

Review Article

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Risk of Mortality and Cardiovascular Complications Following Retinal Vein Occlusion: A Mini-Review

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Keywords

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Abstract

Introduction: Retinal vein occlusion (RVO) is a condition in which one of the veins supplying the retina is occluded, causing acute vision changes. This event is often a signal of underlying cardiovascular disease and increases the risk for future adverse cardiovascular events.

Aim & method: This mini-review aims to provide an overview of pertinent studies investigating the relationship between RVO and increased risk of cardiovascular events and mortality, as well as treatments proposed to mitigate this risk.

Results: Pubmed database was searched for studies on RVO and risk of cardiovascular and mortality. 41 studies met criteria for inclusion. Following RVO, there was found to be a consistently elevated risk for myocardial infarction and cerebrovascular event, highest in the first year. There was also a mildly increased risk post-RVO for deep vein thrombosis and atrial fibrillation, but not for pulmonary embolism. Recent studies have also suggested that there is an increased risk for all-cause mortality after RVO. Central RVO may be more strongly associated with cardiovascular events as compared to branch RVO. To mitigate cardiovascular morbidity and mortality, treatment has been aimed at preventing atherosclerosis. Statin therapy post-RVO was found to significantly decrease cardiovascular risk.

Conclusion: While the relationship between RVO and cardiovascular disease has yet to be fully elucidated, evidence indicates that RVO is a strong predictor for future cardiovascular events, particularly acute myocardial infarction and cerebrovascular event. Ophthalmologists should work closely with primary care physicians and cardiologists following RVO to ensure proper risk assessment and treatment. These preventative interventions could reduce mortality and morbidity after RVOs.

Introduction

Retinal vein occlusion (RVO) is a condition in which one of the veins supplying the retina is occluded, causing acute vision changes and often indicating underlying vascular dysfunction. There are two subtypes of RVO, differentiated by the exact location of the blockage, whether it is the central retinal vein (CRVO) or the branch retinal vein (BRVO). RVO generally presents with painless vision loss with combination of retinal hemorrhage, cotton wool spots, optic disc swelling, and macular edema on fundus exam. There is as of yet no treatment capable of reversing RVO¹. RVO is the second most common retinal vascular disease, impacting approximately 28.06 million people globally in 2015². While it is independently an important cause of visual impairment, it is also a vital sentinel signal of cardiovascular disease, the leading cause of death globally³.

Major risk factors for cardiovascular disease identified since the mid 21st century include advanced age, obesity, atherosclerosis, hypertension, hyperlipidemia, and diabetes mellitus³. These are also the most prominent risk factors for RVO, explaining why RVO often signals underlying cardiovascular disease⁴. Underlying hypercoagulable state is a much less common risk factor for RVO, but can also contribute to disease development, especially in patients under 50 years of age¹. RVO has been well documented as a predictor of overall mortality and major adverse cardiovascular events (MACEs), including acute myocardial infarction, cerebrovascular event, atrial fibrillation, and deep vein thrombosis. Identification of RVO as an early signal of underlying cardiovascular dysfunction can help practitioners identify high risk patients earlier, allowing for proper risk stratification and more effective treatment.

Methods

A comprehensive search of Pubmed was performed, producing 249 articles published between January 1976 and October 2025. The search terms used in combination included 'retinal vein occlusion', 'stroke', 'cerebrovascular accident', 'myocardial infarction', 'mortality', 'arrhythmia', 'pulmonary embolism', and 'deep vein thrombosis'.

The articles yielded by this search were filtered on the basis of the relevancy of their abstract and title to the topic. Selection criteria focused on selecting articles that explored connections between RVO and mortality or cardiovascular disease. Of the initial 249 articles screened, 41 qualified for inclusion.

Results

Table 1 provides a detailed summary of the synthesized literature below discussing the risk of RVO to future cardiovascular events.

Cerebrovascular Event (CVE)

CVE is ischemia of the brain, most commonly caused by thrombotic occlusion of cerebral vasculature⁵. It is the leading cause of disability in the United States and the 5th leading cause of mortality⁶. There is robust evidence that RVO increases the risk of a patient experiencing a CVE. In large studies, the overall relative risk of CVE following RVO has ranged between 1.36-1.73⁷⁻¹⁵. Risk was found to be significantly elevated for both ischemic and hemorrhagic stroke^{11; 13; 15}. The incident rate ratio (IRR) was found to be elevated to 2.66 within the first 30 days post RVO (95% confidence interval (CI), 2.06-3.43)¹⁶. Wai et al also reported an increased risk of CVE immediately following RVO with relative risk of 1.61 ($p < 0.01$) at 1 year, 1.33 ($p < 0.01$) at 5 years, and 1.18 ($p < 0.01$) at 10 years¹⁰. In young patients (<50 years) with a history of RVO, this risk was more pronounced, with relative risk of 3.3 at 1 year (95% CI, 1.63-6.68), 4.0 at 3 years (95% CI, 2.00-7.98), and 4.0 at

5 years (95% CI, 2.00-7.98)¹⁷. A large meta-analysis found that 37.5% of patients with RVO experienced a CVE within 10 years (95% CI: 37.3%-37.8%). Further, the mortality rate after CVE in patients who had previously experienced an RVO was 69.0% (95% CI: 68.4%-69.5%), highlighting the importance of identifying this event early and treating the underlying cause¹⁸.

Acute Myocardial Infarction (AMI)

AMI is a leading cause of mortality and morbidity globally, affecting 523 million people and killing 18.6 million in 2019¹⁹. AMI is the occlusion of a coronary artery, most commonly caused by the rupture of an atherosclerotic plaque in the coronary arteries²⁰. Past research produced some controversy over the predictive value of RVO for AMI^{7; 16; 21-27}. However, more recent large cohort and meta-review studies have strongly demonstrated the association between RVO and increased risk of AMI^{8-10; 28; 29}. These studies reported a relative risk of AMI after RVO of 1.13-1.28^{8-10; 28-30}. Wai et. al reported an increased AMI risk after RVO that was most notable at shorter time frames, with relative risk of 1.26 at 1 year ($p < 0.01$), 1.13 at 5 years ($p < 0.01$), and no significant risk observed at 10 years¹⁰. Given the elevated risk profile, patients should be monitored for AMI most closely in the first year after RVO.

Atrial Fibrillation (AF), Pulmonary Embolism (PE), and Deep Vein Thrombosis (DVT)

AF is an abnormal rhythm of the heart, often caused by underlying structural heart disease. It is the most common arrhythmia and the leading cardiac cause of stroke³¹. PE is an obstructing thrombosis in the lung vasculature³², whereas DVT is a blood clot formed in the deep veins of the leg, occluding blood flow³³. There have been conflicting results describing the relationship between AF and RVO. While one study described significantly increased risk of developing AF post-RVO (HR, 1.35; 95% CI, 1.09-1.67)³⁴, another similar study found no increased rate of AF development after RVO diagnosis (95% CI; 0.74-1.39, $p = 0.920$)³⁵. There has not yet been strong evidence supporting increased risk of PE after RVO, though there is early work supporting RVO as a predictor of DVT¹⁰. PE alone has not been found to be increased post-RVO, but one study found, in a sample of patients under 50 years old with history of RVO, relative risk of DVT or PE was greatly elevated to 5.5 at 1 year (95% CI, 2.81-10.77), 6.4 at 3 years (95% CI, 3.29-12.44), and 5.67 at 5 years (95% CI, 3.08-10.44)¹⁷. Wai et. al found a mildly elevated risk of DVT alone at one year post RVO (relative risk = 1.65, $p < 0.01$) in a cohort of over 90,000 patients. This effect, however, disappeared at 5 and 10 years¹⁰. These differing effects between age groups may indicate a more prolonged and elevated risk for thrombosis in young patients presenting with RVO, likely due to the fact that young patients with RVO are more likely

Table 1: Brief summation of the results of included studies

| | Study | Study type | Country | Average age participants (RVO+/control) | Follow up period | Sample size (RVO+/control) | Events (RVO+/control) | Risk (risk measure (95% CI)) |
|----------------------------------|---|---|--|---|--------------------------------------|----------------------------|---|--|
| Cerebrovascular Event | Bakhom et al. ¹² , 2023 | Cross-sectional | United States | 71/59 | 6 years | 925/79829 | 116/3777 | OR: 1.73 (1.40–2.12) |
| | Capua et al. ²³ , 2009 | Retrospective cohort | Italy | 54/54.4 | 8 years | 45/145 | 4/3 | OR: 5.44 (1.16-25.41) |
| | Chen et al. ¹¹ , 2018 | Retrospective cohort | Taiwan | 61.8 | Average 4.9/5.4 years | 22919/114595 | 5481/22235 | Adjusted HR: 1.36 (95% CI: 1.32–1.40) |
| | Hashemi et al. ¹⁵ , 2024 | Meta-analysis | Taiwan, United States, Denmark, Italy, South Korea | <40 to >80 | 1.5 to 13 years | 86300/342352 | 8466/33178 | Pooled RR: 1.38 (1.34–1.41) |
| | Li et al. ¹⁴ , 2016 | Meta-analysis | USA, Australia, China, Denmark, Korea | >18 | 1.5-12 | 6408/31063 | 431/unspecified | Pooled RR: 1.50 (1.19–1.90) |
| | Park et al. ¹⁶ , 2015 | self-controlled case series | South Korea | 61.6/68.1 | 1 year | 44,603 | 1016/113 | IRR Ischemic stroke: 1.35 (1.08–1.69), IRR Hemorrhagic stroke: 1.40 (0.81–2.40) |
| | Rim et al. ¹³ , 2015 | Retrospective cohort | South Korea | <40 to >80 | 9 years | 1031/5074 | 173/543 | HR: 1.48 (1.24-1.76) |
| | Wai et al. ¹⁰ , 2023 | Retrospective cohort | United states and globally | 68.1 | Up to 10 years | 45303/45303 | 4992/4231 | RR 1.18 (1.10, 1.27) |
| | Werther et al. ⁷ , 2011 | Retrospective cohort | United States | 64 | Up to 4 years (average 550/506 days) | 4500/13500 | 78/96 | adjusted RR, 1.72 (1.27-2.34) |
| | Wu et al. ⁸ , 2018 | Meta-analysis | Denmark, Taiwan, Singapore, United States, Australia, Italy, South Korea | <50 to >80 | Average >1 year | 60069/414397 | 1349/22777 | RR: 1.45 (1.31–1.60) |
| | Zhang et al. ¹⁷ , 2025 | Retrospective cohort | United states and globally | 18-50 (average 33.8/33.8) | Up to 5 years | 2731/2731 | 40/≤10 | Adjusted RR: 4 (2.004–7.982) |
| Zhong et al. ⁹ , 2016 | Meta-analysis | Denmark, Taiwan, United States, Australia, Italy, South Korea | <40 to >80 | 1.5 to 12 years | 13684/169451 | 1258/9971 | Adjusted RR: 1.50 (1.32-1.69) | |
| Acute Myocardial Infarction | Capua et al. ²³ , 2009 | Retrospective cohort | Italy | 54/54.4 | 8 years | 45/145 | 8/4 | No significant difference was noted between cohorts |
| | Chen et al. ²⁹ , 2017 | Retrospective cohort | Taiwan | 62.4 | 5.52/5.55 | 37921/113763 | 1240/2616 | Adjusted HR: 1.21 (1.13 to 1.30) |
| | Frederiksen et al. ³⁰ , 2023 | Retrospective cohort | Denmark | 71.8 | Median 15.5 years | 15665/4179116 | 2983/59743 | Adjusted HR: 1.13 (1.09-1.17) |
| | Hu et al. ²¹ , 2008 | Retrospective cohort | Taiwan | <50 to >70 | 3 years | 591/2955 | 11/23 | No significant difference was noted between cohorts |
| | Park et al. ¹⁶ , 2015 | self-controlled case series | South Korea | 61.6/68.1 | 1 year | 44,603 | 172/36 | IRR: 1.28 (0.81–2.04) |
| | Rim et al. ²⁸ , 2016 | Retrospective cohort | South Korea | <50 to >80 | 12 | 1677/8367 | 128/444 | HR: 1.25 (1.02-1.52) |
| | Wai et al. ¹⁰ , 2023 | Retrospective cohort | United states and globally | 68.1 | 10 years | 45303/45303 | 4439/4186 | Adjusted RR: 1.06 (0.99-1.14) |
| | Werther et al. ⁷ , 2011 | Retrospective cohort | United States | 64 | Up to 4 years (average 550/506 days) | 4500/13500 | 58/125 | Adjusted RR: 1.03 (0.75-1.42) |
| | Wu et al. ⁸ , 2018 | Meta-analysis | Denmark, Taiwan, Singapore, United States, Australia, Italy, South Korea | <50 to >80 | Average >1 year | 60069/414397 | 1511/7872 | RR: 1.26 (1.17–1.37) |
| | Zhong et al. ⁹ , 2016 | Meta-analysis | Denmark, Taiwan, United States, Australia, Italy, South Korea | <40 to >80 | 1.5 to 12 years | 16162/170644 | 539/4812 | Adjusted RR: 1.28 (1.17-1.41) |
| | Christiansen et al., 2018 | Retrospective cohort | Denmark | 71.4 | Up to 15 years | 1368/6840 | 1.74/1.22 incident cases per 100 person-years | HR: 1.26 (1.04–1.53) |
| Rim et al. ³⁴ , 2016 | Retrospective cohort | South Korea | <50 to >80 | Average 7.4 years (up to 12) | 1801/8930 | 117/353 | HR: 1.35 (1.09–1.67) | |

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|-----------------------------|---|----------------------|--|-------------------------------|--------------------------------------|----------------|---|--|
| Acute Myocardial Infarction | Wai et al. ¹⁰ , 2023 | Retrospective cohort | United states and globally | 68.1 | 10 years | 45303/45303 | 4439/4186 | Adjusted RR: 1.06 (0.99-1.14) |
| | Werther et al. ⁷ , 2011 | Retrospective cohort | United States | 64 | Up to 4 years (average 550/506 days) | 4500/13500 | 58/125 | Adjusted RR: 1.03 (0.75-1.42) |
| | Wu et al. ⁸ , 2018 | Meta-analysis | Denmark, Taiwan, Singapore, United States, Australia, Italy, South Korea | <50 to >80 | Average >1 year | 60069/414397 | 1511/7872 | RR: 1.26 (1.17–1.37) |
| | Zhong et al. ⁹ , 2016 | Meta-analysis | Denmark, Taiwan, United States, Australia, Italy, South Korea | <40 to >80 | 1.5 to 12 years | 16162/170644 | 539/4812 | Adjusted RR: 1.28 (1.17-1.41) |
| Atrial Fibrillation | Christiansen et al., 2018 | Retrospective cohort | Denmark | 71.4 | Up to 15 years | 1368/6840 | 1.74/1.22 incident cases per 100 person-years | HR: 1.26 (1.04–1.53) |
| | Rim et al. ³⁴ , 2016 | Retrospective cohort | South Korea | <50 to >80 | Average 7.4 years (up to 12) | 1801/8930 | 117/353 | HR: 1.35 (1.09–1.67) |
| Deep Venous Thrombosis | Wai et al. ¹⁰ , 2023 | Retrospective cohort | United states and globally | 68.1 years | 10 years | 45303/45303 | 2098/1993 | Adjusted RR: 1.05 (95% CI, 0.94, 1.18) |
| | Zhang et al. ¹⁷ , 2025 | Retrospective cohort | United states and globally | 33.8/33.8 (range 18-50) | Up to 5 years | 2731/2731 | DVT or PE 68/12 | Adjusted RR for DVT or PE: 5.667 (95% CI, 3.075–10.444) |
| Pulmonary Embolism | Wai et al. ¹⁰ , 2023 | Retrospective cohort | United states and globally | 68.1 ± 14.3 years | 10 years | 45303/45303 | 1540/1626 | Adjusted RR: 0.85 (0.83-1.08) |
| | Zhang et al. ¹⁷ , 2025 | Retrospective cohort | United states and globally | 18-50 (average 33.8/33.8) | Up to 5 years | 2731/2731 | DVT or PE 68/12 | Adjusted RR for DVT or PE: 5.667 (3.075–10.444) |
| Mortality | Bertelsen et al. ²⁷ , 2014 | Retrospective cohort | United States | <50 to >80 | 5.1/5.7 | 439/2195 | 26/94 | Adjusted HR: 1.45 (1.19-1.76) |
| | Chen et al. ¹¹ , 2018 | Retrospective cohort | Taiwan | Avg 61.8 ± 13.0 SD | Avg 4.9/5.4 | 22919/114595 | 3111/15106 | HR: 1.03 (0.99–1.07) |
| | Cugati et al. ²⁶ , 2007 | Retrospective cohort | United states | 43-97 (61.8) | 2 years | 96/8282 | 25/1416 | Adjusted HR: 1.2 (0.8–1.8) |
| | Frederiksen et al. ³⁰ , 2023 | Retrospective cohort | Denmark | Average 71.8 years old | Median 15.5 years | 15665/ 4179116 | 5216/97889 | Adjusted HR: 1.00 (0.97 to 1.03) |
| | Voigt et al. ³⁷ , 2025 | Retrospective cohort | Germany | Average age 52.4 years old | Up to 15 years | 67/6378 | unavailable | Adjusted HR BRVO: 2.27 (1.08-3.0) Adjusted HR CRVO: 3.83 (1.95-7.9) |
| | Wai et al. ¹⁰ , 2023 | Retrospective cohort | United states and globally | average age 68.1 ± 14.3 years | 10 years | 45,303/45,303 | 29424/27150 | Adjusted RR: 1.08 (1.06, 1.10) |
| | Wu et al. ⁸ , 2018 | Meta-analysis | Denmark, Taiwan, Singapore, United States, Australia, Italy, South Korea | <50 to >80 | Average >1 year | 60,069/414,397 | 3546/14403 | RR: 1.36 (1.02–1.81) |

to have underlying thrombophilic pathology¹. A large meta-analysis found a raw event rate of 0.05% of DVT following RVO¹⁸. While very rare following RVO, these results indicate that DVT may still warrant monitoring post-RVO, especially in the first year and in high-risk populations.

All-Cause Mortality

There have been conflicting reports as to whether RVO predicts increased mortality. Several studies prior to 2016 observed no significant association between RVO and increased all-cause mortality over follow up periods ranging from 2-31 years^{11; 26; 27; 36}. One study in this period reported an increase in mortality in patients with CRVO (HR, 1.45; 95% CI, 1.19-1.76). However, when the analysis was adjusted for overall occurrence of cardiovascular disease and diabetes, the cohorts were comparable (HR, 1.19; 95% CI, 0.96-1.46)²⁷. Newer evidence indicates that RVO may indicate increased risk of mortality over a follow up period ranging from 5-19 years, even when adjusting for cardiovascular risk factors and diabetes^{10; 30; 37}. Wai et al observed an increased risk of all-cause mortality in RVO patients as compared to propensity score matched controls without RVO at 1 year (RR = 1.30, P < .01), 5 years (RR = 1.22, P < .01), and 10 years (RR = 1.08, P < .01)¹⁰. This risk was strongest within the first year, aligning with other studies that showed increased CVE and MI risk within the first year^{10; 16}. This indicates a need for close monitoring and counseling post-RVO, especially within the first year. More research is needed to further clarify the relationship between RVO and mortality to better understand underlying factors and develop effective interventions.

Underlying Pathology

RVO is caused by vascular inflammation and damage, causing narrowing and compression of the retinal veins and atherosclerotic embolism. Though RVO is caused by obstruction of a retinal vein, it has been strongly associated with underlying atherosclerotic pathology. Patients who suffered an RVO had significantly greater arterial stiffness as measured by cardio-ankle vascular index³⁸. In rare cases, typically in younger patients (<50 years), it can also be caused by clotting disorders, creating an embolism that blocks a retinal vein^{1; 4}. In a study that investigated the impact of all known thrombophilic risk factors on RVO, only hyperhomocysteinemia and anticardiolipin antibodies were found to increase risk for RVO (8.9 and 3.9 odds ratio (OR) respectively), while MTHFR mutation, factor V Leiden mutation, and prothrombin gene mutation were found to have non-significant impact. The conditions that were found to increase risk were associated with both venous thrombosis and arterial vascular disease, while the conditions not found to be associated were linked to venous thrombosis only, suggesting that atherosclerotic pathology likely underlies the increase in RVO risk³⁹.

This association with atherosclerosis likely explains some differences observed between CRVO and BRVO. It has been shown that CRVO patients had higher risk of mortality and AMI than BRVO at up to 10 years post RVO^{10; 29; 40}. One study found that BRVO did not change risk of mortality as compared to controls³⁶. Another observed an increase in mortality in patients with CRVO, however, this difference was non-significant when cardiovascular disease and diabetes were controlled for, supporting the theory that the increased risk of mortality and AMI in CRVO patients is due to underlying atherosclerotic disease, which may be more predictive of CRVO than BRVO²⁷.

The relative weakness of RVO as a predictor of DVT and PE as compared to its strong association with AMI and CVE risk is likely because the primary causative mechanism of DVT and PE is hypercoagulability and venous stasis. The conditions which have been more strongly associated with RVO, such as AMI and CVE, have underlying arterial atherosclerotic pathology, the most common cause of RVO.

Changing the treatment paradigm

This work demonstrates that RVO is an important "sentinel signal" communicating increased patient risk for major adverse cardiac events, most prominently AMI and CVE. It is vital for ophthalmologists to work closely with primary care physicians, cardiologists, and neurologists to communicate these patients' increased risk profile, allowing for optimization of their cardiovascular management plan accordingly.

Recognition of the shared underlying pathology of RVO and MACEs could help to guide medical therapy. Aligned with the theory that RVO is primarily due to atherosclerotic rather than thrombophilic pathology, a systematic review recommended against universal thrombophilia screening of patients with RVO⁴¹. Anti-thrombotic agents were not found to significantly affect mortality in patients following RVO⁴². Additionally, a meta-analysis of anti-platelet therapy following RVO did not find a significant decrease in the incidence of cardiovascular events or RVO recurrence⁴³. Past work has suggested that retinal vein inflammation is a primary cause of RVO in young patients, whereas atherosclerosis is a major causative factor in older patients^{44; 45}. Therefore, screening for thrombophilia and initiation of anti-thrombotic therapy is most applicable to young patients (<50 years) presenting with RVO.

Conversely, statin therapy to address patients' underlying atherosclerotic risk factors has been the only intervention associated with a lower risk of cardiovascular events after RVO (adjusted OR, 0.604; 95% CI, 0.557 to 0.655)⁴⁶. As statin exposure length increased post-RVO, adjusted OR of cardiovascular events decreased. When compared to a statin time course post-RVO of less than 90

days, the OR of cardiovascular events at 91-365 days was 0.832 (95% CI, 0.747 to 0.926), at 1-2 years was 0.579 (95% CI, 0.509 to 0.658), and at greater than 2 years was 0.502 (95% CI, 0.451 to 0.560)⁴⁶. However, it is unclear if this reduction is due to increased efficacy of statin therapy over time or decreasing risk of CVE and MI over time after RVO^{10, 16}. The risk reduction observed with statin therapy after RVO was significant for both CVE (adjusted OR, 0.563; 95% CI, 0.513 to 0.617) and AMI (adjusted OR, 0.782; 95% CI, 0.657 to 0.932)⁴⁶. Though more work is necessary to clarify these pathways, modifying patient therapy to address their cardiovascular disease post-RVO may reduce overall mortality and morbidity.

Conclusion

Robust evidence demonstrates that RVO, especially CRVO, is an important sign of increased risk for all-cause mortality, AMI, and CVE, possibly signaling dangerous underlying atherosclerotic disease. It is important that ophthalmologists recognize this signal and work with cardiologists to identify and manage underlying cardiovascular disease. Given the frequently atherosclerotic etiology of RVO, statin therapy is more effective than anti-thrombotic agents in preventing MACEs following RVO. Future work should focus on clarifying the underlying pathology connecting RVO and cardiovascular disease and developing effective treatment regimens to prevent cardiovascular complications following RVO.

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None

Conflicts of interest

The authors report no relevant conflicts of interest

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