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Case Report

Central Retinal Vein Occlusion Associated with Mesangial IgA Glomerulonephritis: A Case Report

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Abstract

Purpose: To describe a rare association between mesangial IgA glomerulonephritis and Central Retinal Vein Occlusion (CRVO) in a young patient.

Methods: Clinical, imaging and laboratory findings of a 28-year-old man with biopsy-proven IgA nephropathy who presented with acute visual loss were reviewed.

Results: Fundus examination revealed widespread flame-shaped hemorrhages, while optical coherence tomography confirmed severe cystoid macular edema (central macular thickness 487 μm). Fluorescein angiography demonstrated a non-ischemic CRVO pattern without significant capillary non-perfusion. A comprehensive thrombophilic work-up was unremarkable. The patient received intravenous corticosteroids followed by aspirin, with complete anatomical and near-complete visual recovery. Two years later, recurrence of CRVO coincided with worsening renal function; fluorescein angiography identified ischemic features with extensive peripheral non-perfusion exceeding 10 disc areas. A single intravitreal bevacizumab injection was administered prior to kidney transplantation, after which renal function stabilized and no further ocular relapses occurred. The temporal association between renal deterioration and ocular relapse supports a shared pathophysiological mechanism involving immune-mediated endothelial dysfunction, complement activation and systemic hypercoagulability.

Conclusion: Central retinal vein occlusion may represent an ocular manifestation of systemic vascular injury secondary to immune-mediated glomerulopathies such as IgA nephropathy. Recognition of this association is essential to ensure multidisciplinary evaluation and prevent recurrence through early systemic control.

Keywords: Central Retinal Vein Occlusion; Hypercoagulability; Retinal Vein Occlusion; Iga Nephropathy

Introduction

Hemostasis maintains the balance between coagulation and fibrinolysis. When this balance shifts toward coagulation, a state of hypercoagulability ensues, predisposing to venous or arterial thrombosis in different territories. Retinal Vein Occlusions (RVOs) have specific local determinants such as intraocular pressure, vascular architecture and limited fibrinolytic efficacy while systemic risk factors include arterial hypertension, diabetic retinopathy, hyperlipidemia, glaucoma, smoking and atherosclerosis. In young adults without overt vascular comorbidities, Central Retinal Vein Occlusion (CRVO) should prompt investigation for systemic causes such as autoimmune disease, inherited thrombophilia or immune-mediated glomerulopathies, including IgA nephropathy.

Case Report

A 28-year-old man presented with a 24-hour history of sudden visual loss in the Right Eye (RE). Baseline visual acuity was 0.2 in the RE and 1.0 in the Left Eye (LE). Anterior segment examination was unremarkable. Fundus examination of the RE revealed diffuse flame-shaped hemorrhages involving the posterior pole, consistent with CRVO (Fig. 1). Optical Coherence Tomography (OCT) confirmed severe cystoid macular edema, with a Central Macular Thickness (CMT) of 487 μm. Fluorescein angiography demonstrated delayed venous filling without significant areas of capillary non-perfusion, consistent with a non-ischemic CRVO pattern. The patient was admitted for interdisciplinary evaluation by Internal Medicine and Nephrology.

Past medical history included biopsy-proven mesangial IgA glomerulonephritis diagnosed two years earlier, with 2% proteinuria and well-controlled mild hypertension. On admission, nephrotic syndrome had worsened (proteinuria 89 mg/dL, creatinine 2.72 mg/dL) with persistent hematuria.

A comprehensive thrombophilic work-up was performed, including protein C, protein S, antithrombin III, factor V Leiden mutation, prothrombin G20210A mutation, lupus anticoagulant, anticardiolipin and anti-β2-glycoprotein I antibodies, serum homocysteine and Antinuclear Antibodies (ANA and ANCA). All results were within normal limits, supporting IgA nephropathy as the primary systemic condition predisposing to CRVO in the absence of an identifiable primary thrombophilic disorder.

During hospitalization, he received three days of intravenous methylprednisolone followed by long-term aspirin. This approach was preferred over primary intravitreal therapy given its dual benefit: suppression of the underlying immune-mediated nephropathy and simultaneous control of macular edema. The favorable anatomical response complete resolution of macular edema (CMT 198 μm) and hemorrhages with a final visual acuity of 0.9 in the RE at six months (Fig. 1) confirmed the appropriateness of this strategy and precluded the need for intravitreal intervention at that stage.

Two years later, a recurrence of CRVO in the same eye reduced visual acuity to 0.1. Fundus examination and OCT showed extensive hemorrhages and diffuse macular edema with a CMT of 628μm (Fig. 2). Fluorescein angiography demonstrated extensive areas of peripheral capillary non-perfusion exceeding 10 disc areas, reclassifying this episode as ischemic CRVO. Renal function had markedly deteriorated (urea 191 mg/dL, creatinine 5 mg/dL) and the patient was listed for kidney transplantation. Given the concurrent systemic deterioration, a single intravitreal injection of bevacizumab (1.25 mg/0.05 mL) was administered to address the ischemic macular edema while renal replacement therapy was being arranged. Post-transplant, renal function stabilized with no further ocular relapses. At the last follow-up, 24 months after kidney transplantation, visual acuity was 0.3 in the RE and 1.0 in the LE without angiographic activity (Fig. 3). Renal biopsy immunofluorescence demonstrated granular mesangial IgA deposition, confirming active IgA nephropathy (Fig. 4).

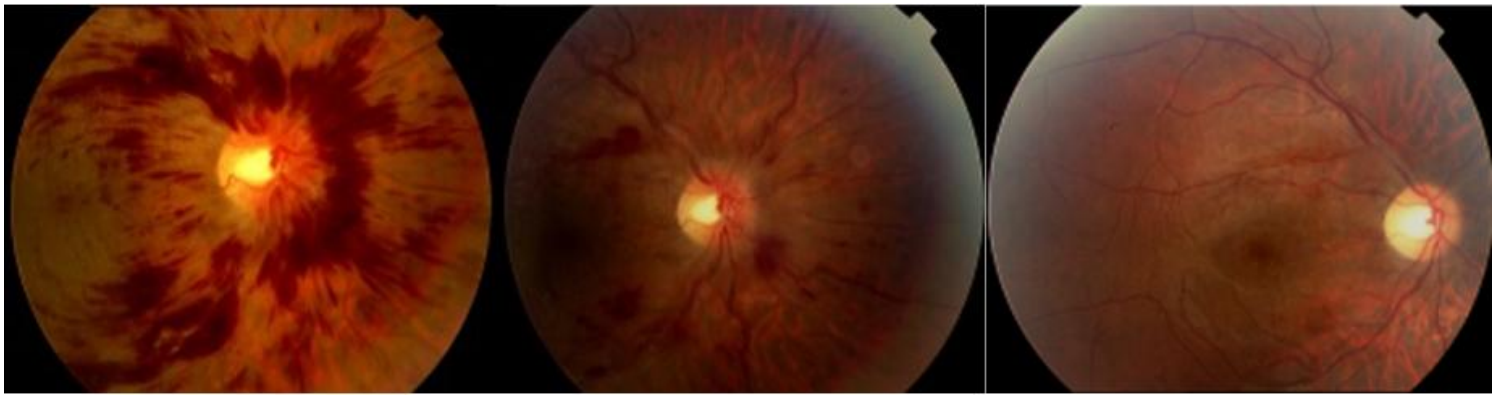

Figure 1: Fundus photographs of the right eye (RE). (A) Baseline image showing diffuse flame-shaped hemorrhages involving all four quadrants of the posterior pole, consistent with Central Retinal Vein Occlusion (CRVO). Fluorescein angiography at presentation demonstrated delayed venous transit without significant areas of capillary non-perfusion, consistent with non-ischemic CRVO; OCT confirmed cystoid macular edema with a central macular thickness (CMT) of 487 μm; (B) Three-month follow-up showing partial resolution of hemorrhages with improvement in macular edema; (C) Six-month follow-up demonstrating complete resolution of hemorrhages and macular edema (CMT 198 μm), with visual acuity recovering to 0.9.

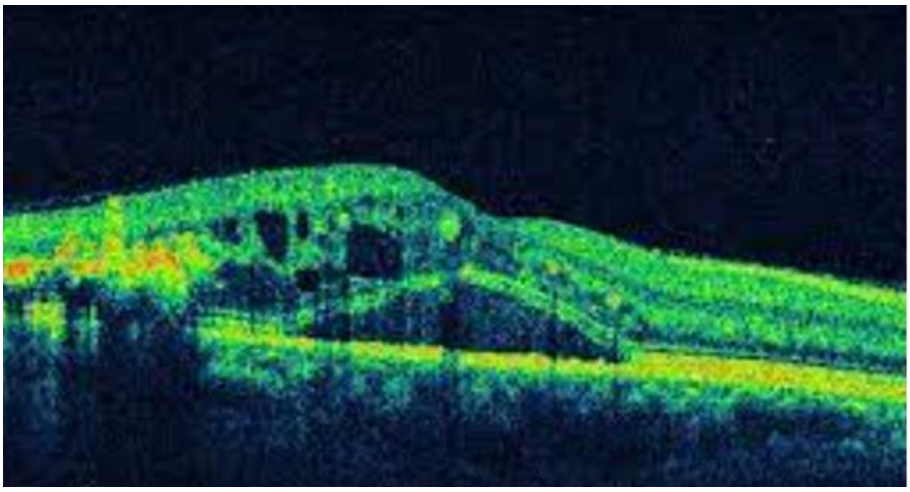

Figure 2: Recurrence of CRVO in the right eye two years later. Fundus photography demonstrates extensive retinal hemorrhages involving all four quadrants. OCT reveals diffuse cystoid macular edema (CMT 628μm). Fluorescein angiography shows extensive areas of peripheral capillary non-perfusion exceeding 10 disc areas, consistent with ischemic CRVO.

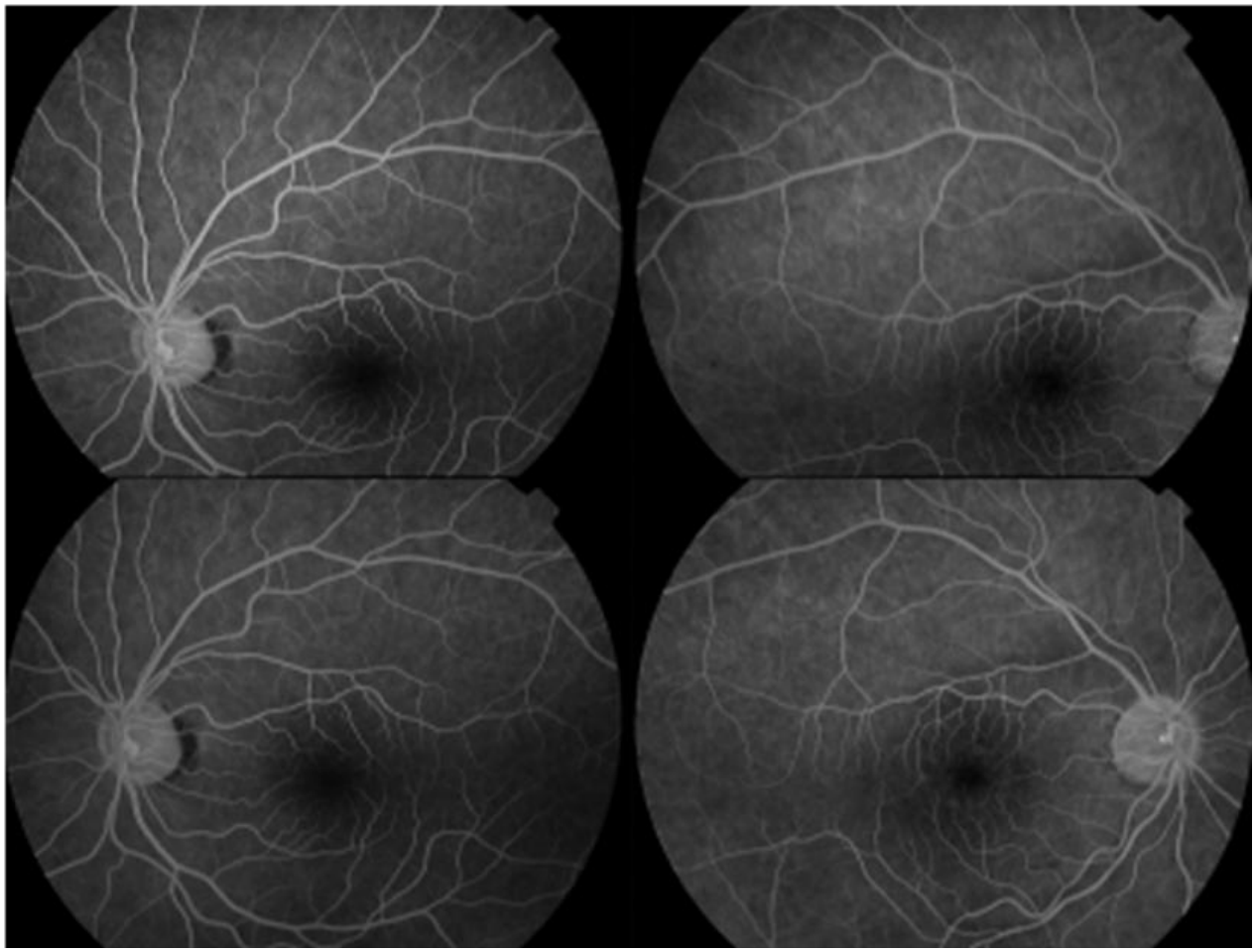

Figure 3: Final follow-up images 24 months after kidney transplantation. Fundus photography shows resolution of hemorrhages with an atrophic appearance of the optic disc consistent with prior vascular injury. OCT demonstrates preserved macular architecture without intraretinal fluid (CMT within normal limits). Fluorescein angiography reveals no angiographic activity. Visual acuity was 0.3 in the RE and 1.0 in the LE.

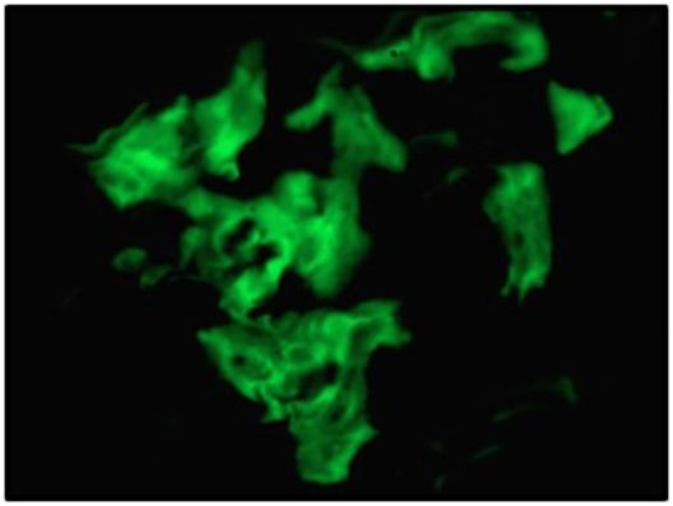

Figure 4: Renal biopsy immunofluorescence demonstrating granular mesangial IgA deposition, confirming active IgA nephropathy (Berger disease).

Literature Review and Discussion

Pathophysiology

IgA nephropathy (Berger disease) is the most common primary glomerulonephritis worldwide, characterized by mesangial deposition of aberrantly galactosylated IgA1, formation of circulating immune complexes and complement activation via the alternative and lectin pathways, driving mesangial inflammation, cytokine release and diffuse endothelial dysfunction [1]. The prothrombotic milieu generated by this condition is multifactorial: urinary loss of antithrombin III and proteins C and S reduces natural anticoagulant defenses; elevated plasma fibrinogen, factors V and VIII and increased blood viscosity promote coagulation; augmented von Willebrand factor and endothelial adhesion molecules (ICAM-1, VCAM-1) facilitate platelet recruitment; platelet activation and procoagulant microvesicles amplify the thrombotic signal; and reduced local fibrinolysis impairs clot resolution. In nephrotic states, depletion of anticoagulant proteins further promotes thrombotic events in both renal and retinal circulations, rendering the retinal venous system particularly vulnerable given its limited fibrinolytic reserve [1].

Literature Review

The association between IgA nephropathy and retinal vascular occlusions remains uncommon but clinically relevant, particularly in young patients without classic vascular risk factors. Table 1 summarizes the principal reported cases. Miguel, et al., described bilateral retinal arterial occlusive vasculitis secondary to IgA nephropathy with favorable response to immunomodulation [2]. Mack, et al., reviewed retinal findings across glomerulonephritides, underscoring complement activation and endothelial damage as shared mechanisms [3]. El Matri, et al., reported bilateral ischemic retinal vasculopathy linked to IgA nephropathy using widefield angiography and OCT-A, suggesting diffuse immune microvascular involvement [4]. Population-based data show that end-stage renal disease confers a significantly increased risk of retinal vein occlusion, which decreases though not to baseline after kidney transplantation and earlier nationwide data indicated approximately a threefold higher risk versus the general population [5,6].

Authors (Year)	Journal	Ocular Finding	Imaging	Renal Status	Treatment	Visual Outcome
Miguel, et al.,	Retina Cases Brief Rep	Bilateral retinal arterial occlusive vasculitis	FA	Active IgA nephropathy	Immunomodulation	Favorable
Mack, et al.,	Kidney Int Rep	Review: retinal findings in GN; complement activation	Fundus photo	Multiple GN subtypes	Varies	Mechanism review

Authors (Year)	Journal	Ocular Finding	Imaging	Renal Status	Treatment	Visual Outcome
El Matri, et al.,	BMC Ophthalmol	Bilateral ischemic retinal vasculopathy	Widefield FA, OCT-A	Active IgA nephropathy	Not specified	Partial recoger
Lee, et al.,	Sci Rep	RVO risk in ESRD	Nationwide cohort	End-stage renal disease	Kidney transplant	Reduced risk post-Tx
Chang, et al.,	Medicine (Baltimore)	RVO risk ×3 vs. general population	Nationwide cohort	End-stage renal disease	—	Elevated baseline risk
Present case	—	Recurrent CRVO (non-ischemic → ischemic)	Fundus, OCT, FA	Mesangial IgA nephropathy	IV corticosteroids; bevacizumab IVT; renal transplant	VA 0.3 RE at 24 months

FA: Fluorescein Angiography; GN: Glomerulonephritis; CRVO: Central Retinal Vein Occlusion; OCT: Optical Coherence Tomography; OCT-A: Optical Coherence Tomography Angiography; IVT: Intravitreal; VA: Visual Acuity; RE: Right Eye; Tx: Transplantation.

Table 1: Summary of previously reported ocular vascular events associated with IgA nephropathy.

Differential Diagnosis

In young adults presenting with CRVO without conventional vascular risk factors, a systematic approach to etiological diagnosis is essential. The principal differential diagnoses include inherited thrombophilia (deficiencies of protein C, protein S or antithrombin III; factor V Leiden or prothrombin G20210A mutations), antiphospholipid syndrome, hyperviscosity disorders (paraproteinemia, polycythemia vera), systemic vasculitis (Behçet disease, systemic lupus erythematosus) and hematological malignancies. Medications including oral contraceptives and immunosuppressants and local ocular factors (elevated intraocular pressure, optic disc drusen) should also be considered. Systemic and metabolic comorbidities are key risk factors for CRVO and their systematic exclusion is essential for appropriate attribution [7]. In the present case, the complete thrombophilic work-up was unremarkable and no alternative systemic cause was identified, strengthening the causal inference linking CRVO to the underlying IgA nephropathy.

Clinical Implications

Multimodal ophthalmic imaging is fundamental in CRVO associated with systemic disease. Fluorescein angiography allows classification of ischemic versus non-ischemic status which determines prognosis and guides intravitreal treatment decisions while OCT-A enables noninvasive quantification of macular perfusion and ischemic index. In our patient, the evolution from non-ischemic CRVO (first episode, no significant non-perfusion, CMT 487 μm) to ischemic CRVO (recurrence, >10 disc areas of non-perfusion, CMT 628μm) paralleled the progressive deterioration of renal function, supporting the concept of cumulative immune-mediated vascular injury. Partial anatomical recovery after kidney transplantation suggests that at least part of the retinal vascular damage is driven by reversible systemic immune mechanisms. Recommended work-up in young adults with CRVO includes: (1) detailed history (prior thrombosis, autoimmune disease, prothrombotic drugs); (2) fluorescein angiography and OCT-A to quantify ischemia and detect subclinical vasculitis; (3) comprehensive laboratory evaluation encompassing complete blood count, lipid profile, glucose, protein electrophoresis, autoimmunity screening (ANA, anticardiolipin, antiphospholipid antibodies) and thrombophilia testing (protein C, protein S, antithrombin III, homocysteine, factor V Leiden, prothrombin G20210A mutation) and (4) in unclear cases, consideration of an immunosuppressive or anti-inflammatory trial. Management should be multidisciplinary, combining optimal blood pressure and renal control with targeted ocular therapy anti-VEGF agents or intravitreal corticosteroids for macular edema in close coordination with Nephrology [7,8].

Conclusion

The association between central retinal vein occlusion and mesangial IgA glomerulonephritis, though uncommon, carries clinically meaningful implications. In young patients with CRVO lacking classic vascular risk factors and an unremarkable

thrombophilic work-up, underlying immune-mediated renal disease should be considered. Complement-driven endothelial dysfunction and systemic hypercoagulability may represent shared pathophysiological mechanisms linking both conditions. A multidisciplinary approach encompassing systemic disease control and targeted ocular therapy is essential to prevent recurrences and optimize visual outcomes.

Conflict of Interest
The authors declared no potential conflicts of interest with respect to the research, authorship and/or publication of this article.

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Data Availability Statement
The data supporting the findings of this study are available from the corresponding author upon reasonable request.

Ethical Statement
The project did not meet the definition of human subject research under the purview of the IRB according to federal regulations and therefore was exempt.

Informed Consent Statement
Informed consent was obtained from all participants included in the study.

Authors' Contributions
All authors contributed equally to this paper.

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