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Review

Revisiting Retinal Vein Occlusion: An Etiologic Classification for Enhanced Patient Management

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ABSTRACT: Retinal vein occlusion (RVO) represents one of the most prevalent retinal vascular disorders and continuously stands as a leading cause of visual impairment worldwide. Conventional classification systems for RVO—predominantly featured by anatomical site as central, hemi-retinal, or branch retinal vein occlusion—often fail to reflect the underlying etiologic factors exerting crucial influence on individualized patient management. In an effort to mitigate this gap, we propose an etiologic classification that divides retinal venous stasis disorders—used here to describe a pathophysiological continuum of impaired retinal venous outflow, ranging from early venous congestion to retinal vein occlusion—into four distinct groups: (1) intrinsic retinal venous pathology, (2) local venous compression at arteriovenous crossing points, (3) venous congestion secondary to translaminar pressure gradient abnormality, and (4) venous outflow obstruction correlated with cavernous sinus pathology. To introduce and illustrate this new classification, we conducted a narrative literature review and compiled retrospectively identified illustrative clinical cases. As underscored by our findings, differing underlying etiologies necessitate distinct diagnostic evaluations and tailored therapeutic interventions in spite of similar clinical fundus presentations. For example, RVO cases stemming from systemic inflammatory diseases require targeted immunologic management, whereas those associated with elevated intracranial pressure demand neurological assessment and appropriate intervention. This etiologically oriented approach complements traditional anatomical classifications by promoting comprehensive systemic evaluations and personalized treatment strategies. We propose that implementing this etiologic classification could not only contribute to ameliorated clinical outcomes but also encourage its further evaluation for potential integration into routine ophthalmological practice.

Keywords: Retinal vein occlusion, retinal venous stasis disorders, intracranial hypertension, retinal vasculitis, carotid–cavernous fistula.

INTRODUCTION

Retinal vein occlusion (RVO) encompasses a heterogeneous group of disorders and is the second most common retinal vascular disease after diabetic retinopathy [1-3]. Typically, attributable to a combination of systemic and local factors, it occurs when venous outflow from the retina is partially or completely obstructed. This impaired drainage brings about venous dilation, retinal edema and hemorrhage [4-6]. As of 2015, RVO affected an estimated

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0.77% of individuals aged 30 to 89 years globally, corresponding to approximately 28.06 million cases [7]. The incidence increases with age, with cumulative five-year and ten-year incident rates of 0.86% and 1.63%, respectively [8-12].

Conventionally, RVO is classified in line with the anatomical location of the occlusion: branch retinal vein occlusion (BRVO), hemi-retinal vein occlusion (HRVO) and central retinal vein occlusion (CRVO) [13]. CRVO refers to a blockage at or just behind the optic nerve head [14], HRVO involves one of the major venous trunks [13], and BRVO affects a peripheral branch vein [15]. Apart from topographic classification, RVO is also clinically categorized into ischemic and non-ischemic types, grounded in the degree of retinal nonperfusion observed on fundus fluorescein angiography (FFA) [16]. Ischemic RVO is characterized by extensive capillary dropout, the presence of a relative afferent pupillary defect (RAPD), and a substantially higher risk of neovascular complications [17]. In contrast, non-ischemic RVO typically exhibits limited retinal ischemia, absence of RAPD, and a more favorable visual prognosis [18]. While anatomical and ischemic classifications offer important clinical value, they often provide limited guidance for etiologic evaluation and individualized management. For example, two patients presenting with clinically similar CRVO may have fundamentally different pathogenic triggers—one resulting from a systemic hypercoagulable state, and the other stemming from localized mechanical compression from an adjacent sclerotic artery. Nonetheless, both cases are categorized identically under the current framework.

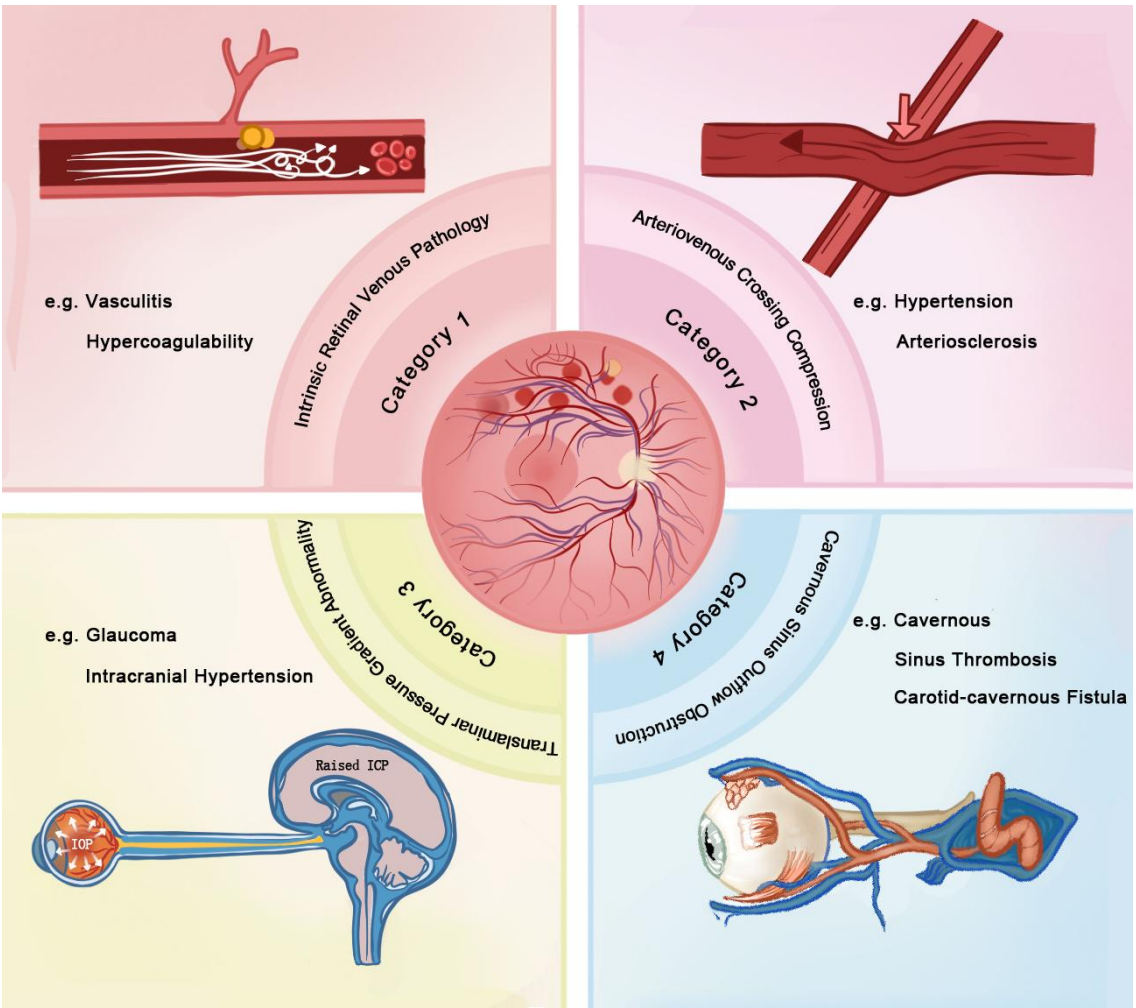

Figure 1. Etiology-based classification of retinal venous stasis disorders. Retinal venous outflow disorders can be categorized into four major types: Category 1 involves intrinsic venous abnormalities such as vasculitis and hypercoagulability; Category 2 is caused by arteriovenous compression, commonly due to hypertension or arteriosclerosis; Category 3 results from disturbances in the translaminar pressure gradient, as seen in glaucoma or intracranial hypertension; and Category 4 reflects post-retinal outflow obstruction, including cavernous sinus thrombosis and carotid-cavernous fistula.

Recognizing the diverse etiologies that can give rise to RVO, we propose a reclassification grounded in the primary mechanism of impaired venous outflow. In this review, we use the term "retinal venous stasis disorders" as an umbrella concept for conditions involving impaired retinal venous outflow, ranging from early venous congestion to retinal vein occlusion. This approach is inspired by classification systems used in cerebral venous disorders, which categorize conditions on the basis of mechanisms such as thrombosis [19], impaired venous outflow [20], or idiopathic intracranial hypertension [21]. By focusing on the pathophysiological drivers rather than fundoscopic anatomy, this model encourages clinicians to broaden the diagnostic work-up to incorporate systemic, structural, and neurological contributors. This paradigm supports more individualized investigations, encompassing thrombophilia screening or neuroimaging, when clinically appropriate.

Specifically, we propose an etiologic classification system comprising four major mechanistic categories (Fig. 1). As shown in Figure 1, retinal venous stasis disorders are conceptualized as arising from distinct sources along the venous drainage pathway: Category 1, intrinsic retinal venous pathology (e.g., thrombosis, vasculitis/periphlebitis, hypercoagulability); Category 2, focal mechanical compression at arteriovenous crossing sites; Category 3, impaired venous outflow secondary to an abnormal translaminar pressure gradient at the optic nerve head (e.g., glaucoma or elevated intracranial pressure); and Category 4, post-retinal outflow obstruction involving the cavernous sinus/retro-orbital venous system (e.g., cavernous sinus thrombosis or carotid–cavernous fistula).

This framework is intended to complement traditional anatomic classification by providing a structure for etiologic reasoning. We suggest that adopting an etiologic model may support more accurate etiologic evaluation and inform more targeted therapeutic strategies for patients with retinal venous stasis disorders. Here, we outline the four etiologic categories, illustrate them with representative cases, and compare this cause-based framework with conventional anatomy-based classifications to show how etiologic reasoning may guide a more tailored work-up and management in RVO.

METHODS

Study Design and Methodological Framework

This work presents a conceptual narrative review, supplemented by illustrative clinical cases, with the aim of proposing an etiologic, mechanism-oriented classification for retinal venous stasis disorders. The review focuses on integrating existing pathophysiological knowledge rather than on quantitative evidence synthesis or hypothesis testing. The included cases are intended for illustrative purposes to demonstrate the clinical application of the proposed framework, rather than for formal statistical validation. Cases were assigned according to the dominant mechanism outlined in Figure 1, with overlapping contributors noted when applicable.

Narrative Literature Review

We conducted a narrative literature review to establish the conceptual basis for our proposed etiologic classification of RVO and related venous stasis disorders. Relevant literature was identified through targeted searches of PubMed/MEDLINE, Web of Science, and Google Scholar through December 2025, supplemented by manual screening of reference lists and authoritative ophthalmology and neurology textbooks. Search terms included combinations of "retinal vein occlusion," "venous stasis," "etiology," "pathophysiology," "arteriovenous crossing," "arteriovenous compression," "arteriolosclerosis/atherosclerosis," "hypertension," "hypercoagulable," "vasculitis," "inflammation," "translaminar pressure gradient," "intracranial hypertension," and "cavernous sinus." Articles were screened for mechanistic and clinical relevance; duplicates were removed and non-English publications were excluded. Discrepancies in study selection were resolved by author consensus. The selected evidence was synthesized thematically to support the rationale and structure of the four proposed etiologic categories.

Illustrative Clinical Cases

To complement the literature-based conceptual framework, we retrospectively identified representative clinical cases to exemplify the four proposed etiologic categories of retinal venous stasis disorders. Cases were selected based on: (1) a definitive diagnosis of retinal vein stasis disorders; (2) clinical, laboratory, or imaging evidence supporting a predominant mechanism consistent with a specific category; and (3) availability of complete clinical documentation and multimodal imaging. Etiological categorization was determined by consensus of two senior retinal specialists following review of multimodal ocular imaging and relevant systemic evaluation. These cases are presented for illustrative purposes to demonstrate the clinical application of the proposed framework.

Data Collection

For each illustrative case, relevant clinical information was summarized descriptively, including patient demographics, presenting symptoms, and ophthalmologic examination findings (best-corrected visual acuity, intraocular pressure, and fundus examination), as well as multimodal retinal imaging, including fundus photography, FFA, and optical coherence tomography (OCT). Ancillary systemic evaluations and neuroimaging findings were included when available and clinically relevant. Management strategies and clinical courses were summarized descriptively.

Ethical Considerations

The study complied with the principles of the Declaration of Helsinki and received approval from the Ethics Committee of Xuanwu Hospital, Capital Medical University (Approval No. 2022101). Written informed consent was obtained from all patients for the use of anonymized clinical and imaging data for research and publication. All data were fully de-identified to protect patient confidentiality.

Etiologic Classification of Retinal Venous Stasis Disorders

Grounded in the underlying mechanism of venous outflow impairment, retinal venous stasis disorders may be stratified into four principal etiologic subtypes. While each subtype may overlap with traditional anatomical classifications, they are primarily differentiated by their distinct pathophysiological mechanisms.

In the following section, we summarize the pathophysiology, clinical features and key diagnostic or therapeutic considerations associated with each etiological group. This framework lays a solid foundation for an all-round comprehension of the representative case reports, each illustrating one of the recommended subtypes.

Category 1: Intrinsic Retinal Venous Disease

Definition and Mechanism

Intrinsic retinal venous disease refers to etiologies of RVO that arise from within the retinal vein itself or result from systemic conditions that alter the intravascular environment. The primary pathogenic mechanisms involve intrinsic abnormalities of the blood, such as hypercoagulability, or of the vessel wall, such as vasculitis, rather than external compression by surrounding anatomical structures. In this context, RVO is driven by the classical elements of Virchow's triad—venous stasis, endothelial injury, and hypercoagulability—rather than by mechanical obstruction. Accordingly, this category encompasses both thrombotic occlusion, which principally encompasses intraluminal clot formation, and inflammatory occlusion, thereby stemming from immune-mediated damage to retinal veins.

Thrombotic Causes

Thrombophilic Disorders

Both inherited and acquired thrombophilic conditions are well-established contributors to the pathogenesis of RVO, particularly in younger patients or those without traditional cardiovascular risk factors. Among the inherited forms, the Factor V Leiden mutation, which confers resistance to activated protein C, has gained the most profound attention both domestically and internationally. Meta-analyses have demonstrated a modestly increased risk of RVO in individuals carrying this mutation, with odds ratios ranging from 1.49 to 1.66 [22, 23]. Other hereditary thrombophilic disorders have also been extensively investigated, encompassing protein C deficiency, protein S deficiency, antithrombin deficiency, and the prothrombin G20210A mutation [24]. Hyperhomocysteinemia is another non-negligible risk factor implicated in RVO. It is universally acknowledged that heightened plasma homocysteine levels elevate thrombotic risk by triggering endothelial toxicity, oxidative stress, and boosting platelet aggregation. Multiple meta-analyses have confirmed a statistically significant association between hyperhomocysteinemia and RVO [22, 25-27] .

Antiphospholipid antibody syndrome (APS) is a major acquired thrombophilic disorder characterized by the presence of lupus anticoagulant, anticardiolipin antibodies, and/or anti-β2 glycoprotein I antibodies, along with clinical manifestations such as venous or arterial thrombosis. A recent meta-analysis demonstrated a remarkably higher prevalence of APS in RVO patients in contrast to control groups, which thereby highlights its pivotal role in retinal vascular thrombosis [28, 29]. Given the systemic implications and increased risk of recurrent thromboembolic events, APS warrants long-term anticoagulation with vitamin K antagonists once diagnosed.

Hyperviscosity Syndromes and Hematologic Abnormalities

Systemic disorders that elevate blood viscosity can hinder retinal venous outflow and encourage thrombus formation, particularly under low-shear or slow-flow conditions. Notably, polycythemia vera, essential thrombocythemia, and Waldenström's macro-

globulinemia are tightly correlated with elevated hematocrit or serum immunoglobulin levels [30-33] .

The resultant hyperviscosity facilitates erythrocyte aggregation and reduces laminar flow within the retinal circulation, which is especially vulnerable due to its low perfusion pressure and absence of collateral drainage. In some cases, RVO may even represent the initial clinical manifestation of an occult hematologic malignancy or paraproteinemia [33, 34].

A comprehensive diagnostic workup is recommended in patients subject to atypical presentations—such as bilateral RVO, absence of atherosclerotic risk factors, or systemic symptoms. This evaluation should encompass complete blood count, peripheral blood smear, serum protein electrophoresis, and serum viscosity testing when clinically indicated.

Inflammatory Causes

Retinal venous occlusion may also result from vascular inflammation associated with systemic inflammatory diseases, comprising Behçet's disease [35], systemic lupus erythematosus [36], and sarcoidosis [37], or from idiopathic retinal periphlebitis[38-40]. These cases usually necessitate both systemic and local ocular therapy.

A term occasionally applied to mild CRVO in young patients is "retinal disc vasculitis", considered a possible inflammatory variant of CRVO. These patients typically present with optic disc edema and venous congestion but retain relatively preserved visual function and tend to have a benign clinical course [41-43]. This phenomenon is normally considered to mirror an inflammatory-thrombotic process involving the central retinal vein.

Clinical Features

Patients subject to intrinsic retinal venous disease are typically younger than 50 years and often lack conventional cardiovascular risk factors [44]. The condition may be bilateral or recurrent. Atypical presentations—such as CRVO in younger individuals—should prompt evaluation for hypercoagulable states or systemic vasculitis. For this reason, it is essential to conduct a full laboratory workup, which encompasses coagulation studies, autoimmune panels, and inflammatory markers.

Management Implications

Other than standard treatments for RVO (e.g., intravitreal anti-VEGF injections, laser photocoagulation for non-perfusion areas or macular edema), it also holds paramount implications to throw light upon and cope well with the underlying systemic cause. Typically, under hematologic guidance and systemic anticoagulation, antiplatelet therapy, or plasmapheresis may be indispensable for thrombotic events in severe cases. More importantly, systemic corticosteroids or steroid-sparing immunosuppressants may be indicated for inflammatory etiologies, and it is often beneficial to establish a collaboration with a rheumatologist or internist.

Prognosis

Visual outcomes are highly variable in intrinsic retinal venous disease. Prognosis is generally favorable with prompt diagnosis and treatment of the systemic condition and limited retinal ischemia. Nonetheless, delays in managing systemic factors can give rise to recurrence or involvement of the contralateral eye. Long-term follow-up is advised, especially in patients suffering from systemic diseases that may affect multiple organ systems, comprising patients' eyes. The following case illustrates an example of intrinsic retinal venous disease secondary to systemic inflammation.

Case 1: RVO on account of Systemic Inflammation

A 39-year-old woman presented with a 6-month history of progressive bilateral visual loss and floaters. Best-corrected visual acuity (BCVA)was 20/50 OD and 20/63 OS. IOP and anterior segment examination were within normal limits. No hemorrhages were observed on fundus exam. FFA disclosed mid-peripheral capillary telangiectasia and late leakage in the right and left eyes, respectively. OCT was unremarkable (Fig. 2). She had a past medical history of SLE, antiphospholipid syndrome, Sjogren's, and Hashimoto's disease, with positive immunological markers encompassing anti-Ro-52, anti-SS-B, anticardiolipin IgG, and augmented anti-dsDNA antibodies.

Given the presence of inflammation and the capability of hypercoagulability to affect venous outflow without occlusive phenomenon, the diagnosis was classified on the basis of an immune-mediated periphlebitis. In the aftermath of the rheumatology consultation, she received IV methylprednisolone followed by oral prednisone and hydroxychloroquine. Bilateral PRP was performed. Vision remained stable with no neovascularization or hemorrhage.

Category 2: Retinal Vein Compression from Sclerotic Artery

Definition and Mechanism

This category describes RVO resulting from mechanical obstruction of the vein by neighboring arteries, usually at arteriovenous crossings (AVCs) [45]. Retinal arteries may

stiffen and thicken with time associated with systemic disorders like arteriolosclerosis and hypertension [46]. When an arteriosclerotic artery is lying across the vein it is likely to press upon, occlude the flow of blood through it, and trigger thrombosis at the point of pressure. This phenomenon is classically involved in BRVO: small veins that traverse beneath arteries are less severely affected than those that cross over arteries [47]. In other instances, this compressive force may conduct a pivotal role in hemi-CRVO or CRVO, as the central retinal vein passes through the tight anatomic confines of the lamina cribrosa and sheath of the optic nerve. To be more specific, this force may be compressed by neighboring arterial structures, especially in the setting of atherosclerosis or conditions such as glaucoma [48].

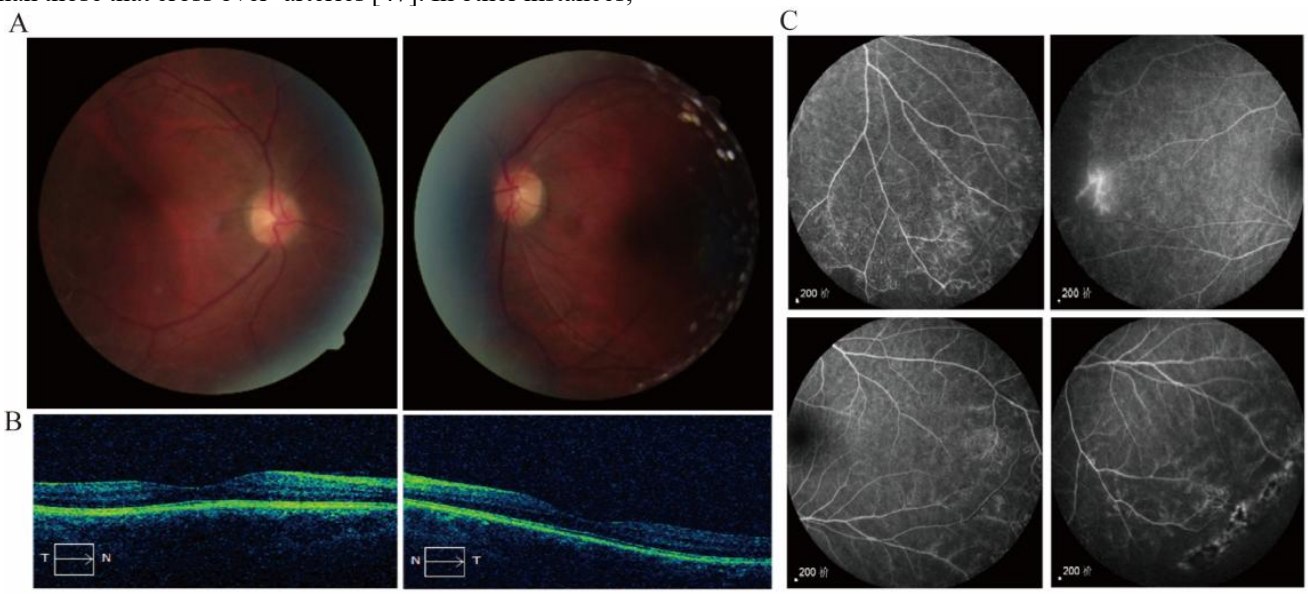

Figure 2. Case 1 – RVO on account of Systemic Inflammation. (A) Color fundus photographs show bilateral venous engorgement without retinal hemorrhage. **(B)** OCT reveals preserved macular architecture in both eyes. **(C)** FFA demonstrates mid-peripheral capillary telangiectasia with late-phase vascular leakage in both eyes.

Epidemiology and Risk Factors

RVO arising from this underlying reason is most frequently presented as BRVO and typically affects patients aged over 50 who have systemic vascular risk factors [49]. The superotemporal quadrant of retina is the commonly involved locus, which potentially can be attributable to the fact that the AVCs are more common in this area. The most powerful and consistent risk factor for BRVO is hypertension, which accelerates dramatic changes in the arterial wall and predisposes to compression at crossing sites. Other major risk factors comprise diabetes, hyperlipidemia and smoking, all giving rise to atherosclerosis. Local ocular factors, encompassing optic disc drusen and glaucoma can also be implicated in the case of compression-related CRVO [50].

Pathophysiology

At AVCs, artery and vein have a common adventitial sheath. In BRVO, the artery presses on the underlying vein (often after crossing over it) bringing about mechanical compression of the vein, which causes venous luminal narrowing, reduced axial flow velocity and turbulent flow. This altered hemodynamics increases shear stress on the endothelium with endothelial injury, cellular proliferation, and wall remodeling with venous obstruction. The result is a venous obstruction with retinal bleeding in the area supplied by post-thrombosis [51]. A similar compressive scenario may take place in HRVO or CRVO in the close quarters of the lamina cribrosa, particularly in the presence of atheromatous vessels or predisposing anatomical factors such as crowded discs or glaucoma [52].

Clinical Presentation

Patients usually complain the sudden onset of painless decreased visual acuity or scotomas. Fundoscopy in BRVO illustrates flame-shaped hemorrhages, macular edema, dilatation and tortuosity of veins, exudates, and cotton-wool spots that are localized to the quadrant of abnormality[53]. In compression type CRVO, such changes are diffusely observed in all quadrants. As a consequence of arterial disease, RVO is complicated by more severe retinal ischemia and larger areas of nonperfusion. From a clinical standpoint, this disorder is likely attributable to a pre-existing susceptibility of the

retinal perfusion system since the abrupt increase in venous outflow resistance following the occlusion may tip the balance from compensated to ischemic marginal perfusion, with universal capillary dropout [1].

Diagnosis

Fundus examination emerges as the predominant methodology for precise clinical diagnosis. FFA not only demonstrates delay in the filling of the involved vein, but also suggests non-perfused capillary areas in ischemic presentations. Relative afferent pupillary defect may be present. OCT is mandatory to look for macular edema, which is the most prevalent complication [54]. Beyond conventional imaging, emerging evidence suggests that structural and vessel-related features at arteriovenous crossings may serve as early indicators of compression-related BRVO. Using multimodal deep learning applied to pre-onset fundus hemisection images can predict the future development of BRVO, supporting a mechanism-based interpretation of venous compression before clinical occlusion becomes evident [55]. A thorough and systematic assessment should be conducted to detect and monitor cardiovascular risk factors, including blood pressure, blood glucose levels, lipid profiles, and arterial health. Testing for thrombophilia is typically not recommended unless the patient is very young or has bilaterally affected eyes that may indicate other causes. Several studies have suggested that compression-related RVO may serve as a predictor of future cerebrovascular events [51, 56].

Management

Ocular management for compression-induced BRVO or CRVO revolved around macular edema and ischemia. Intravitreal injection of anti-VEGF therapy is considered as the first-line treatment for decreasing macular edema and improving visual acuity. For persistent edema, grid or focal laser photocoagulation can be used as the second-line or adjuvant therapy. In patients suffering from widespread retinal ischemia by FA, panretinal photocoagulation (PRP) is the treatment of choice to avoid complications like vitreous hemorrhage, or neovascular glaucoma. Altogether, treatment lowering intraocular pressure should be initiated upon the presence of glaucoma or intraocular hypertension.

Systemic Risk Factor Control

Equally crucial is the management of systemic vascular risk factors. Tight control of hypertension, diabetes, and lipid profile is useful for preventing recurrence of RVO or involvement of the contralateral eye. In such case, it is strongly recommended to probe deep into modifiable lifestyle factors comprising smoking cessation and regular physical activity. Consultation with internal medicine, cardiology, or neurology may be indicated to provide systemic vascular evaluation and therapy.

Prognosis

A significant proportion of BRVO patients will regain at least some visual function, particularly if macular edema is treated expeditiously. Prognosis is grounded in macular involvement and degree of ischemia. Ischemic CRVO illustrates a less satisfactory visual prediction and a more significant chance of developing complications such as neovascular glaucoma. Despite the fact that this new etiologic classification does not change clinical practice as it currently stands for ocular therapies, it underlines the magnitude of understanding the underlying pathophysiology and reinforces the necessity of managing systemic vascular risk factor management in these patients [17, 57].

Case 2: RVO secondary to AVCs

A 52-year-old male suffering from hypertension and hypercholesterolemia presented with four days of visual field defect and decreased right eye vision. Visual acuity remained 20/50 OD and 20/40 OS. Fundus displayed dilated veins, hemorrhages and arterial compression in the inferotemporal quadrant. FA revealed leakage and nonperfusion; OCT illustrated cystoid macular edema >500 μm. Systemic workup revealed uncontrolled hypertension with 200/90mmHg and high low-density lipoprotein (LDL). There is no evidence of thrombophilia or inflammation. Glaucoma was ruled out. (Fig. 3)

He received two intravitreal ranibizumab injections, improving visual acuity to 20/40 and reducing edema. A total of five injections were given over one year with stable disease. No laser was performed.

This classic ischemic BRVO case emphasizes both local compression and systemic risk control.

Category 3: Venous Stasis Related to Translaminar Pressure Gradient Abnormality

Definition and Background

Venous stasis retinopathy resulting from translaminar pressure gradient (TPG) abnormalities represents a distinct clinical entity characterized by impaired retinal venous outflow spawned from an imbalance between intraocular pressure (IOP) and intracranial pressure (ICP). The lamina cribrosa, a sieve-like structure in the sclera through which the optic nerve fibers and central retinal

vein pass, normally maintains a delicate balance of pressures [58]. Under physiological conditions, a stable gradient between IOP anteriorly and ICP posteriorly ensures normal venous drainage and axoplasmic flow. Disruption of this pressure equilibrium as a result of either heightened ICP, such as idiopathic intracranial hypertension (IIH), cerebral venous sinus thrombosis (CVST), venous sinus stenosis (VSS), intracranial tumors, or meningitis [59] or heightened IOP (as observed in acute angle-closure glaucoma or advanced open-angle glaucoma) [60] triggers venous congestion at the optic nerve head, mimicking or contributing to RVO.

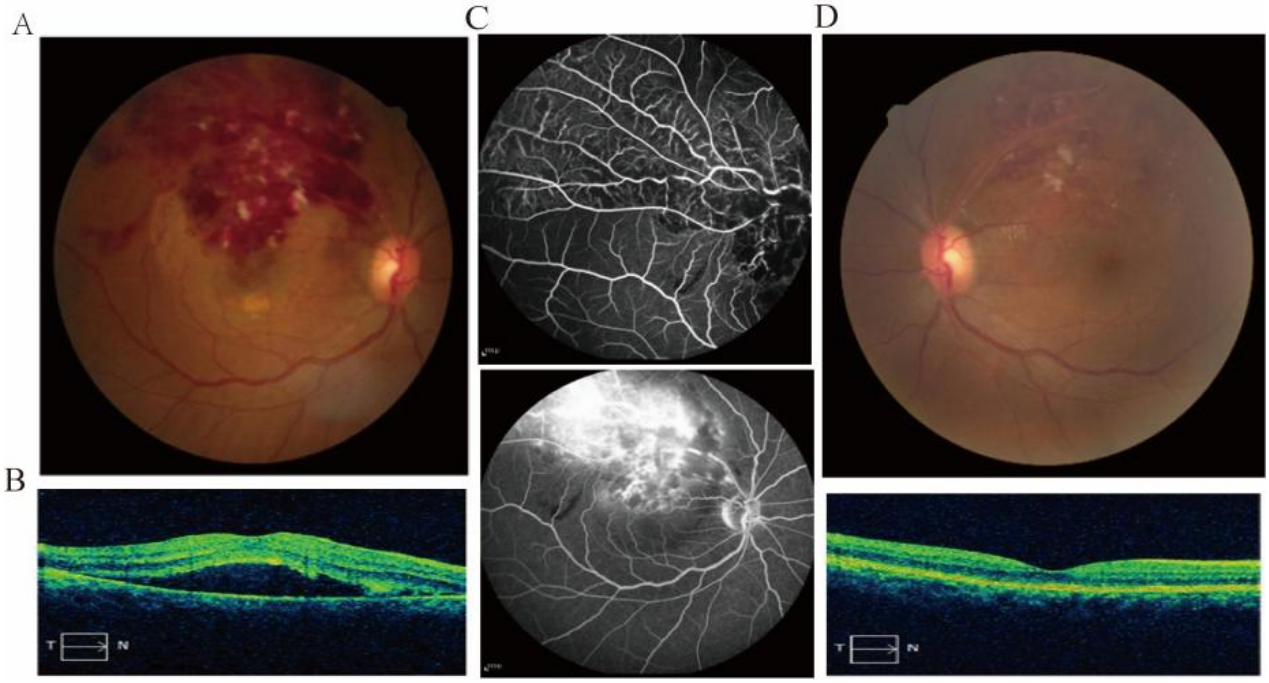

Figure 3. Case 2 – RVO secondary to AVCs. (A) Color fundus photograph of the right eye shows extensive intraretinal hemorrhages and venous dilation in the inferotemporal quadrant, with arteriovenous crossing suggestive of local compression. **(B)** OCT reveals cystoid macular edema with central retinal thickness exceeding 500 μm. **(C)** FFA demonstrates capillary nonperfusion and late-phase leakage in the corresponding area. **(D)** Fundus and OCT images at 6-month follow-up show significant resolution of hemorrhages and restoration of normal macular contour after intravitreal ranibizumab treatment.

Pathophysiology

Under normal conditions, retinal venous blood drains efficiently through the central retinal vein at the lamina cribrosa without significant resistance. Nonetheless, the pressure within the subarachnoid space surrounding the optic nerve increases with the increment of ICP, giving rise to axoplasmic flow obstruction and subsequent papilledema. The resulting edema compresses the optic nerve head, mechanically narrowing the peripapillary retinal veins, increasing venous outflow resistance and resulting in venous stasis [61].

Likewise, remarkably augmented IOP, especially in glaucomatous eyes, leads to anterior displacement and mechanical compression of the lamina cribrosa, constricting the lumen of the central pathophysiological endpoint of impaired venous drainage across the lamina cribrosa, defined as translaminar pressure gradient abnormality [62].

Clinical Features

Patients experiencing augmented ICP typically present with symptoms suggestive of raised intracranial pressure, comprising persistent headaches, transient visual obscurations, pulsatile tinnitus, diplopia, and occasionally nausea or vomiting [19]. Ophthalmoscopic examination classically reveals bilateral optic disc swelling (papilledema), peripapillary hemorrhages, and marked venous congestion. These retinal findings are frequently bilateral, symmetric, and accompanied by minimal peripheral retinal ischemia [63].

In contrast, patients suffering from heightened IOP on account of acute angle-closure glaucoma often present acutely with ocular pain, blurred vision, halos around lights, corneal edema, and heightened intraocular pressure measured via tonometry. Chronic forms, such as advanced primary open-angle glaucoma (POAG), may exhibit subtle and slowly progressive visual field defects until advanced structural optic nerve damage occurs. Retinal examination may reveal optic nerve head edema,

dilated and tortuous retinal veins, flame-shaped retinal hemorrhages, and macular edema, clinically resembling unilateral CRVO [48].

Diagnostic Evaluation

The key diagnostic objective is to distinguish true thrombotic RVO from venous stasis arising from abnormal translaminar pressure gradients. For suspected ICP elevations, initial evaluation should encompass comprehensive neuroimaging—magnetic resonance imaging (MRI) with venography (MRV)—to identify CVST, intracranial masses, or sinus stenosis. Lumbar puncture is crucial for measuring cerebrospinal fluid (CSF) opening pressure and analyzing CSF composition [64]. OCT imaging quantifies papilledema severity, while automated perimetry assesses visual field defects associated with optic nerve dysfunction [65].

For heightened IOP-related cases, ophthalmic examination with precise tonometry, gonioscopy, and anterior segment imaging is critical. Fundus photography and OCT of the optic nerve head provide detailed structural information, differentiating glaucomatous cupping from papilledema. FFA helps identify characteristic delays in venous filling or peripapillary disc leakage, usually without extensive peripheral capillary non-perfusion seen in thrombotic RVO.

Treatment and Management

The cornerstone of managing translaminar pressure gradient abnormalities involves promptly restoring equilibrium between ICP and IOP. Elevated ICP management typically starts with acetazolamide therapy to reduce CSF production, supplemented by weight loss and lifestyle modifications in idiopathic cases [66]. Anticoagulation remains essential for CVST [67], with endovascular procedures such as venous sinus stenting indicated when trans-stenotic pressure gradients exceed 8 mmHg [68]. Surgical options, encompassing cerebrospinal fluid diversion [69] (ventriculoperitoneal or lumboperitoneal shunts) or optic nerve sheath fenestration (ONSF) [70], may be warranted in vision-threatening cases refractory to medical therapy.

Elevated IOP management comprises topical and systemic ocular hypotensive medications, hyperosmotic agents, and prompt laser iridotomy or surgical interventions for angle-closure glaucoma. In general, advanced POAG necessitates sustained medical therapy or surgical interventions (e.g., trabeculectomy or glaucoma drainage devices) [71] to reach target pressures and prevent irreversible optic nerve damage. Anti-vascular endothelial growth factor (anti-VEGF) therapy generally plays a less paramount role, reserved for persistent macular edema following adequate pressure normalization.

Prognosis

With timely recognition and appropriate intervention, retinal venous stasis can suggest pronounced reversibility and visual recovery, which generally arises from translaminar pressure gradient abnormalities. Nevertheless, prolonged or untreated pressure elevations risk irreversible optic nerve atrophy, severe visual field deficits, and permanent vision loss. As a result, it is crucial to conduct early differentiation between pressure-mediated venous stasis and thrombotic retinal vein occlusion, which apparently underscores the significance of comprehensive interdisciplinary collaboration between ophthalmologists, neurologists, and glaucoma specialists in managing these complex cases.

Case 3: Venous Stasis Retinopathy and Intracranial Hypertension

A 21-year-old woman developed persistent headache, nausea, vomiting and then diplopia and acute bilateral vision loss. Fundus exam illustrated bilateral papilledema with hemorrhages and exudates. OCT confirmed optic disc swelling with RNFL thickening (133 μm OD, 143 μm OS), while the macula was preserved. Black-blood MRI revealed bilateral transverse sinus stenosis (Fig. 4); lumbar puncture revealed ICP >330 mmH_2O.

She underwent optic nerve sheath fenestration as well as venous sinus stenting. Symptomatic improvement was accompanied by follow-up findings of resolved papilledema and enhanced visual function. This case demonstrates the requirement for early ICP management to preserve vision.

Category 4: Venous Outflow obstruction in Cavernous Sinus Disease

Definition and Mechanism

The cavernous sinus is a venous plexus located at the base of the skull that receives blood from the superior and inferior ophthalmic veins, which in turn drain the retina and orbit. Pathological changes within the cavernous sinus can elevate venous pressure in the ocular circulation and result in retinal venous stasis [72]. Cavernous sinus thrombosis (CST) and carotid-cavernous fistula (CCF) are the two principal conditions in this category. Notwithstanding the fact that both can obstruct venous outflow, they demonstrate substantial differences in their pathophysiology and clinical presentation.

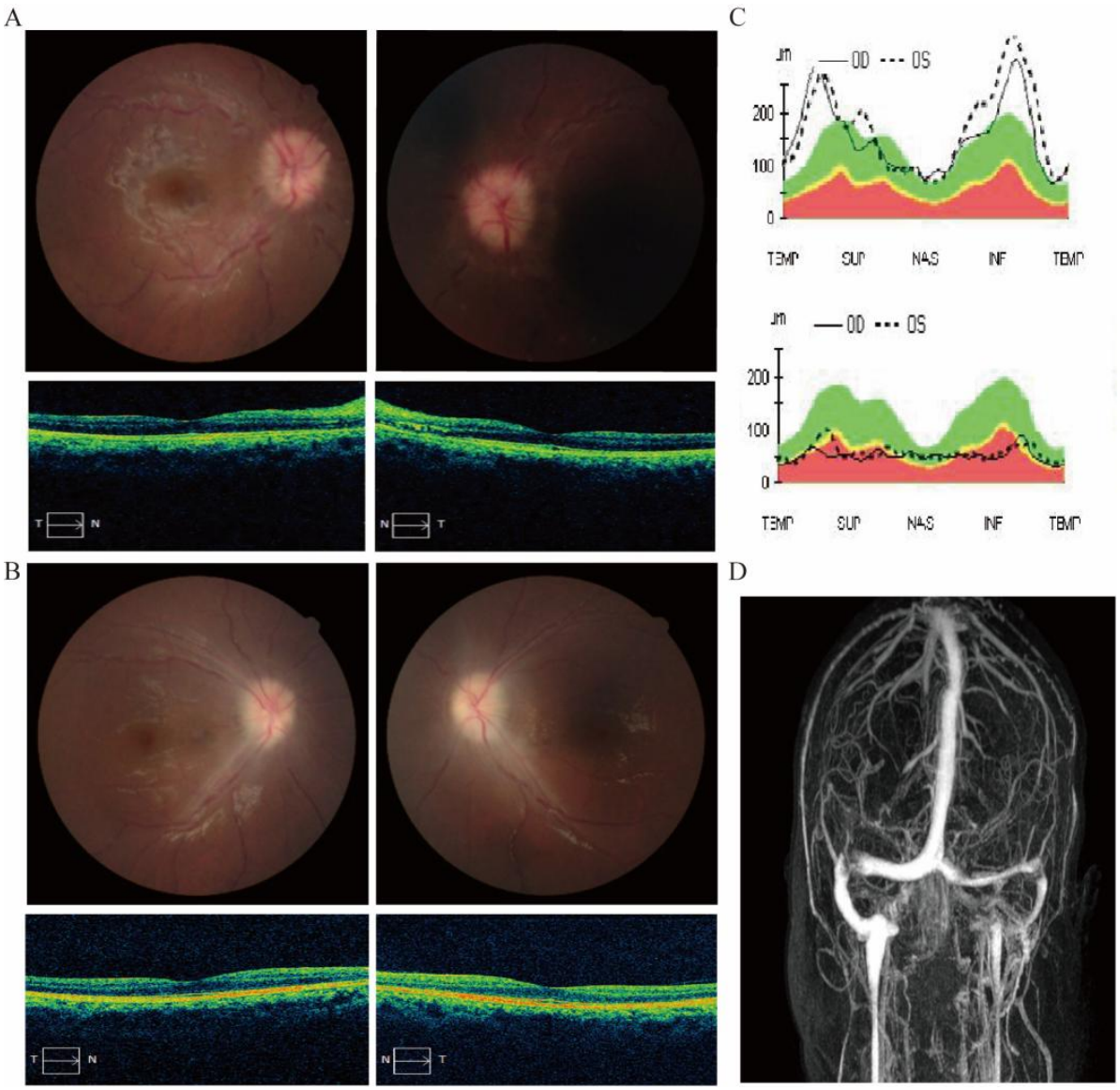

Figure 4. Case 3 – Bilateral venous stasis retinopathy induced by intracranial hypertension. (A) Color fundus photographs and OCT from initial presentation show bilateral optic disc edema with flame-shaped hemorrhages and hard exudates. **(B)** Follow-up imaging reveals resolution of papilledema and stable macular contour. **(C)** RNFL thickness plots confirm initial swelling and subsequent improvement. **(D)** Black-blood Magnetic Resonance reveals focal stenosis at the junction of the right transverse and sigmoid sinuses.

Cavernous Sinus Thrombosis (CST)

CST is a deadly disorder usually secondary to infection (e.g., the extension of untreated sinus or facial infections through venous channels) or a hypercoagulable state [73].

Clinical Presentation

Clinical presentation of CST is usually acute onset of symptoms of severe headache, which may be accompanied by a fever, orbital pain, proptosis with eyelid swelling, ophthalmoplegia secondary to cranial nerve involvement along the course of the sinuses and an altered mental status. The fundus may illustrate venous stasis along with hemorrhages that mimic CRVO on account of high venous pressure [74]. Of significance, CST is frequently bilateral (as a consequence of communication between the cavernous sinuses) and is rapidly progressive as a whole.

Management

CST is a medical emergency. In case of contagious aspect, debuted intravenous antibiotics are necessary. Anticoagulation may be employed to prevent thrombus

propagation. High-dose corticosteroids can be administered to lower inflammation. In general, intensive care is indispensable [75]. Despite the fact that retinal findings coincide with resolution of the thrombosis, priority should be given to life-saving treatment and prevention of stroke or permanent neurologic damage.

Carotid-Cavernous Fistula (CCF)

Definition and Classification

CCF is a pathological arteriovenous shunt between the internal carotid artery (ICA) and the cavernous sinus. It can be spontaneous (SCCF) and traumatic (TCCF) which comprises up to 75% of cases. CCF are typically classified by employing the Barrow classification system, which divides them into four types (A–D) rooted in the arterial supply and fistula configuration. Type A fistulas are direct connections between the internal carotid artery (ICA) and the cavernous sinus (usually traumatic), while Types B–D are indirect dural shunts involving branches of the ICA and/or external carotid artery (ECA) [76].

Clinical Features

CCF presentation strikingly hinges on arterial supply, venous drainage and the engagement of cerebral or cranial nerve structures. Blood from the ICA is diverted directly into the cavernous sinus, which contributes to the elevation of its flow and pressure. This can result in distal ICA hypoperfusion, resulting in cerebral and ophthalmic ischemia. Larger shunts bring about more severe symptoms and faster progression, while smaller ones may present gradually with milder signs if the circle of Willis is well-compensated.

Because of the extensive venous connections of the cavernous sinus, the influx of arterial blood causes dilatation and arterialization of veins. The most common drainage pattern is anterior via the superior ophthalmic vein, bringing about pulsatile proptosis, dilated episcleral and orbital veins, optic disc edema, retinal venous congestion, chemosis and cranial nerve palsies affecting ocular motility [77].

Intracranial hemorrhage may be attributable to accompanying dural arteriovenous malformations or the rupture of enlarged veins. Continuous retinal venous stasis may result in retinal hemorrhage and loss of vision. Epistaxis may be induced by nasal or nasopharyngeal venous distension.

Despite the fact that CCF differs from the primary RVO, this lesion may secondarily cause CRVO by its high venous pressure and poor ocular outflow. Differing from idiopathic RVO, secondary CRVO as a consequence of CCF generally presents with marked ocular and systemic findings [78, 79].

Diagnosis

CCF is initially suspected in line with clinical signs and features and finally established through neuroimaging. CT or MR angiography may reveal superior ophthalmic vein dilatation with abnormal flow patterns. Nevertheless, digital subtraction angiography (DSA) remains the gold standard, providing precise information on fistula location, size and hemodynamics essential for treatment planning [79].

Management

The primary treatment goals for CCF reside in vision preservation, bruit cure and proptosis, and cerebral ischemia prevention. Treatment is to secure the fistula with preservation of the patency of the ICA. Management therapy is rooted in shunt rate, source of arterial circulation and venous outflow site.

Some conservative options such as carotid compression can be tried in some slow-progressing situations; nevertheless, a direct high-flow TCCF in general does not seem to close spontaneously. The primary endovascular treatment is to perform a transarterial or transvenous approach and obliterate the fistula with detachable balloons, micro-coils, Onyx glue or a covered stent [80, 81]. These maneuvers, more often than not, bring about substantial symptom resolution. Adjunctive therapy consists of intraocular pressure control and treatment of exposure keratopathy secondary to proptosis. Surgery may be rarely imperative in refractory cases.

Prognosis

In the absence of treatment, CCF can be associated with substantial morbidity and mortality. Visual prognosis is usually favorable with prompt treatment. However, persistent venous hypertension can result in optic neuropathy or secondary glaucoma [77, 82]. Although ischemic insult can give rise to blindness, it is typical that in such cases the retinal features resolve following successful intervention. This updated etiological classification emphasizes that not all cases appearing as CRVO are primary ocular events, and some warrant urgent systemic evaluation and management.

Case 4: CRVO-like Stasis from Carotid-Cavernous Fistula

A 33-year-old male presented with 2-month duration of right eye redness, proptosis, vision loss, pulsatile tinnitus and history of head trauma six months ago. Examination revealed conjunctival injection, corkscrew veins, mild

proptosis, intraocular pressure of 28 mmHg OD, sixth nerve palsy and fundus findings consistent with CRVO.

CTA and DSA demonstrated a right dural AV fistula with involvement of the cavernous sinus and compromised venous drainage. He was successfully managed with transcatheter coil embolization. Ocular medications were topical IOP -reducing agents and lubricants (Fig. 5).

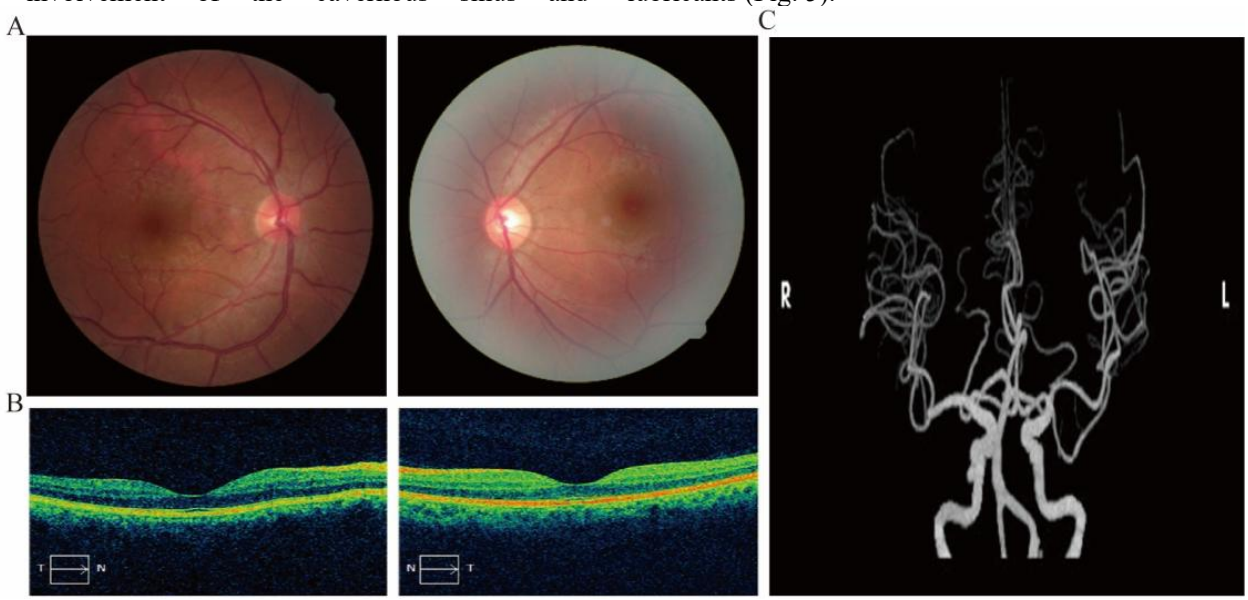

Figure 5. Case 4 –CRVO-like retinal venous stasis secondary to CCF. (A) Color fundus photographs show retinal venous dilation and mild congestion in the right eye, with normal appearance in the left eye. **(B)** OCT reveals preserved macular contour bilaterally. **(C)** CTA confirms a right-sided carotid-cavernous fistula involving the cavernous sinus.

The patient underwent successful transvenous embolization via the femoral vein, with coil deployment to occlude the fistula. Adjunctive ocular management encompassed preoperative topical beta-blockers and carbonic anhydrase inhibitors for IOP control, as well as artificial tears for ocular surface protection. Within days, Within just a few days, there was a dramatic amelioration in both the conjunctival injection and proptosis. Additionally, the intraocular pressure (IOP) returned to a normal level of 18 mmHg, with no need for further medication. No intravitreal injections were required; retinal signs resolved spontaneously.

Although a follow-up fundus photo wasn't accessible, all the eye-related symptoms had completely cleared up. This case illustrates a rare cause of retinal venous stasis related to post-traumatic DAVF and underscores the importance of recognizing intracranial venous anomalies as a cause of ocular pathology.

Other Rare Causes

The four major groups will embrace the majority of mechanisms for retinal venous stasis. Less frequent etiologies may comprise other conditions such as CRVO secondary to a decrease in perfusion pressure, which may commonly arise from proximal arterial stenosis as a consequence of carotid artery or ophthalmic artery stenosis, resulting in a decrease of retinal arterial inflow and venous congestion [83]. On top of that, orbital masses [84] or thyroid-related orbitopathy [85] can induce a mechanical compression of peripheral retinal veins. In rare cases, these causes should be taken into account for specific clinical situations.

DISCUSSION

Illustrated by representative cases, this review proposes a reclassification of retinal venous stasis disorders and demonstrates its clinical relevance through four representative cases. Although there might be some overlap among the different types of venous stasis disorders, each case illustrates a different pathogenic mechanism. The major hypothesis is that RVO is a heterogeneous disease. On this basis, our new classification takes into account the different etiology of RVO, whereas traditional classifications have predominantly taken anatomic localization of the disorder into consideration.

Our classification proposes an etiology-based framework encompassing intrinsic venous pathology, local mechanical compression, translaminar pressure gradient–related outflow impairment, and cavernous sinus disorders which have received limited emphasis in the ophthalmic literature. As summarized in Figure 1, the framework maps these drivers along the venous drainage pathway and links each category to an etiology-directed

diagnostic work-up. Distinguishing CRVO from BRVO remains important for visual prognosis; however, anatomic labels alone provide limited guidance for etiologic evaluation or systemic management. In contrast, the proposed framework supports a broader diagnostic work-up—for example, thrombophilia evaluation in Category 1 and intracranial pressure assessment in Category 3.

It is particularly noteworthy that this etiological classification is tailored for an addition rather than a replacement to the anatomical description. In clinical practice, cases will still be read out as CRVO or BRVO, but with the added flavor of something like "CRVO associated with hypercoagulable state" or "BRVO on account of compression at the arteriovenous crossing", such an integrative method allows for a more global view of the patient.

From an etiologic viewpoint, clinical care can be reinforced in a diverse array of paramount ways. To start with, it encourages a more in-depth exploration of non-typical RVO presentations. Moreover, systemic workup is imperative as a consequence of higher risk in young patients or bilaterality (in Cases 1 and 3). Percussive proptosis and evidence of conjunctival vascular congestion are signs that should cause one to have suspicion for pathology of the cavernous sinus (Case 4). Even quite typical BRVO requires cardiovascular risk evaluation (Case 2). On top of that, this categorization facilitates personalized care by treating etiology.

Intravitreal anti-VEGF injections are currently the first-line treatment for macular edema and neovascular complications. Nonetheless, between 50 to 56% of these patients fail to achieve an optimal response and develop recurrent disease. Identification and treatment of the underlying cause—e.g., anticoagulation for thrombosis, immunosuppression for vasculitis, or neurosurgical intervention for intracranial hypertension or AV fistulas—may give rise to continuous betterment in both ocular findings and systemic symptoms. Last but not least, it is important to know the cause as it affects prognosis. On account of systemic uncontrolled disease (e.g., severe hypertension, and coagulation disorders), RVO has a tendency to relapse. A clear understanding of etiology enables more tailored counseling and fosters sustained engagement with chronic systemic treatment.

The cases and classification we present are consistent with scattered reports in the literature highlighting atypical causes of RVO. Prior studies have linked BRVO with systemic hypertension, CRVO in young adults with hypercoagulable states, and CRVO-like findings in conditions such as IIH or CCF. Nevertheless, to our knowledge, no prior publication has formally brought forth an integrative classification of this kind. By unifying these diverse mechanisms, our framework encourages ophthalmologists to expand their diagnostic thinking when faced with retinal hemorrhage and venous congestion.

Despite its conceptual utility, this reclassification has several limitations. Many cases of RVO are multifactorial, particularly in older patients. For instance, hypertension may contribute to both local compression and a prothrombotic state. The four categories are not mutually exclusive, and overlapping mechanisms may contribute to the same clinical event. In addition, the present case series is small and retrospectively assembled, which limits generalizability and may introduce selection bias in the illustrative cases. Moreover, the framework is grounded in qualitative, narrative synthesis rather than quantitative assessment and lacks external validation. Future multicenter, prospective studies are needed to characterize the distribution of etiologic mechanisms in broader RVO populations and to further evaluate and refine the proposed classification.

CONCLUSION

To sum up, this new categorization offers a new and wider view on retinal venous stasis disorders. Through the examination of illustrative cases, we encourage clinicians to move beyond a one-size-fits-all concept of "venous occlusion" and toward a more personalized approach that takes into account the patient's overall systemic condition rather than centering around nothing but ocular findings.

Author Contributions

XX was responsible for the literature search and drafting of the manuscript. XXZ, CZ conceived the study idea. YG contributed to the revision and organization of the references. WYZ, YFZ, HL and HMJ collected data and provided critical revisions to the manuscript. XXZ approved the final version of the manuscript. All authors have read and approved the final manuscript.

Conflicts of Interest

The authors declare no conflict of interest.

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References

[1] Scott IU, Campochiaro PA, Newman NJ, Biousse V (2020). Retinal vascular occlusions. Lancet (London, England), 396:1927-1940.

[2] Lendzioszek M, Bryl A, Poppe E, Zorena K, Mrugacz M (2024). Retinal vein occlusion-background knowledge and foreground knowledge prospects-A review. J Clin Med, 13.
[3] Romano F, Lamanna F, Gabrielle PH, Teo KYC, Battaglia Parodi M, Iacono P et al. (2023). Update on retinal vein occlusion. Asia Pac J Ophthalmol (Phila), 12:196-210.
[4] Hayreh SS, Zimmerman MB (2015). Fundus changes in central retinal vein occlusion. Retina, 35:29-42.
[5] Jee D, Park S, Kwon JW (2024). Subretinal fluid in macular edema secondary to branch retinal vein occlusion. Sci Rep, 14:13623.
[6] Choi YJ, Jee D, Kwon JW (2022). Characteristics of major and macular branch retinal vein occlusion. Sci Rep, 12:14103.
[7] Song P, Xu Y, Zha M, Zhang Y, Rudan I (2019). Global epidemiology of retinal vein occlusion: a systematic review and meta-analysis of prevalence, incidence, and risk factors. J Glob Health, 9:010427.
[8] Rogers S, Mcintosh RL, Cheung N, Lim L, Wang JJ, Mitchell P et al. (2010). The prevalence of retinal vein occlusion: pooled data from population studies from the United States, Europe, Asia, and Australia. Ophthalmology, 117:313-319.e311.
[9] Ponto KA, Elbaz H, Peto T, Laubert-Reh D, Binder H, Wild PS et al. (2015). Prevalence and risk factors of retinal vein occlusion: the Gutenberg Health Study. J Thromb Haemost, 13:1254-1263.
[10] Koh V, Cheung CY, Li X, Tian D, Wang JJ, Mitchell P et al. (2016). Retinal Vein Occlusion in a Multi-Ethnic Asian Population: The Singapore Epidemiology of Eye Disease Study. Ophthalmic Epidemiol, 23:6-13.
[11] Yasuda M, Kiyohara Y, Arakawa S, Hata Y, Yonemoto K, Doi Y et al. (2010). Prevalence and systemic risk factors for retinal vein occlusion in a general Japanese population: the Hisayama study. Invest Ophthalmol Vis Sci, 51:3205-3209.
[12] Thapa R, Bajimaya S, Paudyal G, Khanal S, Tan S, Thapa SS et al. (2017). Prevalence, pattern and risk factors of retinal vein occlusion in an elderly population in Nepal: the Bhaktapur retina study. BMC Ophthalmol, 17:162.
[13] Hayreh SS (1994). Retinal vein occlusion. Indian J Ophthalmol, 42:109-132.
[14] Green WR, Chan CC, Hutchins GM, Terry JM (1981). Central retinal vein occlusion: a prospective histopathologic study of 29 eyes in 28 cases. Retina, 1:27-55.
[15] Frangieh GT, Green WR, Barraquer-Somers E, Finkelstein D (1982). Histopathologic study of nine branch retinal vein occlusions. Arch Ophthalmol, 100:1132-1140.
[16] Hayreh SS (1983). Classification of central retinal vein occlusion. Ophthalmology, 90:458-474.
[17] Khayat M, Williams M, Lois N (2018). Ischemic retinal vein occlusion: characterizing the more severe spectrum of retinal vein occlusion. Surv Ophthalmol, 63:816-850.
[18] Sabates R, Hirose T, Mcmeel JW (1983). Electroretinography in the prognosis and classification of central retinal vein occlusion. Arch Ophthalmol, 101:232-235.
[19] Silvis SM, De Sousa DA, Ferro JM, Coutinho JM (2017). Cerebral venous thrombosis. Nat Rev Neurol, 13:555-565.
[20] Bai C, Wang Z, Stone C, Zhou D, Ding J, Ding Y et al. (2021). Pathogenesis and Management in Cerebrovenous Outflow Disorders. Aging Dis, 12:203-222.
[21] Li H, Liu L, Zhou Y, Jiang H, Zhang W, Zhou C et al. (2024). Metabolic Dysfunction in Idiopathic Intracranial Hypertension: Current Theories and Updates. Aging Dis.
[22] Janssen MC, Den Heijer M, Cruysberg JR, Wollersheim H, Bredie SJ (2005). Retinal vein occlusion: a form of venous thrombosis or a complication of atherosclerosis? A meta-analysis of thrombophilic factors. Thromb Haemost, 93:1021-1026.
[23] Rehak M, Rehak J, Müller M, Faude S, Faude F, Siegemund A et al. (2008). The prevalence of activated protein C (APC) resistance and factor V Leiden is significantly higher in patients with retinal vein occlusion without general risk factors. Case-control study and meta-analysis. Thromb Haemost, 99:925-929.
[24] Rehak M, Wiedemann P (2010). Retinal vein thrombosis: pathogenesis and management. J Thromb Haemost, 8:1886-1894.
[25] Cahill MT, Stinnett SS, Fekrat S (2003). Meta-analysis of plasma homocysteine, serum folate, serum vitamin B(12), and thermolabile MTHFR genotype as risk factors for retinal vascular occlusive disease. Am J Ophthalmol, 136:1136-1150.
[26] Li D, Zhou M, Peng X, Sun H (2014). Homocysteine, methylenetetrahydrofolate reductase C677T polymorphism, and risk of retinal vein occlusion: an updated meta-analysis. BMC Ophthalmol, 14:147.
[27] Mcgimpsey SJ, Woodside JV, Cardwell C, Cahill M, Chakravarthy U (2009). Homocysteine, methylenetetrahydrofolate reductase C677T polymorphism, and risk of retinal vein occlusion: a meta-analysis. Ophthalmology, 116:1778-1787.e1771.
[28] Rehak M, Müller M, Scholz M, Wiercinska J, Niederwieser D, Wiedemann P (2009). [Antiphospholipid syndrome and retinal vein occlusion. Meta-analysis of Published Studies]. Ophthalmologe, 106:427-434.
[29] Hernández JL, Sanlés I, Pérez-Montes R, Martínez-Taboada VM, Olmos JM, Salmón Z et al. (2020). Antiphospholipid syndrome and antiphospholipid antibody profile in patients with retinal vein occlusion. Thromb Res, 190:63-68.
[30] Alexander P, Flanagan D, Rege K, Foss A, Hingorani M (2008). Bilateral simultaneous central retinal vein occlusion secondary to hyperviscosity in Waldenstrom's macroglobulinaemia. Eye (Lond), 22:1089-1092.
[31] Charles KS, Leelah N, Boodoo L, Murray DC (2013). Ophthalmic manifestations of haematological disorders. West Indian Med J, 62:99-103.
[32] Remolí Sargues L, Montero Hernández J, Navarro Palop C, Monferrer Adsuara C, Castro Navarro V, Cervera Taulet E (2022). Multimodal imaging of two cases of

retinal vein occlusion secondary to Waldenstrom macroglobulinemia. Eur J Ophthalmol, 32:Np50-np55.
[33] Shrestha S, Poddar E, Bashyal B, Adhikari A, Pathak P, Acharya S et al. (2023). Bilateral central retinal vein occlusion as an initial presentation of Waldenström macroglobulinemia: a case report. J Med Case Rep, 17:59.
[34] Rajagopal R, Apte RS (2016). Seeing through thick and through thin: Retinal manifestations of thrombophilic and hyperviscosity syndromes. Surv Ophthalmol, 61:236-247.
[35] Zou Y, Yang K, Zhang X, Li SG (2025). Uncharted territory: retinal vasculitis and cryoglobulinemia in Behçet's disease - a case report. Front Immunol, 16:1533595.
[36] Stafford-Brady FJ, Urowitz MB, Gladman DD, Easterbrook M (1988). Lupus retinopathy. Patterns, associations, and prognosis. Arthritis Rheum, 31:1105-1110.
[37] Herbort CP, Rao NA, Mochizuki M (2009). International criteria for the diagnosis of ocular sarcoidosis: results of the first International Workshop On Ocular Sarcoidosis (IWOS). Ocul Immunol Inflamm, 17:160-169.
[38] Datoo O'keefe GA, Rao N (2021). Retinal vasculitis: A framework and proposal for a classification system. Surv Ophthalmol, 66:54-67.
[39] Arepalli SR, Thomas AS (2022). Occlusive retinal vasculitis: novel insights into causes, pathogenesis and treatment. Curr Opin Ophthalmol, 33:147-156.
[40] Horvath D, Aljameey U, Douglas E (2023). Double Trouble: Eales Disease in a Background of Paradoxical Embolism. Cureus, 15:e44708.
[41] Eski Yücel O, Güngor I, Gül A, Turgut M, Aritürk N (2015). Papillophlebitis associated with the use of oral contraceptive: a case report. Gynecol Endocrinol, 31:601-603.
[42] Fong AC, Schatz H, Mcdonald HR, Burton TC, Maberley AL, Joffe L et al. (1992). Central retinal vein occlusion in young adults (papillophlebitis). Retina, 12:3-11.
[43] Ellenberger C, Jr., Messner KH (1978). Papillophlebitis: benign retinopathy resembling papilledema or papillitis. Ann Neurol, 3:438-440.
[44] Zhang XT, Zhong YF, Xue YQ, Li SQ, Wang BY, Zhang GQ et al. (2022). Clinical Features of Central Retinal Vein Occlusion in Young Patients. Ophthalmol Ther, 11:1409-1422.
[45] Tomita R, Goto K, Ueno Y, Yamaguchi K, Takeuchi J, Akahori T et al. (2023). Narrowing Ratio of Retinal Veins at Arteriovenous Crossing in Patients With Branch Retinal Vein Occlusion Versus That in Healthy Individuals. Invest Ophthalmol Vis Sci, 64:22.
[46] Muraoka Y, Tsujikawa A (2019). Arteriovenous crossing associated with branch retinal vein occlusion. Jpn J Ophthalmol, 63:353-364.
[47] Iida Y, Muraoka Y, Ooto S, Suzuma K, Murakami T, Iida-Miwa Y et al. (2017). Morphologic and Functional Retinal Vessel Changes in Branch Retinal Vein Occlusion: An Optical Coherence Tomography Angiography Study. Am J Ophthalmol, 182:168-179.
[48] Xu K, Wu L, Ma Z, Liu Y, Qian F (2019). Primary angle closure and primary angle closure glaucoma in retinal vein occlusion. Acta Ophthalmol, 97:e364-e372.
[49] Albrecht EA, Shukla P, Zhao AH, Markle JC, Maatouk CM, Singh RP et al. (2025). Risk of Adverse Systemic Events in Retinal Vein Occlusion. Ophthalmol Retina.
[50] Darabuş DM, Dărăbuş RG, Munteanu M (2025). The Diagnosis and Treatment of Branch Retinal Vein Occlusions: An Update. Biomedicines, 13.
[51] Larrousse Morellón M, López Loureiro Y, Ruiz Bilbao S (2024). Retinal venous occlusion and its association with atherosclerotic vascular disease. Med Clin (Barc), 163:199-207.
[52] Nicholson L, Talks SJ, Amoaku W, Talks K, Sivaprasad S (2022). Retinal vein occlusion (RVO) guideline: executive summary. Eye (London, England), 36:909-912.
[53] Jaulim A, Ahmed B, Khanam T, Chatziralli IP (2013). Branch retinal vein occlusion: epidemiology, pathogenesis, risk factors, clinical features, diagnosis, and complications. An update of the literature. Retina, 33:901-910.
[54] Munk MR, Ceklic L, Stillenmunkes R, Chaudhary V, Waheed N, Chhablani J et al. (2024). Integrated Assessment of OCT, Multimodal Imaging, and Cytokine Markers for Predicting Treatment Responses in Retinal Vein Occlusion Associated Macular Edema: A Comparative Review of Anti-VEGF and Steroid Therapies. Diagnostics (Basel), 14.
[55] Choi EY, Kim D, Kim J, Kim E, Lee H, Yeo J et al. (2025). Predicting branch retinal vein occlusion development using multimodal deep learning and pre-onset fundus hemisection images. Sci Rep, 15:2729.
[56] Woo SC, Lip GY, Lip PL (2016). Associations of retinal artery occlusion and retinal vein occlusion to mortality, stroke, and myocardial infarction: a systematic review. Eye (Lond), 30:1031-1038.
[57] Seong HJ, Lee JH, Heo JH, Kim DS, Kim YB, Lee CS (2021). CLINICAL SIGNIFICANCE OF RETINAL VASCULAR OCCLUSION IN MOYAMOYA DISEASE: Case Series and Systematic Review. Retina, 41:1791-1798.
[58] Liu KC, Fleischman D, Lee AG, Killer HE, Chen JJ, Bhatti MT (2020). Current concepts of cerebrospinal fluid dynamics and the translaminar cribrosa pressure gradient: a paradigm of optic disk disease. Surv Ophthalmol, 65:48-66.
[59] Iencean SM, Ciurea AV (2008). Intracranial hypertension: classification and patterns of evolution. J Med Life, 1:101-107.
[60] Stingl JV, Nunez LP, Schuster AK, Hoffmann EM (2020). Venous stasis retinopathy in a ten-year-old boy with ocular hypertension: a case report. BMC Ophthalmol, 20:428.
[61] Dutta P, Sontakke R, Tyagi S, Dhaka U (2025). Venous stasis retinopathy in increased intracranial pressure. BMJ Case Rep, 18.
[62] Krakau CE (1994). Disk hemorrhages and retinal vein occlusions in glaucoma. Surv Ophthalmol, 38 Suppl:S18-21; discussion S22.

[63] Reier L, Fowler JB, Arshad M, Hadi H, Whitney E, Farmah AV et al. (2022). Optic Disc Edema and Elevated Intracranial Pressure (ICP): A Comprehensive Review of Papilledema. Cureus, 14:e24915.

[64] Beier D, Korsbæk JJ, Bsteh G, Macher S, Marik W, Pemp B et al. (2024). Magnetic Resonance Imaging Signs of Idiopathic Intracranial Hypertension. JAMA Netw Open, 7:e2420138.

[65] Remolí-Sargues L, Monferrer-Adsuara C, López-Salvador B, García-Villanueva C, Gracia-García A, Castro-Navarro V et al. (2024). Optical coherence tomography angiography analysis in patients with intracranial hypertension. Eur J Ophthalmol, 34:1586-1593.

[66] Markey KA, Mollan SP, Jensen RH, Sinclair AJ (2016). Understanding idiopathic intracranial hypertension: mechanisms, management, and future directions. Lancet Neurol, 15:78-91.

[67] Ferro JM, Aguiar De Sousa D (2019). Cerebral Venous Thrombosis: an Update. Curr Neurol Neurosci Rep, 19:74.

[68] Puffer RC, Mustafa W, Lanzino G (2013). Venous sinus stenting for idiopathic intracranial hypertension: a review of the literature. J Neurointerv Surg, 5:483-486.

[69] Salih M, Enriquez-Marulanda A, Khorasanizadeh M, Moore J, Prabhu VC, Ogilvy CS (2022). Cerebrospinal Fluid Shunting for Idiopathic Intracranial Hypertension: A Systematic Review, Meta-Analysis, and Implications for a Modern Management Protocol. Neurosurgery, 91:529-540.

[70] Trucchi L, Cohen M, Nahon-Esteve S, Lagier J, Leal C, Almairac F et al. (2023). Optic nerve sheath fenestration: Current status in France and comparison of 6 different surgical approaches. J Fr Ophtalmol, 46:137-147.

[71] Stein JD, Khawaja AP, Weizer JS (2021). Glaucoma in Adults-Screening, Diagnosis, and Management: A Review. Jama, 325:164-174.

[72] Balcerzak A, Tubbs RS, Zielinska N, Olewnik Ł (2023). Clinical analysis of cavernous sinus anatomy, pathologies, diagnostics, surgical management and complications - Comprehensive review. Ann Anat, 245:152004.

[73] Long B, Field SM, Singh M, Koyfman A (2024). High risk and low prevalence diseases: Cavernous sinus thrombosis. Am J Emerg Med, 83:47-53.

[74] Zaninetti M, Stangos AN, Abdo G, Pournaras CJ (2005). Cavernous sinus thrombosis elicited by a central retinal vein venous stasis retinopathy. Graefes Arch Clin Exp Ophthalmol, 243:834-836.

[75] Akarapas C, Wiwatkunupakarn N, Sithirungson S, Chaiyasate S (2025). Anticoagulation for cavernous sinus thrombosis: a systematic review and individual patient data meta-analysis. Eur Arch Otorhinolaryngol, 282:1127-1134.

[76] Barrow DL, Spector RH, Braun IF, Landman JA, Tindall SC, Tindall GT (1985). Classification and treatment of spontaneous carotid-cavernous sinus fistulas. J Neurosurg, 62:248-256.

[77] Alam MS, Jain M, Mukherjee B, Sharma T, Halbe S, Jaisankar D et al. (2019). Visual impairment in high flow and low flow carotid cavernous fistula. Sci Rep, 9:12872.

[78] Al-Shalchy A, Al-Wassiti AS, Hashim MA, Al-Khazaali YM, Talib SH, Bani-Saad AA et al. (2024). Neuro-Ophthalmic Manifestations of Carotid Cavernous Fistulas: A Systematic Review and Meta-Analysis. Cureus, 16:e65821.

[79] Henderson AD, Miller NR (2018). Carotid-cavernous fistula: current concepts in aetiology, investigation, and management. Eye (Lond), 32:164-172.

[80] Baharvahdat H, Qoorchi Moheb Seraj F, Al-Raaisi A, Blanc R, Najafi S, Mirbolouk MH et al. (2024). Long-term outcome of endovascular treatment for indirect carotid-cavernous fistulas. Neurosurg Focus, 56:E5.

[81] Yuan J, Yang R, Zhang J, Liu H, Ye Z, Chao Q (2024). Covered Stent Treatment for Direct Carotid-Cavernous Fistulas: A Meta-Analysis of Efficacy and Safety Outcomes. World Neurosurg, 187:e302-e312.

[82] Ishijima K, Kashiwagi K, Nakano K, Shibuya T, Tsumura T, Tsukahara S (2003). Ocular manifestations and prognosis of secondary glaucoma in patients with carotid-cavernous fistula. Jpn J Ophthalmol, 47:603-608.

[83] Pournaras JA, Konstantinidis L, Wolfensberger TJ (2010). Sequential central retinal artery occlusion and retinal vein stasis as a result of ocular ischemic syndrome. Klin Monbl Augenheilkd, 227:338-339.

[84] Foroozan R (2004). Combined central retinal artery and vein occlusion from orbital inflammatory pseudotumour. Clin Exp Ophthalmol, 32:435-437.

[85] Jeeva-Patel T, Emami S, Margolin E (2022). Central Retinal Vein Occlusion and Compressive Optic Neuropathy Secondary to Thyroid Eye Disease. Can J Neurol Sci, 49:302-304.