Retinal vein occlusion: Epidemiology, clinical manifestations, and diagnosis

AUTHORS: Dennis P Han, MD, Baseer Ahmad, MD
SECTION EDITOR: Jonathan Trobe, MD
DEPUTY EDITOR: Jane Givens, MD, MSCE

All topics are updated as new evidence becomes available and our peer review process is complete.

Literature review current through: **May 2024.**
This topic last updated: **Jan 03, 2024.**

INTRODUCTION

Retinal vein occlusion (RVO) is an important cause of visual loss among adults throughout the world [1,2].

The epidemiology, clinical manifestations, and diagnostic evaluation of RVO will be discussed here. The treatment of RVO, as well as issues related to retinal artery occlusion, are discussed separately. (See "Retinal vein occlusion: Treatment" and "Central and branch retinal artery occlusion".)

CLASSIFICATION

The classification of retinal vein occlusion (RVO) is dependent on the anatomic location of the occlusion and includes: branch retinal vein occlusion (BRVO), central retinal vein occlusion (CRVO), and hemiretinal vein occlusion (HRVO) (figure 1).

- BRVO occurs when a vein in the distal retinal venous system is occluded, most often due to compression by an overlying arteriole, leading to hemorrhage along the distribution of a small vessel of the retina (picture 1).
- CRVO occurs due to thrombus within the central retinal vein at the level of the lamina cribrosa of the optic nerve, leading to involvement of the entire retina (picture 2). The

lamina cribrosa is the connective tissue "sieve" consisting of holes through which the nerve fibers pass from the retina to the optic nerve.

- HRVO occurs when the superior and inferior retinal drainage does not merge into a single central retinal vein and one of the two trunks is occluded (generally more similar to CRVO than BRVO), leading to involvement of one half of the retina (picture 3).

EPIDEMIOLOGY

Prevalence — Retinal vein occlusion (RVO) is the second most common cause of vision loss from retinal vascular disease, following diabetic retinopathy [3]. The prevalence of RVO increases with age. Branch retinal vein occlusion (BRVO) is more common than central retinal vein occlusion (CRVO). Hemiretinal vein occlusion (HRVO) is relatively rare and less well studied [1,2].

- In a population cohort of adults in the United States, the 15-year cumulative incidence of BRVO and CRVO were 1.8 and 0.5 percent respectively [4,5].
- In an Australian population, the prevalence of any form of RVO was 0.7 percent in persons younger than 60 years and 4.6 percent in those 80 years or older [6].
- In a pooled analysis of data from 15 studies (68,751 individuals) conducted in the United States, Europe, Asia and Australia, the estimated prevalence of BRVO was 2.8 per 1000 in White, 3.5 in Black, 5.0 in Asian, and 6.0 in Hispanic individuals [1]. Prevalences of CRVO were 0.88 per 1000 in White, 0.37 in Black, 0.74 in Asian, and 1.0 in Hispanic individuals.

Risk factors — The following are risk factors for RVO [3,7-13]:

- Older age
- Hypertension
- Diabetes (associated with CRVO but not BRVO)
- Cardiovascular disease
- Smoking
- Obesity
- Hypercoagulable state, particularly factor V Leiden and activated protein C resistance
- Hyperlipidemia
- Glaucoma (particularly for CRVO)
- Retinal arteriolar abnormalities

Thrombophilic risk factors may be more prevalent in younger patients, particularly those who are healthier and have not faced the age-related vasculopathies listed above [14].

PATHOPHYSIOLOGY

The pathophysiology of branch retinal vein occlusion (BRVO) and central retinal vein occlusion (CRVO) are different [15-19]. The pathophysiology of hemiretinal vein occlusion (HRVO) shares many features of CRVO.

- **BRVO** – BRVO is thought to be due to blockage of retinal veins at arteriovenous crossings [12,15]. It has been hypothesized that an inflexible, atherosclerotic arteriole deforms the relatively distensible veins (contained in an inelastic arteriovenous sheath), resulting in a venous occlusion. The increased incidence in the superotemporal quadrant is thought to be due to increased arteriovenous crossings in that quadrant. Several studies have suggested that the risk is greater among eyes where the retinal artery is anterior to the retinal vein [16-18].

- **CRVO** – In CRVO, the pathophysiology is due to primary formation of a thrombus consisting of fibrin and platelets in the central retinal vein [19]. Obstructions located in the anterior lamina cribrosa or distally (toward the retina) are presumed to reduce the ability to form collateral flow from the retinal circulation to the peripapillary choroid. The resultant high-grade obstruction leads to poorer retinal capillary perfusion and ischemia. Eyes with greater ischemia have more profound visual impairment. Ischemic or nonischemic (perfused) CRVO are classified according to retinal angiography.

- **HRVO** – The pathophysiology of HRVO is presumed to be similar to CRVO, but with greater potential for collateralization within the retinal circulation, a smaller area of affected retina, and a correspondingly lower potential for severe nonperfusion and ischemia.

CLINICAL MANIFESTATIONS

Symptoms — Patients with retinal vein occlusion may describe a scotoma or visual field deficit with blurred or gray vision corresponding to the area of occlusion. The visual field deficit develops over hours to weeks and is commonly due to macular edema. Pain is notably absent because the retina has no trigeminal innervation.

- Patients with BRVO may be asymptomatic or may present with peripheral visual field defects in the affected eye. However, if the occlusion involves the macula or results in

macular edema, patients will complain of blurred central vision.

- Patients with CRVO usually complain of the acute onset of broad-based painless blurred vision in one eye. CRVO is seldom asymptomatic.

- Patients with hemiretinal vein occlusion (HRVO) usually complain of blurred central vision because the occlusion typically involves the macula.

Patients with ischemic CRVO may go on to develop neovascular glaucoma due to neovascularization of the iris and/or the anterior chamber angle. These patients complain of a red, painful eye secondary to elevated intraocular pressure, in addition to decreased vision. Neovascular glaucoma has been deemed “90-day glaucoma” because of its common onset within a few months of onset of ischemic CRVO. (See 'Ocular neovascularization' below.)

Findings on eye examination

- Examination findings of the ocular fundus in RVO include retinal hemorrhage, edema, dilated retinal venules, and cotton wool spots throughout the distribution of the affected vein.

  - In BRVO, the area of retinal hemorrhage is focal or wedge-shaped, with the apex situated at the offending arteriovenous crossing (picture 4). There may also be venous dilatation radiating from the arteriovenous crossing (image 1).

  - In CRVO, optic disc edema may occur. The classic appearance of CRVO has been termed "blood and thunder" fundus. Dilated and tortuous veins are visualized. All four quadrants have intraretinal hemorrhages (picture 2). Cotton wool spots are observed in approximately half of patients with CRVO [20]. The arterioles may be attenuated, indicating generalized arteriosclerotic disease.

- Visual acuity can be normal but is more likely to be decreased, with greater potential for severe visual loss in CRVO than in BRVO.

- Extraocular motility is normal.

- Intraocular pressure is unaffected in BRVO. In CRVO, the initial intraocular pressure may be slightly lower in the affected eye than the unaffected eye, presumably due to ciliary body hyposecretion. In cases of neovascular glaucoma (a later finding), it can be severely elevated due to outflow obstruction through the trabecular meshwork by neovascularization or by synechial angle closure.

- Pupillary function is normal in BRVO. In CRVO, pupillary examination can be normal, but in cases of nonperfused CRVO there is typically an ipsilateral relative afferent pupillary defect (picture 5).

- Anterior segment examination (slit lamp findings) are unaffected, except in the case of neovascular glaucoma (a later finding) in which neovascularization of the iris and anterior chamber angle occurs. Neovascularization of the iris is often detected earliest in the peripupillary region but can develop initially in the anterior chamber angle, particularly in aphakic eyes. Neovascularization of the iris and anterior chamber angle is best detected prior to pharmacologic pupillary dilation, which puts the peripupillary iris on stretch, making abnormal vessels less visible.

- Confrontation visual fields are variable, depending upon the location and density of visual field loss. In BRVO, detailed confrontation visual fields with a small test object may detect paracentral scotomas or peripheral defects; in CRVO, there may be generalized loss of sensitivity without a focal defect seen. Low levels of vision, such as when the patient is only able to perceive hand motions or light perception, may permit only a gross assessment of visual field involvement.

Natural history and complications — The natural history of visual acuity after RVO varies based on the size and type of occlusion. Complications of RVO can include macular edema, neovascularization, and, less commonly, vitreous hemorrhage. In addition, as RVO may be a marker of underlying cardiovascular disease, it has been associated with future stroke, myocardial infarction, and cardiovascular mortality in observational studies [21-23].

Effects on visual acuity — Whether visual loss progresses depends largely on the type and location of the occlusion as well as any development of macular edema [24,25].

- **BRVO** – Patients with BRVO without macular edema are commonly asymptomatic and maintain good vision. Patients with macular edema may experience spontaneous vision improvement in the first few months after onset of symptoms due to self-limiting resolution or improvement of the edema. However, after three months, the likelihood of spontaneous improvement in visual acuity diminishes. Three-year visual acuity outcomes in untreated eyes with BRVO-associated macular edema and visual acuity of 20/40 or worse at three months were as follows: 34 percent with visual acuity of 20/40 or better, 23 percent with visual acuity of 20/200 or worse, and an average visual acuity of 20/70 [26].

- **CRVO** – Patients with CRVO have generally worse outcomes, with final visual acuity dependent upon visual acuity at presentation. The Central Retinal Vein Occlusion Study followed 725 patients with CRVO over three years [25]. Two-thirds of patients who

presented with visual acuity better than 20/40 maintained visual acuity in the same range, and only 10 percent had worsening of visual acuity to less than 20/200. Among patients who presented with visual acuity 20/50 to 20/200, nearly half maintained visual acuity in the same range and one-third progressed to a visual acuity of less than 20/200. Only 20 percent of patients with initial visual acuity of less than 20/200 had some improvement in vision. Patients with ischemic CRVO are at high risk for poor visual acuity at initial presentation and over the long term compared with those with nonischemic CRVO [27].

Ocular neovascularization — Neovascularization of the iris, the anterior chamber angle, and retina are common complications of CRVO and BRVO, although these complications occur more commonly in CRVO. In the Central Retinal Vein Occlusion Study, neovascularization of the iris and/or anterior chamber angle developed in 16 percent of patients [25]. The strongest predictors of neovascularization of the iris and/or anterior chamber angle were visual acuity at diagnosis and the size of the area of nonperfusion on fluorescein angiogram. For eyes initially categorized as nonperfused (ischemic) or indeterminate, 35 percent developed neovascularization of the iris and/or anterior chamber angle, compared with 10 percent of those initially categorized as perfused [25].

Neovascularization of the retina can lead to vitreous hemorrhage, traction retinal detachment, and neovascular glaucoma. Vitreous hemorrhage may produce visual disturbances such as severe floaters that obscure vision. Development of neovascular glaucoma may result in significant ocular morbidity, such as pain, chronic ocular redness, unremitting intraocular pressure elevation, and profound visual loss that is often unresponsive to anti-glaucoma drops or surgery. Neovascularization of the iris and neovascularization of the anterior chamber angle are considered harbingers of neovascular glaucoma and are most likely to occur in eyes with ischemic CRVO. (See "Retinal detachment" and "Approach to the adult with acute persistent visual loss", section on 'Vitreous hemorrhage'.)

Differential diagnosis — History taking and symptomatology can help to broadly consider a range of diagnoses that may apply to sudden, painless, severe visual loss involving one eye:

- Central or branch retinal artery occlusion
- Retinal detachment
- Ischemic optic neuropathy
- Vitreous hemorrhage

Other causes of monocular vision loss are discussed elsewhere. (See "Approach to the adult with acute persistent visual loss" and "Amaurosis fugax (transient monocular or binocular visual loss)".)

If dilated eye examination with funduscopy or retinal fundus photography allow for visualization of examination findings, then the differential diagnosis for intraretinal hemorrhages (with or without optic nerve head or macular edema) can include:

- Diabetic retinopathy
- Acute hypertensive retinopathy
- Ocular ischemic syndrome
- Retinal vasculitis
- Radiation retinopathy
- Viral retinitis (particularly cytomegalovirus retinitis), which can produce hemorrhages and whitish-gray retinal opacification along the vascular arcades

Additional diagnoses for macular edema can include a variety of conditions, including choroidal neovascularization, pseudophakic cystoid macular edema, and diabetic retinopathy.

DIAGNOSTIC EVALUATION

RVO should be suspected in patients, particularly those over age 50, who present with sudden, painless, severe visual loss involving one eye or in asymptomatic patients with suggestive findings on routine examination of the ocular fundus (eg, retinal hemorrhage, edema, dilated retinal venules, and cotton wool spots throughout the distribution of the affected vein). (See 'Symptoms' above and 'Findings on eye examination' above.)

While a clinical diagnosis can be made based on history and characteristic ophthalmologic examination, a fluorescein angiogram may also be performed to confirm the diagnosis. It is usually performed at the initial evaluation. This study is also useful in identifying retinal capillary nonperfusion for purposes of determining the risk of neovascularization and need for follow-up. The most common testing for RVO is optical coherence tomography (OCT), a type of quick and noninvasive retinal scanning, which allows for diagnosis and monitoring of macular edema.

History — Although the diagnosis is based primarily on eye examination and other diagnostic studies, a history is important for documenting the patient's symptomatic visual loss and the presence of other comorbid conditions. Typical ocular symptoms are described in detail above. (See 'Symptoms' above.)

A history of other comorbid conditions should be documented, particularly cardiovascular risk factors that may predispose to RVO. (See 'Risk factors' above and 'Additional testing to identify risk factors' below.)

Eye examination

- Initial examination – The initial eye examination should include evaluation of visual acuity, extraocular motility, intraocular pressure, pupillary function, external and internal structures of the eye, confrontation visual fields, and slit lamp examination. The presence of any injection/redness of the conjunctiva and sclera should also be noted. A dilated ophthalmoscopic examination may be deferred to the ophthalmologist, particularly if unsure about the presence of afferent pupillary defect or if anterior neovascularization (rubeosis) is suggested by a red or painful eye. Typical findings are described above. (See 'Findings on eye examination' above.)

- Subsequent examination – In eyes with severe visual loss and suspected nonperfused central retinal vein occlusion (CRVO), an undilated examination of the iris should be performed within three to six months to detect neovascularization of the iris. Gonioscopy is also advisable to detect neovascularization of the anterior chamber angle, which is associated with increased intraocular pressure. Dilated eye examination should also be performed in conjunction for assessment of retinal neovascularization and macular edema.

  During treatment, visual acuity should be documented at each visit to assess RVO progression or regression (see "Retinal vein occlusion: Treatment"). Assessment of visual acuity is discussed in detail elsewhere. (See "Visual impairment in adults: Refractive disorders and presbyopia", section on 'Screening and diagnostic tests'.)

Ophthalmology referral — Primary care providers who suspect a patient has an RVO associated with sudden visual loss, especially with eye pain or redness, should urgently refer the patient to an ophthalmologist. Prior to referral, visual acuity should be obtained, ideally with the patient's most recent spectacle correction, if available. Unless already done, pharmacologic dilation of the pupils should be avoided so as to allow assessment by the ophthalmologist for a relative afferent pupillary defect and for rubeosis of the iris or anterior chamber angle. If the diagnosis is uncertain, the patient should be instructed to avoid oral intake until cleared by the ophthalmologist, since other conditions requiring urgent surgical intervention may also produce sudden visual loss. However, for patients with suspected RVO but without acute visual loss, a non-urgent referral is appropriate.

Fluorescein angiography — Fluorescein angiography is a procedure lasting approximately 10 to 20 minutes in which fluorescein dye is injected into a peripheral vein, and a special camera is used to visualize retinal blood flow. It is the definitive test to confirm the diagnosis of RVO and to assess the degree of retinal nonperfusion. The fluorescein angiogram may reveal delayed

venous filling, staining of affected retinal veins, and angiographic macular leakage in RVOs of recent and longstanding duration. In patients in whom the diagnosis of RVO is clear on the basis of ophthalmoscopic findings alone and the area of retinal involvement is less than 10 disk areas, the value of performing fluorescein angiography lies mainly in determining whether laser photocoagulation therapy or intraocular injections of vascular endothelial growth factor (VEGF) inhibitors should be considered. (See "Retinal vein occlusion: Treatment", section on 'Retinal laser photocoagulation'.)

The fluorescein angiogram also allows quantification of the surface area of capillary nonperfusion. The Central Retinal Vein Occlusion Study Group established a perfusion classification scheme, based on disk area, that is now commonly used [20]. A disk area represents the surface area of the optic nerve head. In this scheme, cases with <10 disk areas of capillary nonperfusion are classified as "perfused CRVO," and cases with ≥10 disk areas of capillary nonperfusion are classified as "nonperfused CRVO." Cases of CRVO where the extent of capillary nonperfusion cannot be determined due to extensive intraretinal hemorrhage are classified as "indeterminant" [3]. BRVO is further classified into perfused (non-ischemic) or nonperfused (ischemic). Ischemic BRVO is defined as >5 disc diameters of nonperfusion on fluorescein angiography [26]. The amount and location of capillary nonperfusion in branch retinal vein occlusions (BRVOs) is helpful to predict visual acuity and neovascularization risk.

In longstanding BRVO and CRVO that has been present for several months or more, the fluorescein angiogram helps document the presence of collateral vessels, which divert blood from the retinal circulation to the choroidal circulation. These vessels are larger in caliber than those of neovascularization and do not leak in the late frames of the angiogram. By contrast, the neovascular channels appear as fine fronds that leak profusely in the late frames.

Additional testing to identify risk factors — We screen patients for cardiovascular risk factors by obtaining a fasting glucose or hemoglobin A1C and a fasting lipid panel. In addition, we perform a hypercoagulability workup in patients with a high pretest probability or suspicion for potential hypercoagulability; these primarily include patients with a personal or family history suggestive of a hypercoagulable state and patients under age 50 who do not have strong evidence of arteriosclerotic risk factors. Hypercoagulable testing is discussed in detail elsewhere. (See "Evaluating adult patients with established venous thromboembolism for acquired and inherited risk factors", section on 'Hypercoagulable tests'.)

Studies have variable outcomes regarding the potential association between RVO and hypercoagulable markers, and a 2020 meta-analysis did not support routine thrombophilia screening in individuals with retinal vein occlusion, although it acknowledged possible limited applicability of their findings to young patients without cardiovascular risk factors [7,10,28,29].

Patients with an abnormal hypercoagulable workup are referred to hematology for further evaluation and management. Good communication with the receiving hematology service is also advised, as some hematologists prefer to perform the initial workup themselves or in accordance with institutional preference.

Optical coherence tomography for monitoring — Macular optical coherence tomography (OCT) allows high-resolution cross sectional imaging of the retina [30]. Its main use in retinal vein occlusion (RVO) is in quantifying retinal thickening from intraretinal fluid in cases of macular edema (image 2). OCT can then be used on follow-up visits to assess the progression of RVO or response to treatment.

SUMMARY AND RECOMMENDATIONS

- Retinal vein occlusion (RVO) is an important cause of ocular morbidity that has been observed in populations across the globe. Risk factors in descending order of likelihood include, but are not limited to, older age, hypertension, diabetes, cardiovascular disease, smoking and hypercoagulable states. (See 'Epidemiology' above.)

- The classification of RVO is based on the site of retinal vein involvement. Branch retinal vein occlusion (BRVO) occurs when a distal retinal vein is occluded, leading to hemorrhage along the distribution of a small vessel of the retina (picture 1). Central retinal vein occlusion (CRVO) occurs when a proximal retinal vein is occluded, leading to involvement of the entire retina (picture 2). Hemiretinal vein occlusion (HRVO) is the least common of these entities and describes blockage of a vein that drains the superior or inferior hemiretina, leading to involvement of one-half of the retina (picture 3). (See 'Classification' above.)

- BRVO is thought to be due to blockage of retinal veins by compression from overlying arterioles at arteriovenous crossings, most commonly due to age, hypertension, and arteriosclerosis. In CRVO, the pathophysiology is due to primary formation of a thrombus consisting of fibrin and platelets in the central retinal vein, and though age and atherosclerotic risk factors similarly apply, the differential diagnosis may be broader or more systemic. The pathophysiology of HRVO is presumed to be similar to CRVO. (See 'Pathophysiology' above.)

- Patients with BRVO may be asymptomatic, with abnormalities limited to those detected on routine eye examination. Other patients present with visual field defects and, if the occlusion involves the macula, reduced visual acuity. Patients with CRVO usually report

reduced visual acuity. Because the retina has no trigeminal innervation, patients do not report ocular pain. (See 'Symptoms' above.)

- Patients with ischemic CRVO may go on to develop neovascular glaucoma. These patients complain of a red, painful eye secondary to elevated intraocular pressure, in addition to decreased vision. This is because of neovascularization of the iris and/or neovascularization of the anterior chamber angle. Neovascular glaucoma has been deemed "90-day glaucoma" because of its common onset within a few months of onset of ischemic CRVO. (See 'Symptoms' above.)

- Examination findings of the ocular fundus in RVO include retinal hemorrhage, edema, dilated retinal venules, and cotton wool spots throughout the distribution of the affected vein (picture 4 and image 1). Other findings on eye examination are discussed above. (See 'Findings on eye examination' above.)

- The natural history of visual acuity after RVO varies according to the size and type of occlusion. Complications of RVO can include macular edema, neovascularization, and, less commonly, vitreous hemorrhage. (See 'Natural history and complications' above.)

- The diagnosis of RVO can be made presumptively on the basis of a characteristic history and findings on ophthalmologic examination. Important components of the ophthalmic examination include visual acuity and dilated fundus examination looking for hemorrhage, edema, and dilatation of the retinal veins. Fluorescein angiogram is sometimes performed by an ophthalmologist for confirmation of diagnosis, for prognostication, or to assess need for treatment. Optical coherence tomography (OCT) is very commonly used for determination or monitoring of macular edema. (See 'Diagnostic evaluation' above and 'Fluorescein angiography' above.)

- In eyes with severe visual loss and suspected nonperfused CRVO, an undilated examination of the iris should be performed within three to six months to detect neovascularization of the iris. Gonioscopy should be performed to detect neovascularization of the anterior chamber angle, which is associated with increased intraocular pressure. Dilated eye examination should also be performed in conjunction for assessment of retinal neovascularization and macular edema. (See 'Eye examination' above.)

- We screen patients for cardiovascular risk factors by obtaining a fasting glucose or hemoglobin A1C and a fasting lipid panel. In addition, we perform a hypercoagulable workup in patients with a personal or family history suggesting a hypercoagulable state,

and in patients under age 50 who do not have strong evidence of arteriosclerotic risk factors. (See 'Additional testing to identify risk factors' above.)

ACKNOWLEDGMENT

The UpToDate editorial staff acknowledges Douglas Covert, MD, MPH, who contributed to an earlier version of this topic review.

REFERENCES

1. Rogers S, McIntosh RL, Cheung N, et al. The prevalence of retinal vein occlusion: pooled data from population studies from the United States, Europe, Asia, and Australia. Ophthalmology 2010; 117:313.
2. Song P, Xu Y, Zha M, et al. Global epidemiology of retinal vein occlusion: a systematic review and meta-analysis of prevalence, incidence, and risk factors. J Glob Health 2019; 9:010427.
3. Cugati S, Wang JJ, Rochtchina E, Mitchell P. Ten-year incidence of retinal vein occlusion in an older population: the Blue Mountains Eye Study. Arch Ophthalmol 2006; 124:726.
4. Klein R, Klein BE, Moss SE, Meuer SM. The epidemiology of retinal vein occlusion: the Beaver Dam Eye Study. Trans Am Ophthalmol Soc 2000; 98:133.
5. Klein R, Moss SE, Meuer SM, Klein BE. The 15-year cumulative incidence of retinal vein occlusion: the Beaver Dam Eye Study. Arch Ophthalmol 2008; 126:513.
6. Mitchell P, Smith W, Chang A. Prevalence and associations of retinal vein occlusion in Australia. The Blue Mountains Eye Study. Arch Ophthalmol 1996; 114:1243.
7. Rehak M, Rehak J, Müller M, et al. 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 2008; 99:925.
8. Newman-Casey PA, Stem M, Talwar N, et al. Risk factors associated with developing branch retinal vein occlusion among enrollees in a United States managed care plan. Ophthalmology 2014; 121:1939.
9. Hayreh SS, Zimmerman B, McCarthy MJ, Podhajsky P. Systemic diseases associated with various types of retinal vein occlusion. Am J Ophthalmol 2001; 131:61.
10. Janssen MC, den Heijer M, Cruysberg JR, et al. Retinal vein occlusion: a form of venous thrombosis or a complication of atherosclerosis? A meta-analysis of thrombophilic factors.

Thromb Haemost 2005; 93:1021.

11. O'Mahoney PR, Wong DT, Ray JG. Retinal vein occlusion and traditional risk factors for atherosclerosis. Arch Ophthalmol 2008; 126:692.
12. Kaderli AA, Kaderli B, Gullulu S, Avci R. Impaired aortic stiffness and pulse wave velocity in patients with branch retinal vein occlusion. Graefes Arch Clin Exp Ophthalmol 2010; 248:369.
13. Yin X, Li J, Zhang B, Lu P. Association of glaucoma with risk of retinal vein occlusion: A meta-analysis. Acta Ophthalmol 2019; 97:652.
14. Rehak M, Krcova V, Slavik L, et al. The role of thrombophilia in patients with retinal vein occlusion and no systemic risk factors. Can J Ophthalmol 2010; 45:171.
15. Bowers DK, Finkelstein D, Wolff SM, Green WR. Branch retinal vein occlusion. A clinicopathologic case report. Retina 1987; 7:252.
16. Zhao J, Sastry SM, Sperduto RD, et al. Arteriovenous crossing patterns in branch retinal vein occlusion. The Eye Disease Case-Control Study Group. Ophthalmology 1993; 100:423.
17. Weinberg D, Dodwell DG, Fern SA. Anatomy of arteriovenous crossings in branch retinal vein occlusion. Am J Ophthalmol 1990; 109:298.
18. Duker JS, Brown GC. Anterior location of the crossing artery in branch retinal vein obstruction. Arch Ophthalmol 1989; 107:998.
19. Green WR, Chan CC, Hutchins GM, Terry JM. Central retinal vein occlusion: a prospective histopathologic study of 29 eyes in 28 cases. Trans Am Ophthalmol Soc 1981; 79:371.
20. Baseline and early natural history report. The Central Vein Occlusion Study. Arch Ophthalmol 1993; 111:1087.
21. Werther W, Chu L, Holekamp N, et al. Myocardial infarction and cerebrovascular accident in patients with retinal vein occlusion. Arch Ophthalmol 2011; 129:326.
22. Cugati S, Wang JJ, Knudtson MD, et al. Retinal vein occlusion and vascular mortality: pooled data analysis of 2 population-based cohorts. Ophthalmology 2007; 114:520.
23. Wai KM, Ludwig CA, Koo E, et al. Risk of Stroke, Myocardial Infarction, Deep Vein Thrombosis, Pulmonary Embolism, and Death After Retinal Vein Occlusion. Am J Ophthalmol 2024; 257:129.
24. Finkelstein D. Ischemic macular edema. Recognition and favorable natural history in branch vein occlusion. Arch Ophthalmol 1992; 110:1427.
25. Natural history and clinical management of central retinal vein occlusion. The Central Vein Occlusion Study Group. Arch Ophthalmol 1997; 115:486.

26. Argon laser photocoagulation for macular edema in branch vein occlusion. The Branch Vein Occlusion Study Group. Am J Ophthalmol 1984; 98:271.
27. Hayreh SS, Podhajsky PA, Zimmerman MB. Natural history of visual outcome in central retinal vein occlusion. Ophthalmology 2011; 118:119.
28. Napal JJ, Neila S, Pérez-Montes R, et al. The role of coagulation disorders in patients with retinal vein occlusion. QJM 2016; 109:97.
29. Romiti GF, Corica B, Borgi M, et al. Inherited and acquired thrombophilia in adults with retinal vascular occlusion: A systematic review and meta-analysis. J Thromb Haemost 2020; 18:3249.
30. Nobre Cardoso J, Keane PA, Sim DA, et al. Systematic Evaluation of Optical Coherence Tomography Angiography in Retinal Vein Occlusion. Am J Ophthalmol 2016; 163:93.

Topic 15264 Version 41.0

→