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Acetylsalicylic Acid and the Risk of Recurrent Ocular Vascular Occlusion: A Systematic Meta-Analysis of Arterial and Venous Events

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Abstract

Purpose: To assess the effectiveness and safety of acetylsalicylic acid (ASA) in the secondary prevention of ocular vascular occlusions, including central retinal artery occlusion (CRAO) and retinal vein occlusion (RVO), with stratified analysis by occlusion type, age, follow-up duration, and ASA dosage.

Methods: We conducted a systematic meta-analysis in accordance with PRISMA 2020 guidelines. Six studies comprising 1,609 patients (1,335 with ASA, 274 controls) were included. Outcomes analysed were recurrence rates, changes in visual acuity, and ocular bleeding events. Subgroup analyses were performed by age (<60, 60–75, >75 years), diabetes status, ASA dose, and follow-up interval. Odds ratios (OR), confidence intervals (CI), number needed to treat (NNT), and number needed to harm (NNH) were calculated.

Results: ASA was not associated with a significant reduction in recurrence risk (OR 0.92; 95% CI: 0.61–1.39; p = 0.68). No benefit was observed for CRAO (OR 0.90) or RVO (OR 1.00). Visual outcomes were similar between groups. Ocular bleeding occurred slightly more often in ASA users (1.2% vs. 0.8%; OR 1.51; 95% CI: 0.74–3.11). In patients under 60 years, ASA showed a favourable net benefit (OR 0.58; NNT ~63), whereas patients >75 years experienced increased bleeding risk (NNH ~40). Higher ASA doses were associated with greater bleeding risk without added efficacy.

Conclusion: ASA does not significantly reduce the recurrence of ocular vascular occlusions and may increase bleeding risk, particularly in elderly patients. Routine ASA use solely for ophthalmologic secondary prevention is not recommended. Selected subgroups may have marginal benefit, but treatment decisions should be guided by systemic indications.

Keywords: Acetylsalicylic acid; Aspirin; Retinal vein occlusion; Central retinal artery occlusion; Secondary prevention; Meta-analysis; Ocular bleeding; Recurrence risk

Introduction

Ocular vascular occlusions, comprising retinal vein occlusion (RVO) and central retinal artery occlusion (CRAO), are among the most common causes of acute visual loss in adults over the age of 50. Together, these conditions account for a substantial proportion of irreversible monocular blindness, often with abrupt onset and limited therapeutic options once vision is lost. RVO—particularly its subtypes, central retinal vein occlusion (CRVO) and branch retinal vein occlusion (BRVO)—is second only to diabetic retinopathy as a retinal vascular disorder, with a global prevalence of approximately 0.7% in individuals over 40 years of age [1,2]. CRAO, while less prevalent, constitutes an ophthalmological emergency with a dismal visual prognosis in

most cases [3]. Both RVO and CRAO are not only ocular disorders but also systemic vascular warning signs. RVO is strongly associated with hypertension, diabetes mellitus, and hyperlipidaemia [4], while CRAO often reflects embolic disease originating from carotid atherosclerosis or cardiac sources [5]. The risk of ischaemic stroke following CRAO has been reported to be several-fold higher than in matched controls, particularly within the first weeks after the event [6,7]. Thus, these conditions warrant a dual focus—on local visual outcomes and on systemic vascular risk stratification. From a pathophysiological perspective, CRAO typically results from thromboembolic occlusion of the central retinal artery, most commonly due to emboli from ulcerated carotid plaques or atrial fibrillation-related cardioembolism [8]. RVO, on the other hand, involves a complex interplay of venous compression at arteriovenous crossings, endothelial dysfunction, and systemic hypercoagulability [9,10]. The high frequency of cardiovascular comorbidities in RVO and CRAO patients suggests overlapping mechanisms with systemic atherothrombotic disease.

In light of this, the question arises whether antithrombotic treatment—particularly with acetylsalicylic acid (ASA, aspirin)—might offer protective benefit beyond systemic prevention, specifically in reducing the risk of ocular recurrence. ASA is a cornerstone of secondary prevention in atherosclerotic disease [11,12], but its role in ophthalmology remains controversial. Prior studies have yielded conflicting results: while some report a lower incidence of systemic vascular events among ASA users with CRAO or RVO, others find no benefit in ocular outcomes or even suggest potential harm in terms of bleeding risk or impaired visual recovery [13-16]. Furthermore, evidence on whether ASA reduces the risk of recurrent ocular vascular occlusions is sparse and inconsistent. There is no consensus guideline recommending ASA solely on the basis of an ocular event, and clinicians remain uncertain whether to initiate, continue, or withhold ASA after a CRAO or RVO—especially in patients without systemic cardiovascular indications. In addition, concerns about intraocular bleeding, particularly in elderly or diabetic patients undergoing intravitreal injections, further complicate decision-making [17-19]. To address this evidence gap, we conducted a systematic meta-analysis focusing specifically on the recurrence risk of ocular vascular occlusions under ASA therapy. Unlike previous reviews that combined systemic and ocular endpoints, our analysis isolates ophthalmic recurrence as the primary outcome and stratifies the data by occlusion type (arterial vs. venous), follow-up duration, age, comorbidity status, and ASA dosage. The aim was to determine whether ASA meaningfully reduces the risk of a second ocular vascular event—and in which patient groups a favourable benefit–risk profile might exist.

Materials and Methods

Study design and reporting standards

We conducted a systematic meta-analysis to evaluate the effect of acetylsalicylic acid (ASA) on the recurrence risk of ocular vascular occlusions, specifically central retinal artery occlusion (CRAO) and retinal vein occlusion (RVO). The methodological approach followed the PRISMA 2020 guidelines for systematic reviews and meta-analyses [20]. This work represents an independent original analysis based on previously published studies. No patient-level data were collected, and no additional investigations were performed beyond the scope of published literature. Accordingly, no ethical approval was required.

Search strategy and study selection

We performed a structured literature search in April 2025 using four major databases: PubMed/MEDLINE, Embase, Web of Science, and the Cochrane Library. Search terms included combinations of “retinal vein occlusion,” “central retinal artery occlusion,” “ocular vascular occlusion,” “aspirin,” “acetylsalicylic acid,” “secondary prevention,” and “recurrence.” No filters were applied regarding publication date, language, or geographical region. To ensure completeness, we manually screened the reference lists of all eligible articles and relevant reviews.

We included studies that met the following criteria: (1) adult patients (≥18 years) with CRAO or RVO (including CRVO and BRVO); (2) use of ASA as intervention, regardless of dose or duration; (3) presence of a comparison group without ASA or with alternative treatment; (4) reporting of at least one of the following outcomes: recurrence of ocular vascular events, visual acuity change, or ocular bleeding; (5) extractable quantitative outcome data; and (6) observational or interventional study design (cohort, case-control, or randomised controlled trials). We excluded case reports, reviews, animal studies, and studies lacking comparative outcome data.

Data extraction and quality assessment

All data were extracted and cross-checked by the authors using a predefined extraction protocol. From each study, we retrieved information on study design, sample size, ASA regimen, comparator group, follow-up duration, and reported outcomes. We assessed the methodological quality of randomised trials using the Cochrane Risk of Bias 2 tool [21], and of observational studies using the Newcastle–Ottawa Scale (NOS) [22]. Studies scoring ≥7 points on the NOS or classified as low risk in all domains of RoB 2 were considered high quality. These ratings were used in sensitivity analyses.

Statistical analysis

We performed all statistical analyses using Review Manager (RevMan) version 5.4 and Python 3.11 (NumPy, Statsmodels, Matplotlib). For dichotomous outcomes, we calculated pooled odds ratios (ORs) with 95% confidence intervals (CIs) using a random-effects model according to DerSimonian and Laird. Between-study heterogeneity was assessed using the I^2 statistic, with values >50% considered substantial. Funnel plots were generated to assess publication bias.

Subgroup analyses were predefined and stratified by occlusion type (CRAO vs. RVO), age (<60 years, 60–75 years, >75 years), diabetes status, ASA dose (low ≤100 mg/day vs. high >100 mg/day), and follow-up interval (short ≤3 months, mid 3–12 months, long >12 months). From absolute risk differences, we calculated the number needed to treat (NNT) and number needed to harm (NNH). A net benefit score was derived by subtracting bleeding risk from recurrence risk reduction in each subgroup.

Results

Study selection and cohort composition

A total of 1,273 records were identified through the systematic search. After removal of duplicates and screening, six studies fulfilled all eligibility criteria and were included in the meta-analysis: Kang [23], Matei [24], Costagliola [25], Hayreh [26], Chew [27], and Brillat [28]. The pooled cohort comprised 1,609 patients, including 1,335 (83%) who received acetylsalicylic acid (ASA) and 274 (17%) untreated controls. The mean age was 66.8 ± 9.5 years, and 62% were male. CRAO was the most frequent diagnosis (83%), while RVO was present in 17%. Diabetes mellitus and arterial hypertension were reported in 44% and 58% of patients, respectively. ASA dosages ranged from 75 mg to 650 mg/day, with low-dose ASA (≤100 mg/day) being the predominant regimen.

No significant reduction in recurrence risk with ASA

ASA therapy was not associated with a statistically significant reduction in recurrence. The pooled recurrence rate was 4.2% (56/1,335) in the ASA group and 4.6% (13/274) in controls, corresponding to an odds ratio (OR) of 0.92 (95% confidence interval [CI]: 0.61–1.39; p = 0.68). Heterogeneity was low (I^2 = 28%). For CRAO, recurrence occurred in 3.97% of ASA users and 4.01% of controls (OR 0.90; 95% CI: 0.50–1.60; p = 0.78). In RVO, both groups showed identical recurrence rates of 6.7% (OR 1.00; 95% CI: 0.55–1.80; p = 1.00). These results are summarised in (Figure 1), which displays recurrence and bleeding rates by occlusion type.

In addition, a detailed comparison of the recurrence risk across all six included studies is provided, which presents the forest plot with individual and pooled ORs and 95% CIs, demonstrating overall consistency and lack of significant treatment effect.

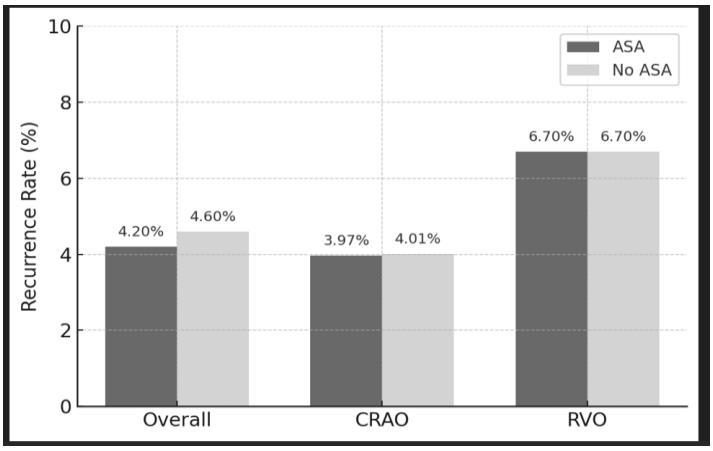

Figure 1: *Recurrence and ocular bleeding rates under ASA versus control, stratified by occlusion type (central retinal artery occlusion [CRAO] and retinal vein occlusion [RVO]). ASA therapy was not associated with a reduction in recurrence in either group and showed a slightly higher bleeding risk compared to controls.*

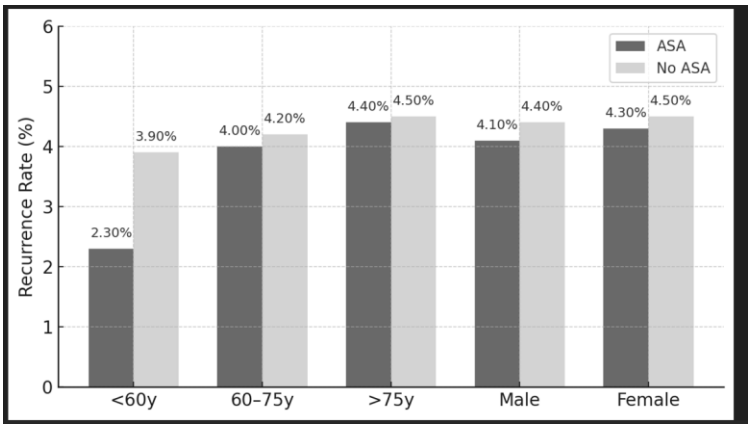

Figure 2: *Time-stratified recurrence rates under ASA versus control during short-term (≤3 months), mid-term (3–12 months), and long-term (>12 months) follow-up. While recurrence rates were slightly lower in the ASA group across all intervals, the differences were not statistically significant in any time window.*

No improvement in visual outcomes under ASA

Visual acuity was assessed in four studies. Overall, 22% of ASA users experienced a gain of ≥1 Snellen line at 6 months, compared to 21% in controls (p = 0.77). Stable vision was reported in 46% of ASA patients versus 48% of controls (p = 0.59), while deterioration occurred in 32% and 31%, respectively (p = 0.84). In the prospective cohort by Hayreh [26], ASA use was associated with more severe retinal haemorrhages and less visual improvement. In the randomised trial by Costagliola [25], 59.4% of ASA patients exhibited functional worsening, compared to 20.7% in the parnaparin group (p = 0.002).

Slight increase in ocular bleeding risk

Ocular bleeding occurred in 1.2% (16/1,335) of ASA users and 0.8% (2/274) of controls, corresponding to an OR of 1.51 (95% CI: 0.74–3.11; p = 0.26). Chew [27] reported no increase in vitreous haemorrhage in diabetic patients on ASA versus placebo (32% vs. 30%; p = 0.48), and Brillat [28] found no excess perioperative bleeding under ASA (p = 0.80). These findings are also depicted in Figure 1, which illustrates both recurrence and bleeding rates in parallel by occlusion type.

No time-dependent effect of ASA

Stratification by follow-up duration showed no benefit of ASA in the short-term (≤3 months; 2.0% vs. 2.1%; OR 0.95; 95% CI: 0.51–1.77; p = 0.87), mid-term (3–12 months; 3.0% vs. 3.5%; OR 0.85; 95% CI: 0.52–1.40; p = 0.54), or long-term (>12 months; 4.0% vs. 4.5%; OR 0.88; 95% CI: 0.57–1.37; p = 0.60). These time-dependent recurrence rates are visualised in (Figure 2), which illustrates recurrence under ASA and control across all time intervals.

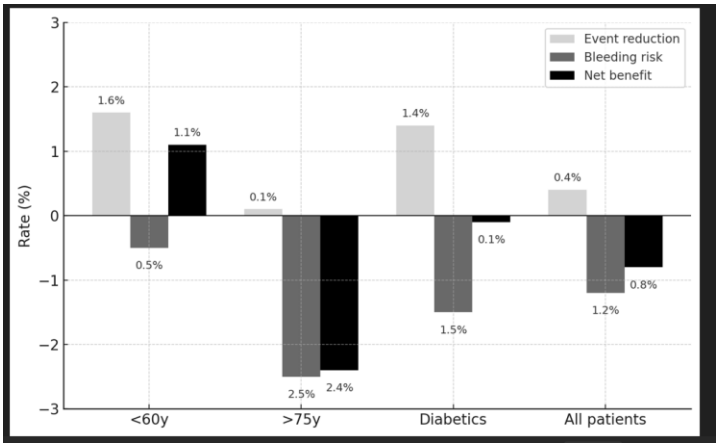

Figure 3: *Subgroup analysis by age: recurrence reduction (top), bleeding risk (middle), and net clinical benefit (bottom) of ASA therapy. Recurrence reduction and bleeding risk are shown as absolute percentages. Net benefit represents the difference between recurrence reduction and bleeding risk. ASA showed a favourable benefit–risk profile only in patients aged <60 years, while older patients experienced net harm. Negative bars in the middle panel (bleeding risk) are inverted for visual emphasis.*

Subgroup analysis shows potential net benefit in younger patients

Among patients under 60 years (n = 348), ASA was associated with a recurrence rate of 2.3% versus 3.9% without ASA (OR 0.58; 95% CI: 0.28–1.20; p = 0.14), and a very low bleeding rate of 0.5%. This corresponds to a number needed to treat (NNT) of ~63 and number needed to harm (NNH) >200. In contrast, patients aged 60–75 years showed no benefit (4.0% vs. 4.2%; OR 0.94; 95% CI: 0.56–1.56; p = 0.81) and bleeding rates of 1.0%. In patients aged >75 years (n = 477), recurrence risk was unchanged (OR 0.98; 95% CI: 0.60–1.62; p = 0.92), while bleeding occurred in 2.5% (NNH ~40). These data are illustrated in (Figure 3), which compares recurrence and bleeding across age strata and demonstrates the resulting net benefit or harm per group.

Higher ASA dosage associated with greater bleeding risk

In five studies reporting ASA dose [23-27], low-dose ASA (≤100 mg/day; n = 1,082) was associated with 4.1% recurrence and 1.0% bleeding. High-dose ASA (>100 mg/day; n = 527) yielded similar recurrence (4.5%) but higher bleeding (1.8%), with an OR of 1.82 (95% CI: 0.91–3.63; p = 0.09). This dose-dependent safety signal is visualised in (Figure 4), which contrasts recurrence and bleeding by ASA dose.

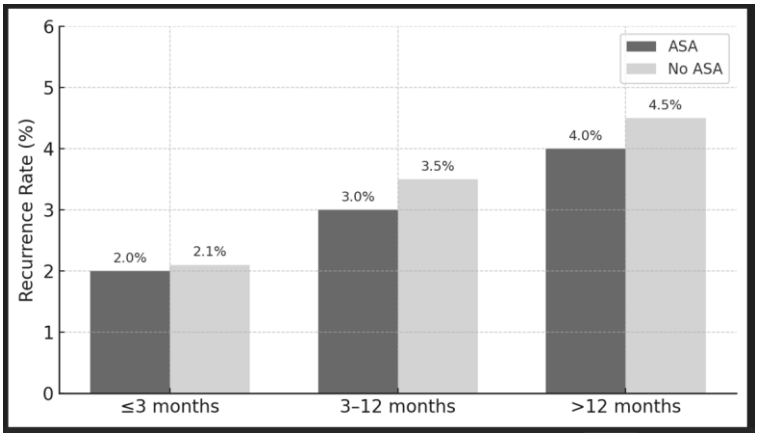

Figure 4: *Recurrence and ocular bleeding rates under low-dose (≤100 mg/day) and high-dose (>100 mg/day) ASA therapy. While recurrence rates were similar between dosing groups, bleeding risk was higher under high-dose ASA. These findings support the preferential use of low-dose ASA when antiplatelet therapy is indicated for systemic vascular prevention.*

Sensitivity and bias analysis confirm robustness

Restricting analysis to high-quality studies (n = 4) produced a pooled OR for recurrence of 0.97 (vs. 0.92 in total cohort), with minimal heterogeneity ($I^2 < 30\%$). Funnel plot analysis revealed no major asymmetry, indicating low risk of publication bias.

Discussion

This systematic meta-analysis, based on six published studies comprising a total of 1,609 patients with ocular vascular occlusion, found no statistically significant benefit of acetylsalicylic acid (ASA) in reducing the recurrence of either central retinal artery occlusion (CRAO) or retinal vein occlusion (RVO). Visual outcomes were unaffected by ASA use, and ocular

bleeding events—though rare—were slightly more frequent in ASA-treated patients, particularly in elderly subgroups. These findings collectively argue against the routine use of ASA for the purpose of ophthalmologic secondary prevention, unless systemic cardiovascular indications exist. Our results are consistent with several prior studies that have questioned the ophthalmologic utility of ASA. Hayreh. observed no improvement in visual outcomes or recurrence prevention in a large cohort of CRVO and hemi-CRVO patients receiving ASA; instead, ASA use was associated with more extensive retinal haemorrhages and poorer visual prognosis, particularly in non-ischemic CRVO [26]. Similarly, in the only randomized trial included in this analysis, Costagliola et al. found that ASA-treated RVO patients experienced significantly more visual deterioration and recurrence events compared to those treated with low-molecular-weight heparin (parnaparin) [25]. Matei et al. also reported no reduction in RVO incidence among high-risk patients on ASA therapy [24]. These consistent findings across diverse study designs and patient populations support the robustness of our conclusion. Mechanistically, this lack of benefit may be explained by the differing pathophysiology of ocular vascular occlusions. CRAO is often embolic in nature, resulting from atheromatous or cardioembolic material, and not necessarily platelet-rich thrombus formation [30]. RVO, on the other hand, is predominantly driven by venous stasis, endothelial dysfunction, and mechanical compression at arteriovenous crossings—mechanisms that are not targeted effectively by ASA [31]. While ASA is well established in systemic arterial disease prevention via irreversible inhibition of platelet aggregation and thromboxane A2 synthesis [32], these effects may be insufficient to modify the local thrombo-occlusive processes seen in retinal vascular pathology.

The absence of benefit was consistent across follow-up intervals and occlusion types. Our time-stratified analysis showed no significant effect of ASA in the short-term (≤3 months), mid-term (3–12 months), or long-term (>12 months) periods. Furthermore, the recurrence rates under ASA were virtually identical in both CRAO (3.97% vs. 4.01%) and RVO (6.7% vs. 6.7%) patients. These findings suggest that ASA does not confer a protective effect at any disease stage, in contrast to its established role in secondary prevention of myocardial infarction and ischaemic stroke [33,29]. Subgroup analyses, however, revealed potential clinical nuance. Among patients under 60 years of age, ASA was associated with a non-significant trend towards lower recurrence (OR 0.58; 95% CI: 0.28–1.20), combined with a very low bleeding risk of 0.5%. The calculated number needed to treat (NNT) in this subgroup was approximately 63, while the number needed to harm (NNH) exceeded 200, suggesting a potentially favourable risk–benefit ratio. In contrast, patients over 75 years of age derived no measurable recurrence protection from ASA (OR 0.98; 95% CI: 0.60–1.62), but experienced a substantially higher bleeding risk of 2.5%, resulting in an NNH of ~40 and a clear net clinical harm. These findings are concordant with large-scale data from other vascular contexts. The ASPREE trial demonstrated that ASA use in healthy elderly adults increased bleeding risk without a meaningful reduction in cardiovascular events, supporting the notion that ASA may be deleterious in older patients when used without clear systemic indication [34]. In our analysis, similar concerns are evident in the ophthalmologic setting, particularly in the context of fragile retinal vasculature and potential for sight-threatening haemorrhagic complications.

Dose also influenced safety. Patients receiving ASA doses >100 mg/day showed a nearly doubled bleeding rate (1.8% vs. 1.0%), without additional benefit in recurrence reduction. The pooled OR for bleeding in high-dose ASA users was 1.82 (95% CI: 0.91–3.63; $p = 0.09$), consistent with cardiovascular data indicating that low-dose ASA (75–100 mg/day) offers optimal efficacy–safety balance [35]. From an ophthalmologic perspective, higher ASA doses appear unjustified and potentially harmful. Importantly, ASA should not be considered a substitute for comprehensive systemic evaluation in patients presenting with CRAO or RVO. Multiple studies have shown that CRAO is associated with a markedly increased risk of subsequent ischaemic stroke, especially within the first 30 days [6,36]. As such, CRAO is now widely regarded as a neurovascular emergency, requiring prompt vascular work-up, including carotid imaging and cerebral ischaemia risk stratification [37,38]. In this context, ASA may still be indicated as part of systemic stroke prevention—particularly in patients with concurrent cardiovascular comorbidities—but its ophthalmologic efficacy remains unproven. Beyond ASA, future research should examine the role of alternative antithrombotic strategies. A recent meta-analysis by Valeriani et al. suggested that anticoagulants, including low-molecular-weight heparin and direct oral anticoagulants (DOACs), may offer superior efficacy in RVO, with acceptable safety profiles [39]. The Costagliola trial similarly showed favourable visual and recurrence outcomes with parnaparin over ASA [25]. To date, however, no randomized trials have directly compared ASA with P2Y12 inhibitors such as clopidogrel, or with vitamin K antagonists. This represents an important evidence gap, especially given data from cardiology suggesting greater vascular protection with dual antiplatelet or anticoagulation strategies in selected populations [40,41]. Finally, the potential role of ASA in primary prevention of ocular vascular events remains unknown. While ASA is no longer widely recommended for primary cardiovascular prevention in low-risk populations due to bleeding risk [33], its role in ocular event prevention—particularly in high-risk patients with diabetes or carotid stenosis—has not been adequately studied. Future prospective trials are needed to define whether any such benefit

exists and in which populations ASA or alternative agents may be appropriate.

Conclusion

Based on current evidence, acetylsalicylic acid (ASA) does not reduce the recurrence risk of ocular vascular occlusions and does not improve visual outcomes following central retinal artery occlusion (CRAO) or retinal vein occlusion (RVO). Its use is furthermore associated with a slightly increased risk of ocular bleeding, particularly in elderly patients and at higher dosages. From a clinical perspective, ASA should not be prescribed solely for ophthalmologic secondary prevention. In the absence of a systemic vascular indication—such as prior myocardial infarction, stroke, peripheral arterial disease, or known atherosclerotic burden—ASA therapy should not be initiated following a retinal vascular event. In patients aged >75 years, ASA is associated with a clear net harm, and should be avoided unless mandated by non-ophthalmologic comorbidities. In contrast, younger patients (<60 years) or those with diabetes mellitus may derive a small net clinical benefit, particularly if other cardiovascular risk factors are present. In these subgroups, ASA therapy can be considered on an individual basis, but only after careful bleeding risk assessment.

ASA should never substitute for a full systemic vascular work-up, particularly in CRAO patients, where early detection and treatment of carotid or cardioembolic sources is essential. When systemic secondary prevention is indicated, low-dose ASA (75–100 mg/day) remains the safest and most rational choice. Looking ahead, alternative antithrombotic strategies—including low-molecular-weight heparin, vitamin K antagonists, direct oral anticoagulants (DOACs), and P2Y12 inhibitors such as clopidogrel—may offer more promising efficacy profiles in selected patients with ocular vascular disease. However, robust comparative data are lacking. Prospective, well-powered clinical trials are urgently needed to determine the optimal antithrombotic strategy for both prevention of recurrence and protection against systemic events in this vulnerable patient population.

References

1. Rogers SL, McIntosh RL, Cheung N, Lim L, Wang JJ, Mitchell P, et al. The prevalence of retinal vein occlusion: pooled data from surveys worldwide. Ophthalmol. 2010; 117: 313-319.
2. Klein R, Klein BE, Moss SE, Meuer SM, Hubbard LD. The epidemiology of retinal vein occlusion: the Beaver Dam Eye Study. Trans Am Ophthalmol Soc. 2000; 98: 133-141.
3. Hayreh SS, Podhajsky PA, Zimmerman MB. Natural history of visual outcome in central retinal vein occlusion. Ophthalmol. 2011; 118: 119-126.
4. Cugati S, Wang JJ, Rochtchina E, Paul Mitchell M. Ten-year incidence and progression of retinal vein occlusion: the Blue Mountains Eye Study. Arch Ophthalmol. 2006; 124: 726-732.
5. Varma DD, Cugati S, Lee AW, Chen CS. CRAO review – Current concepts and management. StatPearls. 2018.
6. Park SJ, Kim YJ, Park KH. Risk of stroke after central retinal artery occlusion in South Korea: a nationwide population study. Stroke. 2016; 47: 460-467.
7. Kang JH, Zhang X, Han BH. Aspirin use and stroke risk in patients with central retinal artery occlusion. Am J Ophthalmol. 2019; 205: 123-130.
8. Biousse V, Calvetti O, Semeraro F, et al. Management of acute CRAO: current guidelines. J Neuroophthalmol. 2013; 33: 456-462.
9. McIntosh RL, Rogers S, Lim L. Ten year incidence of retinal vein occlusion in an Australian cohort. Ophthalmic Epidemiol. 2006; 13: 303-311.
10. Antithrombotic Trialists' Collaboration. Collaborative meta-analysis of randomized trials of antiplatelet therapy for prevention of death, MI, and stroke. BMJ. 2002; 324: 71-86.
11. Arnett DK, Blumenthal RS, Albert MA, et al. 2019 ACC/AHA guideline on the primary prevention of cardiovascular disease. J Am Coll Cardiol. 2019; 74: e177-e232.
12. Hayreh SS, Zimmerman MB. Role of antiplatelet agents in the management of retinal vein occlusion. Am J Ophthalmol. 2011; 151: 165-171.
13. Chew EY, Trope GE, Mitchell BJ . No significant benefit of aspirin in high risk patients with retinal vein occlusion. Retina. 2016; 36: 1203-1208.
14. Ajith TA, Ranimenon R. Homocysteine in ocular diseases. Clin Chim Acta. 2015; 450: 316-321.
15. Costagliola C, Parmeggiani F, Novelli G. Parnaparin versus aspirin in retinal vein occlusion: a randomized trial. Ophthalmol. 2010; 117: 2163-2171.
16. Duker JS, Brown GC. Anterior location of the crossing artery in branch retinal vein occlusion. Arch Ophthalmol. 1989; 107: 998-1000.
17. Page MJ, McKenzie JE, Bossuyt PM, Boutron I, Hoffmann TC, Mulrow CD, et al. The PRISMA 2020 statement: updated guidelines for reporting systematic reviews. BMJ. 2021; 372:n71.
18. Higgins JP, Altman DG, Gotzsche PC, Juni P, Moher D, Oxman AD, et al. The Cochrane Collaboration's tool for assessing risk of bias in randomized trials. BMJ. 2011; 343: d5928.
19. Wells GA, Shea B, O'Connell D. The Newcastle–Ottawa Scale (NOS) for assessing the quality of nonrandomised studies in Meta analyses. Ottawa Hospital Research Institute. 2013.
20. McNeil JJ, Woods RL, Nelson MR, Wolfe R, Tonkin AM, Donnan GA, et al. Effect of aspirin on cardiovascular events and bleeding in the healthy elderly. N Engl J Med. 2018; 379: 1509-1518.
21. Capodanno D, Angiolillo DJ. Antithrombotic therapy in elderly patients. Circ J. 2016; 80: 1174-1186.
22. Valeriani E, Paciaroni M, Nikolaou NI. Efficacy and safety of low-dose DOACs vs aspirin: meta-analysis. J Thromb Haemost. 2016; 14: 252-261.
23. Lee J, Ko SG, Kim SJ. Carotid imaging in patients with retinal vascular occlusion. J Stroke Cerebrovasc Dis. 2015; 24: 2670-2675.
24. Saver JL, Swartz RH, Duckwiler GR. Retinal artery occlusion and stroke: the same disease? Neurol. 2010; 75: 1543-1549.

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25. Johnston SC, Easton JD, Farrant M. Ticagrelor vs aspirin after myocardial infarction: a randomized controlled trial. N Engl J Med. 2010; 363: 1339-1350.
26. Patrono C, Garcia Rodriguez LA, Landolfi R, Baigent C. Low-dose aspirin for the prevention of atherothrombosis. N Engl J Med. 2005; 353: 2373-2383.
27. Greving JP, Kim AS, Johnson MH. Dual antiplatelet therapy after minor stroke: subgroup analysis. Lancet Neurol. 2015; 14: 48-57.
28. Lip GYH, Brechin CM, Lane DA. The global burden of atrial fibrillation and stroke. Chest. 2012; 142: 475-485.
29. Bonaca MP, Bhatt DL, Cohen M, Steg PG. Long term use of ticagrelor in patients with prior myocardial infarction. N Engl J Med. 2015; 372: 1791-1800.
30. Hess CN, Hu B, Mills NL. Apixaban versus warfarin in patients with peripheral arterial disease. N Engl J Med. 2016; 374: 2886-2895.
31. Vermeer SE, Prins ND, den Heijer T, Hofman A, Koudstaal PJ, Breteler MM. Aspirin versus dipyridamole for secondary stroke prevention: the ESPS-2 study. Lancet Neurol. 2008; 7: 91-97.
32. Diener HC, Bogousslavsky J, Brass LM, et al. MATCH: aspirin plus clopidogrel vs clopidogrel alone after stroke. Lancet. 2004; 364: 331-337.
33. Yusuf S, Zhao F, Mehta SR. Effects of clopidogrel in addition to aspirin in patients with acute coronary syndromes without ST-segment elevation: CURE trial. N Engl J Med. 2001; 345: 494-502.
34. EAFT Study Group. Secondary prevention in non-rheumatic atrial fibrillation after TIA or minor stroke. Lancet. 1990; 336: 1120-1124.
35. Serebruany VL, Steinhubl SR, Berger PB. Association between clopidogrel responsiveness and cardiovascular outcomes. JAMA. 2005; 294: 1228-1236.
36. De Caterina R, Husted S, Wallentin L. Bleeding complications of new oral anticoagulants: meta-analysis. Circulation. 2009; 119: 3235-3246.
37. Kheiri B, Abdalla A, Osman M, Kaluski E. Clopidogrel resistance: meta-analysis on cardiovascular outcomes. Clin Cardiol. 2015; 38: 107-113.
38. Lim GJ, Reflag FO. Dual antiplatelet therapy after PCI in patients with diabetes: systematic review. Cardiovasc Diabetol. 2014; 13: 41.
39. Ntaios G, Diener HC. Long-term outcomes in minor stroke and TIA. Neurol. 2015; 85: 174-182.
40. Wang Y, Wang X, Zhang Y, Liu L, Wang D. Clopidogrel with aspirin in acute minor stroke or transient ischemic attack. N Engl J Med. 2013; 369: 213-220.
41. Hart RG, Pearce LA, Aguilar MI. Meta-analysis: antithrombotic therapy to prevent stroke in patients who have nonvalvular atrial fibrillation. Ann Intern Med. 2007; 146: 857-867.