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Review Article

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A TALE OF TWO VEINS: BILATERAL CENTRAL RETINAL VEIN OCCLUSION WITH ASYNCHRONUS ONSET – A RARE CLINICAL ENCOUNTER

Bijoyata Reang, Ojus Kalia*, Sejal Katoch, Surinder Kumar Sharma and Vishakha Dhatwalia

India.

***Corresponding Author: Ojus Kalia**

India.

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ABSTRACT

Retinal vein occlusion (RVO) is a common cause of vision loss in older individuals, and the second most common retinal vascular disease after diabetic retinopathy.[1] It is of 2 distinct types, in central RVO (CRVO), occlusion is at or proximal to lamina cribrosa of optic nerve, where central retinal vein exits eye.[2] CRVO is further divided into perfused (non-ischemic) and non-perfused (ischemic).[3] Bilateral involvement is rare and usually associated with systemic vascular or hematologic disorders. We report a unique case of bilateral CRVO presenting with asynchronous onset – one eye exhibiting sequelae of an old occlusion, and the other with acute findings. This report highlights the importance of systemic evaluation, vigilant follow-up, and individualized management in bilateral, time-lagged presentations.

INTRODUCTION

CRVO typically presents unilaterally and is associated with systemic conditions such as uncontrolled hypertension, diabetes mellitus, and hypercoagulable states. It is believed to follow the principles of Virchow's triad for thrombogenesis, involving vessel damage, stasis, and hypercoagulability.[4] It results from thrombosis of the central retinal vein, leading to impaired venous drainage, retinal haemorrhages, edema, and potential vision loss. Bilateral CRVO is rare, reported in <1% of cases, and suggests underlying systemic pathology.

It warrants immediate and thorough systemic evaluation due to its association with life-threatening conditions, including antiphospholipid antibody syndrome, multiple myeloma, and hyper viscosity syndromes. Clinically, it poses diagnostic and therapeutic challenges due to the compounded visual impairment and complex systemic involvement.

This presentation highlights a rare case of bilateral CRVO, emphasizing the importance of early recognition, appropriate systemic workup, and a multidisciplinary approach in management to prevent further visual and systemic complications.

CASE HISTORY and EXAMINATION

A 73 year-old otherwise healthy male presented to the outpatient department with complaints of sudden, painless diminution of vision in his right eye from 8 days. The patient reported a history of reduced vision in his left eye for 1 year.

Initially 16 months back, patient was diagnosed to have neovascular glaucoma in his left eye with vein occlusion sequalae with cystoid macular edema and vitreo-macular traction for which he had intravitreal injection and three sitting of laser were done, prior to this he had cataract surgery in his left eye 1 month back from previous hospital. But then 8 days back, the patient developed sudden, painless diminution of vision in his right eye and visited the OPD. After thorough examination and history taking, patient was found to have central retinal vein occlusion in his right eye. Patient was already on pressure lowering medications (combination of Brimonidine and Timolol) in view of neovascular glaucoma left eye and raised intra ocular pressure in right eye.

General Examination

The patient was otherwise vitally stable with rise in blood pressure 144/86 mmHg. There was no other systemic involvement.

Ocular Examination

In the right eye, best corrected visual acuity (BCVA) was 5/60. There was immature senile cataract in the anterior segment and no relative afferent pupillary defect. Intraocular pressure (IOP) was 20 mmHg on NCT. On fundus examination, optic disc is clear with well-defined margins, normal size and colour. There are dilated and tortuous retinal veins, radiating symmetrically from the disc. Linear flame-shaped and dot-blot haemorrhages were seen in all four quadrants, few cotton wool spots, splinter haemorrhages around the disc giving a "tomato-splash" appearance and a dull foveal reflex – consistent with acute CRVO.

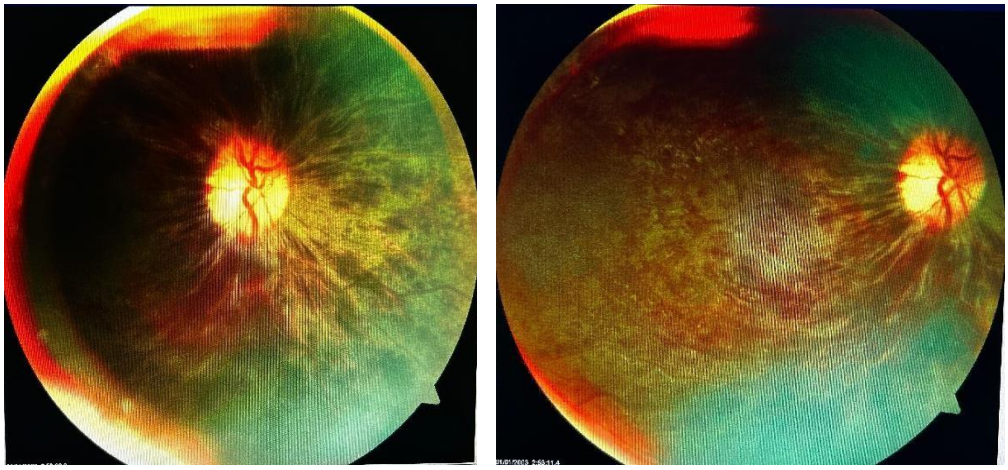

Fig. 1 (a, b): Showing Fundus Finding Of Right Eye With Radiating Flame Shaped Haemorrhages – S/O Acute Crvo.

In the left eye, visual acuity was counting finger at 3 metres. There were no changes in anterior segment, no NVI or NVA. Left eye was pseudophakic. Intraocular pressure on NCT was 16 mmHg. On fundus examination, optic disc appears pale and elevated with peripapillary gliosis and vessels extending from disc. The retinal vessels appear tortuous with venous congestion and arteriolar attenuation (sclerosed and narrowed arteries) s/o long-standing ischaemia. Foveal reflex is dull; no gross exudates or haemorrhages are seen. On the periphery, there is visible sheathing of vessels inferiorly, with areas of pigmentary changes possibly marks of laser photocoagulation. All leads to sequelae of old CRVO.

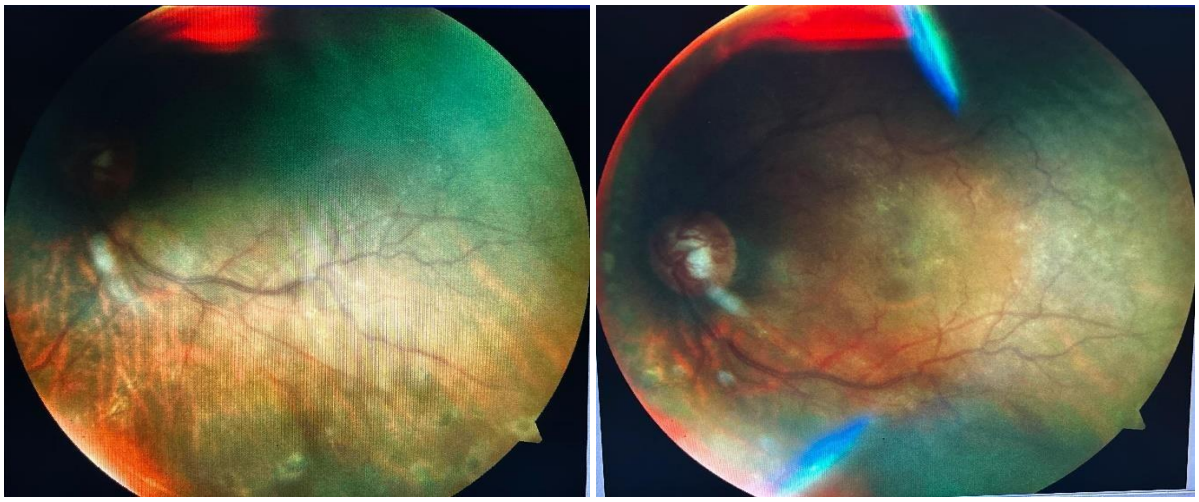

Fig. 2 (a,b): showing Fundus finding of left eye with pale disc, laser spots infero- temporally and sclerosed vessels -s/o Old CRVO.

TREATMENT HISTORY

Patient was earlier put on oral Acetazolamide 250mg thrice daily for 3 days when there was rise in intraocular pressure of 36mmHg on right eye and 24mmHg on left eye. He is instilling topical IOP lowering drugs, a combination of Brimonidine and Timolol. He had intravitreal injection and pan retinal photocoagulation done in his left eye and also had history of pars plana vitrectomy with ERM peeling and ILM peeling. Currently, for active CRVO in right eye, intravitreal Injection Ranibizumab was injected.

INVESTIGATION

Laboratory investigations revealed:
BP: 154/ 88 mmHg
Fasting blood sugar: 148 mg/dl HbA1c: 7.2%
Lipid profile: Elevated LDL ESR, CRP: Mildly raised
Coagulation profile: Elevated homocysteine levels (>50.00 umol/L) ANA, RF, APLA: Negative

The patient was diagnosed with bilateral CRVO secondary to hypertensive and metabolic vasculopathy, with probable contribution from hyper-homocysteinemia.

On OCT, both right and left eye showed significant macular edema.

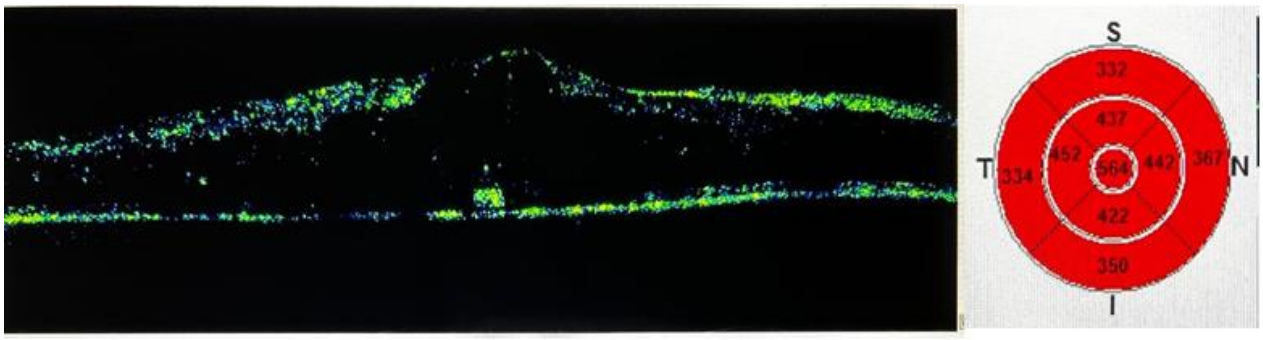

Fig. 3: OCT of right eye focusing on macula showing accumulation of fluid intra-retinal – s/o Cystoid macular edema with central thickness 564 micron.

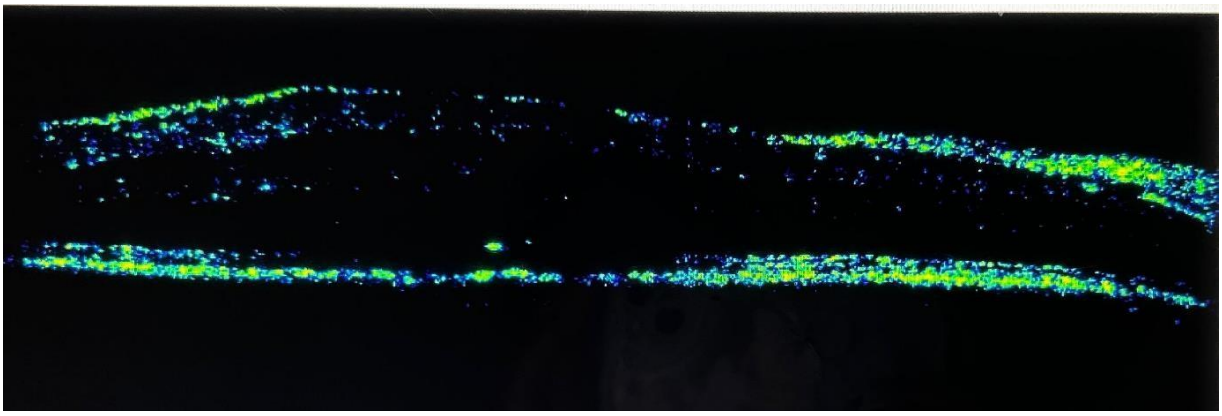

Fig. 4: OCT of Left Eye Showing Mild Fluid Accumulation Intra-Retinal and Exudate.

MANAGEMENT

In addition to ocular treatment, systemic management with folic acid and B-complex for homocysteine control were advised and referred to internist and cardiologist for systemic optimization.

DISCUSSION

Bilateral CRVO is rare and often indicates a systemic cause. The presence of different stages in each eye suggests a progressive vascular insult. In our case, the delayed diagnosis in the left eye underscores the importance of early ophthalmologic evaluation in unilateral vision loss. Hyper-homocysteinemia, although often overlooked, is an important modifiable risk factor and must be addressed.[5]

CONCLUSION

This case emphasizes the need for a high index of suspicion and systemic evaluation in cases of bilateral CRVO. Asynchronous onset should prompt investigations for vascular and hematologic abnormalities. Early intervention can preserve vision in the second eye, even if the first has progressed to atrophic stages.

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