal vein occlusion (RVO) is the second most common retinal vascular disorder after diabetic retinopathy and an important cause of visual morbidity worldwide.[1] Depending on the location of the obstruction, RVO is classified as central retinal vein occlusion (CRVO), hemi-retinal vein occlusion (HRVO), or branch retinal vein occlusion (BRVO). BRVO occurs due to occlusion of a branch retinal vein, most commonly at an arteriovenous crossing, resulting in retinal hemorrhages, ischemia, and macular edema.[3,4]

BRVO predominantly affects older individuals and is strongly associated with systemic vascular disorders such as hypertension, diabetes mellitus, and hyperlipidemia.[2,3] However, its occurrence in younger patients should prompt investigation for less common risk factors, including inherited or acquired thrombophilic conditions. We report a case of superotemporal BRVO with cystoid macular edema in a young adult male with hyperhomocysteinemia.

II. Case Presentation

A 35-year-old Hindu male from Bihar presented to the ophthalmology outpatient department on 14 September 2024 with complaints of sudden-onset, progressive, painless diminution of vision in the left eye for 15 days.

There was no history of floaters, flashes, ocular trauma, transient visual obscurations, glaucoma, or previous ocular disease. No significant systemic illness, medication use, or family history suggestive of thromboembolic disease was elicited.

General physical examination and systemic evaluation were unremarkable.

Best-corrected visual acuity (BCVA) was 6/6 in the right eye and 6/60 in the left eye. Anterior segment examination was normal in both eyes.

Fundus examination of the right eye was unremarkable. Dilated fundus examination of the left eye revealed multiple intraretinal hemorrhages in the superotemporal quadrant extending towards the fovea (Figure 1) . Alteration of the arteriolar wall reflex was noted in the involved quadrant. The affected venule and arteriovenous crossing could not be visualized clearly because of extensive hemorrhages. Scattered hard exudates were present in the foveal and parafoveal regions. Slit-lamp biomicroscopy with a 90D lens demonstrated blunting of the foveal reflex with associated macular thickening.

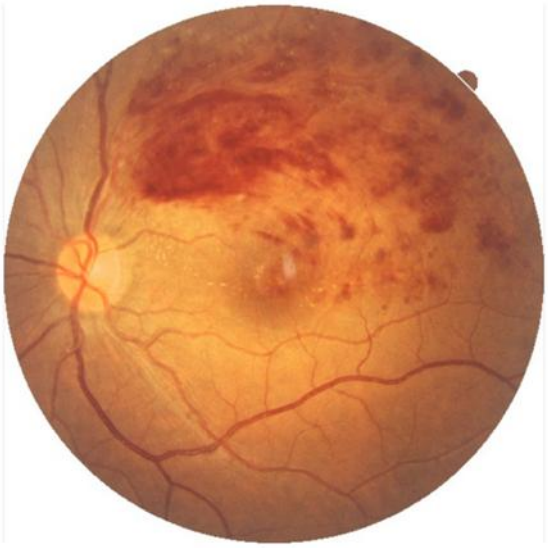

Figure 1. Color fundus photograph of the left eye at presentation

Optical coherence tomography (OCT) revealed loss of normal foveal contour with multiple intraretinal cystoid spaces involving the inner retinal layers (Figure 2). Central macular thickness measured 528 µm. Hyperreflective lesions with posterior shadowing were observed in the outer plexiform layer, corresponding to hard exudates.

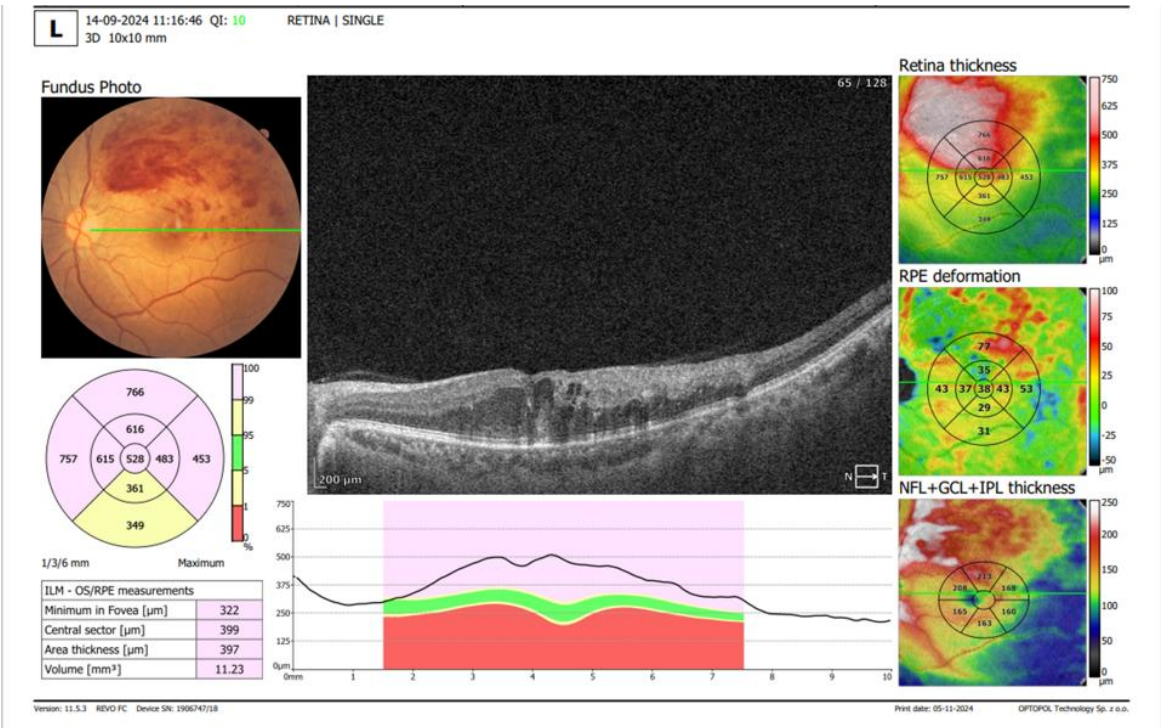

Figure 2. Spectral-domain optical coherence tomography of the left eye at presentation

Laboratory investigations including complete blood count, liver function tests, kidney function tests, lipid profile, and vital parameters were within normal limits. Serum homocysteine level was elevated at 37.04 µmol/L.

Based on the clinical findings and investigations, a diagnosis of left eye superotemporal BRVO with cystoid macular edema associated with hyperhomocysteinemia was established.

The patient received an intravitreal injection of ranibizumab (Razumab®) 0.5 mg/0.05 mL under topical anesthesia on 17 September 2024.

At one-month follow-up, fundus examination demonstrated significant reduction in intraretinal hemorrhages with residual hard exudates near the fovea (Figure 3).

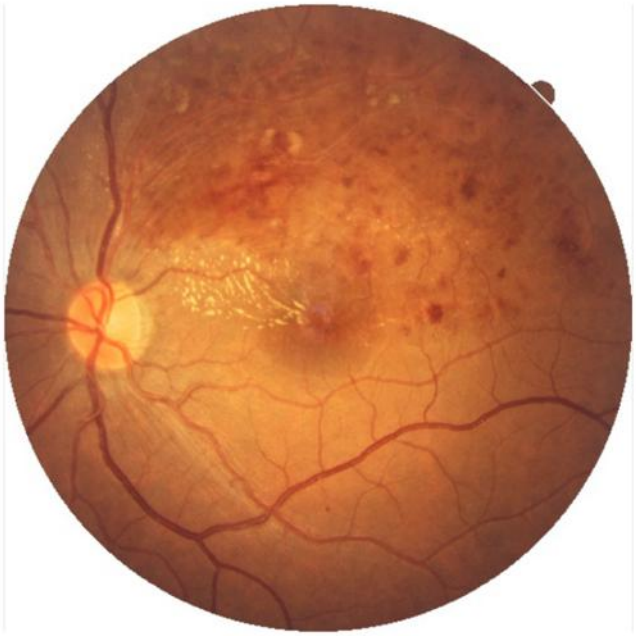

Figure 3. Color fundus photograph one month after intravitreal ranibizumab injection

OCT showed restoration of the normal foveal contour with complete resolution of intraretinal cystoid spaces and reduction in central macular thickness to 352 µm (Figure 4).

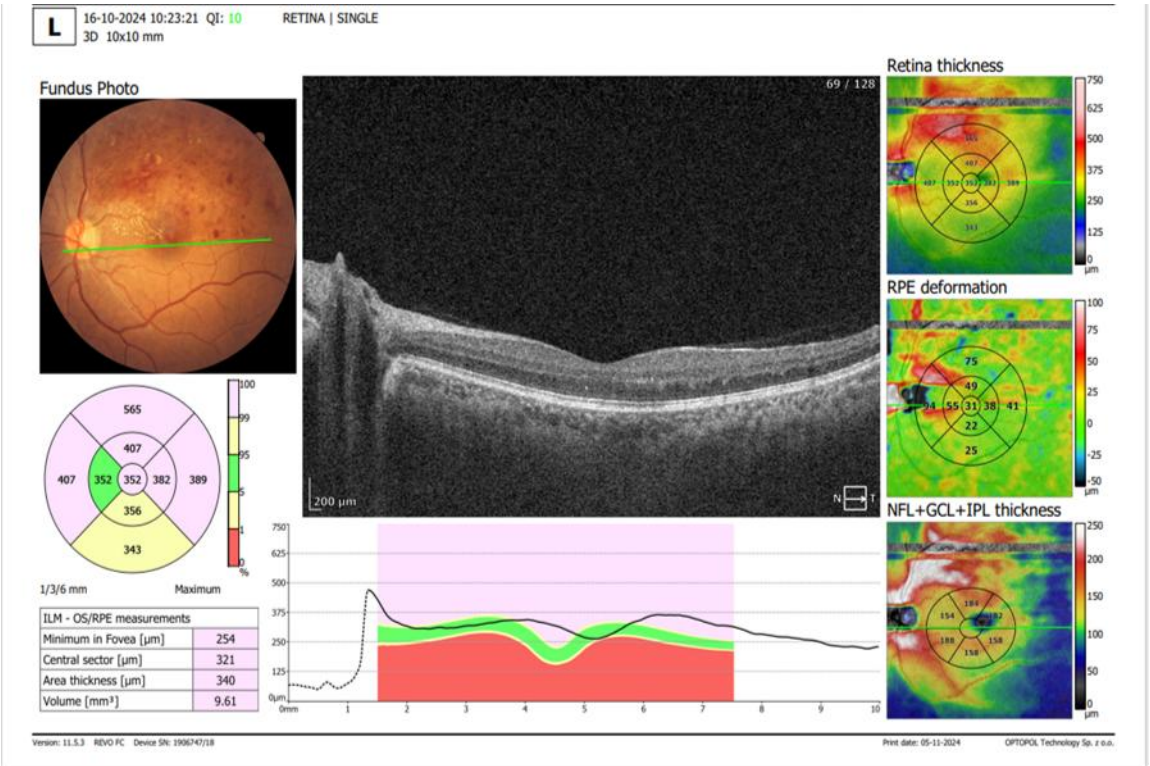

Figure 4. Optical coherence tomography one month after treatment

III. Discussion

BRVO is commonly encountered in patients older than 60 years and is frequently associated with systemic vascular risk factors including hypertension, diabetes mellitus, and dyslipidemia.[2,3] In younger patients, however, retinal vein occlusion is uncommon and often necessitates evaluation for underlying thrombophilic disorders.[5,6]

Hyperhomocysteinemia has been identified as an independent risk factor for both arterial and venous thrombotic events. Elevated homocysteine levels can promote endothelial dysfunction, oxidative stress, platelet activation, and a prothrombotic state, thereby predisposing to retinal vascular occlusive disease.[5,10]

Several studies have demonstrated an association between hyperhomocysteinemia and retinal vein occlusions, particularly in younger patients without conventional cardiovascular risk factors.[5,6] In the present case, the absence of hypertension, diabetes, hyperlipidemia, or other systemic disorders, together with significantly elevated serum homocysteine levels, suggests a contributory role of hyperhomocysteinemia in the development of BRVO.

Macular edema remains the principal cause of visual loss in BRVO.[4] Anti-vascular endothelial growth factor (anti-VEGF) therapy is currently considered the standard of care for BRVO-associated macular edema. The BRAVO study demonstrated significant visual and anatomical improvement in patients treated with intravitreal ranibizumab.[8,9]

Consistent with these findings, our patient exhibited marked anatomical improvement after a single intravitreal ranibizumab injection, with complete resolution of cystoid spaces and substantial reduction in central macular thickness.

This case highlights the importance of thrombophilia screening in young patients presenting with retinal vein occlusion, especially in the absence of traditional cardiovascular risk factors.

IV. Conclusion

Retinal vein occlusion in young adults without conventional systemic vascular risk factors should prompt investigation for thrombophilic disorders, particularly hyperhomocysteinemia. Early identification of the underlying etiology and timely administration of anti-VEGF therapy can result in favorable anatomical and visual outcomes. A thrombophilia profile should be considered in selected young patients presenting with BRVO.

Acknowledgments

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References

[1]. Rogers S, Mcintosh RL, Cheung N, Lim L, Wang JJ, Mitchell P, Et Al. The Prevalence Of Retinal Vein Occlusion: Pooled Data From Population Studies From The United States, Europe, Asia, And Australia. Ophthalmology. 2010;117(2):313-9.E1.

[2]. Hayreh SS, Zimmerman MB. Branch Retinal Vein Occlusion: Natural History Of Visual Outcome. JAMA Ophthalmol. 2014;132(1):13-22.

[3]. Jaulim A, Ahmed B, Khanam T, Chatziralli IP. Branch Retinal Vein Occlusion: Epidemiology, Pathogenesis, Risk Factors, Clinical Features, Diagnosis, And Complications. Retina. 2013;33(5):901-10.

[4]. Rehak J, Rehak M. Branch Retinal Vein Occlusion: Pathogenesis, Visual Prognosis, And Treatment Modalities. Curr Eye Res. 2008;33(2):111-31.

[5]. Cahill M, Karabatzaki M, Meleady R, Refsum H, Ueland PM, Shields D, Et Al. Raised Plasma Homocysteine As A Risk Factor For Retinal Vascular Occlusive Disease. Br J Ophthalmol. 2000;84(2):154-7.

[6]. Glueck CJ, Wang P, Bell H, Rangaraj V, Goldenberg N. Associations Of Thrombophilia And Retinal Vascular Occlusion. Clin Appl Thromb Hemost. 2005;11(4):375-83.

[7]. Lahey JM, Tunc M, Kearney J, Modlinski B, Koo H, Johnson RN, Et Al. Laboratory Evaluation Of Hypercoagulable States In Patients With Central Retinal Vein Occlusion Who Are Less Than 56 Years Of Age. Ophthalmology. 2002;109(1):126-31.

[8]. Brown DM, Campochiaro PA, Singh RP, Li Z, Gray S, Saroj N, Et Al. Ranibizumab For Macular Edema Following Branch Retinal Vein Occlusion: Six-Month Primary Endpoint Results Of A Phase III Study. Ophthalmology. 2010;117(6):1102-12.E1.

[9]. Campochiaro PA, Heier JS, Feiner L, Gray S, Saroj N, Rundle AC, Et Al. Ranibizumab For Macular Edema Following Branch Retinal Vein Occlusion: Twelve-Month Outcomes Of A Phase III Study. Ophthalmology. 2011;118(8):1594-602.

[10]. Schmidt D, Fitzgerald KC. Hyperhomocysteinemia And Retinal Vascular Occlusive Disease. Clin Ophthalmol. 2010;4:1325-32.