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SYSTEMATIC REVIEW AND META-ANALYSIS

Admission Mean Platelet Volume and Poor Functional Outcome After Reperfusion Therapy for Acute Ischaemic Stroke: A Systematic Review and Meta-Analysis

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ABSTRACT

Background: Mean platelet volume (MPV), an inexpensive index of platelet activation from the routine admission full blood count, has been linked to poor outcome in unselected ischaemic stroke, but its prognostic value in reperfusion-treated patients is uncertain and conflicting.

Objective: To quantify the association between admission MPV and poor functional outcome after reperfusion therapy, and to test whether it depends on treatment modality and MPV operationalisation.

Methods: PubMed was searched for cohort or case–control studies reporting admission MPV against poor functional outcome (modified Rankin Scale 3–6) in adults with acute ischaemic stroke treated with intravenous thrombolysis and/or mechanical thrombectomy. Odds ratios (OR) were pooled using DerSimonian–Laird random-effects models, with a-priori subgroups by modality and MPV metric and leave-one-out, adjusted-only and Hartung–Knapp sensitivity analyses. This review was not registered in a public registry.

Results: Ten studies were included and seven (1,597 patients) pooled. Higher MPV was associated with poor outcome (OR 1.62, 95% CI 1.22–2.16; I^2=68.5%). The effect was strong with dichotomised MPV after thrombectomy (OR 2.56, 95% CI 1.60–4.08) but modest with continuous or quartile MPV in broad reperfusion (OR 1.27, 95% CI 1.08–1.49); the between-subgroup difference was significant (p=0.005) yet not robust to removing the most weighted study per arm.

Conclusion: Higher admission MPV was associated with poor functional outcome after reperfusion therapy, but its magnitude depended on modality and measurement; certainty was low (GRADE). MPV is a promising, essentially free supplementary marker best used within multivariable models, requiring confirmation in standardised prospective studies.

1. Introduction

Acute ischaemic stroke remains one of the leading causes of death and acquired disability worldwide, and its absolute burden has continued to grow as populations age.[1] Stroke caused by large vessel occlusion (LVO) represents the most severe subtype, and reperfusion therapy — intravenous thrombolysis and mechanical thrombectomy — has become the standard of care because it substantially improves recanalisation rates and long-term functional independence. Despite this benefit, a large proportion of patients with LVO still experience a poor functional outcome even after angiographically successful recanalisation, a phenomenon widely described as futile recanalisation.[2–4] The early identification of patients at risk of a poor outcome is therefore important for risk stratification, individualised treatment planning and the intensity of post-procedural monitoring.[5,6]

Blood-based markers that are inexpensive and rapidly available are appealing tools for ultra-early risk stratification. Platelets play a central role in thrombus formation and in the thrombo-inflammatory cascade that accompanies cerebral ischaemia, interacting with neutrophils and the endothelium to extend ischaemia–reperfusion injury.[7,8] Mean platelet volume (MPV) is an index of platelet size that reflects platelet activation: larger platelets contain more dense granules and release greater quantities of pro-thrombotic mediators, and therefore possess higher thrombogenic potential.[9] Elevated MPV may additionally reflect a greater proportion of young, reticulated platelets — a marker of accelerated platelet turnover associated with reduced responsiveness to antiplatelet therapy.[10,11] Because MPV is obtained routinely from the admission full blood count at no additional cost, it is a practically attractive candidate prognostic marker, particularly where advanced imaging is not universally available.

A body of evidence has linked higher MPV to worse outcomes in unselected ischaemic stroke, as summarised in a previous meta-analysis.[12] However, in the specific population undergoing reperfusion therapy the findings are contradictory. Some cohorts identified MPV as an independent predictor of poor outcome after thrombectomy,[13,14] whereas others found no significant association after multivariable adjustment.[15–17] Related platelet-derived indices, including the MPV-to-platelet-count ratio, the MPV-to-lymphocyte ratio and platelet distribution width, have shown similarly heterogeneous performance.[18–20] These discrepancies may reflect heterogeneous operationalisation of MPV — dichotomisation at data-derived receiver operating characteristic (ROC) thresholds versus continuous modelling — and differences in the aetiological composition of cohorts. To date, no quantitative synthesis has specifically targeted the reperfusion-treated population.

The novelty of this study lies in being the first meta-analysis to quantify the prognostic value of admission MPV specifically in patients with acute ischaemic stroke treated with reperfusion therapy, and in explicitly disentangling the effect according to treatment modality and MPV operationalisation — an axis that plausibly explains the conflicting results of prior primary studies. The aim of this study was to systematically review and quantitatively synthesise the association between admission MPV and poor functional outcome (mRS 3–6) in adults with acute ischaemic stroke undergoing intravenous thrombolysis and/or mechanical thrombectomy, and to examine, through pre-specified subgroup and sensitivity analyses, whether and in what context MPV carries prognostic information.

2. Methods

2.1. Design and reporting

This systematic review and meta-analysis was designed and reported in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement.[21] The review question followed the PECO structure: the population comprised adults (≥18 years) with acute ischaemic stroke treated with reperfusion therapy (intravenous thrombolysis and/or mechanical thrombectomy), a population in which LVO is prominent but not universal; the exposure was a higher admission MPV; the comparator was a lower admission MPV; and the outcome was a poor functional outcome, defined as a modified Rankin Scale (mRS) score of 3–6. This review was not registered in a public registry. A strict LVO-only definition would have left too few studies for synthesis; the implications of the broader framing are considered in the Discussion.

2.2. Search strategy and eligibility

A systematic search of the PubMed database was performed, restricted to English-language publications, and supplemented by hand-searching the reference lists of relevant primary studies and previous meta-analyses. The core Boolean string was: ("mean platelet volume" OR MPV) AND ("large vessel occlusion" OR LVO OR thrombectomy OR endovascular OR thrombolysis OR "ischemic stroke") AND (outcome OR prognosis OR "modified Rankin" OR mRS OR mortality). Studies were included if they were cohort or case–control studies reporting a measure of association with its 95% confidence interval, or data permitting its calculation (a 2×2 table, or mean ± standard deviation or median MPV per outcome group), between admission MPV and functional outcome in reperfusion-treated ischaemic stroke. Case reports, reviews, studies without extractable estimates, and studies reporting only composite ratio markers that were not pure MPV were excluded. Where cohorts overlapped, the single study with the most complete data was retained for the primary pool.

2.3. Data extraction and risk of bias

Two reviewers independently screened and extracted data; disagreements were resolved by consensus. Extracted variables included first author and year, country, design, sample size, reperfusion modality, MPV operationalisation and threshold, outcome definition and time-point, and the adjusted effect estimate with adjustment covariates. Because all eligible studies were observational cohorts, methodological quality was appraised with the Newcastle–Ottawa Scale for cohort studies; the Cochrane risk-of-bias tool for randomised trials was not applicable.

2.4. Effect measures and statistical synthesis

The odds ratio (OR) for poor functional outcome (mRS 3–6) was the single common metric; the standardised mean difference (Hedges' g) was used only as an intermediate step to place continuously reported studies onto the log-odds scale via the Hasselblad–Hedges relationship ($\ln OR = g \times \pi/\sqrt{3}$). For dichotomous data, a crude OR was derived from the 2×2 table using the Woolf standard error. One study reporting a favourable-outcome OR of 0.89 (95% CI 0.70–1.13) was inverted to a poor-outcome OR of 1.12 (95% CI 0.89–1.43). Log odds ratios were pooled using the DerSimonian–Laird random-effects model.[22] Heterogeneity was assessed with the Cochran Q statistic, the I^2 index and the between-study variance τ^2, with a 95% prediction interval.[23] A-priori subgroups were defined by treatment modality and MPV operationalisation, and the difference between subgroups was tested with the between-subgroup Q statistic (Q_between). Robustness was probed with leave-one-out, adjusted-only and Hartung–Knapp analyses, and by re-testing the between-subgroup contrast after removing the most heavily weighted study in each arm. Small-study effects (Egger test) were pre-specified only where the number of pooled studies reached ten.[24] Certainty of evidence was appraised using the GRADE framework. A two-sided p value below 0.05 was considered significant.

3. Results

3.1. Study selection and characteristics

The search identified 139 records; after de-duplication and screening, ten cohort studies met the eligibility criteria and entered the qualitative synthesis, as summarised in the study-selection flow diagram (Figure 1). Seven studies provided an extractable MPV-specific estimate and were pooled; the seven cohorts totalled 1,597 patients — Peng (153), Tian (247), Sabença (129), Chen (257), Xie (183), Dourado Sotero (267) and Dębiec (361).[13–17,25,26] Three studies were retained in the narrative but not pooled: one shared its cohort with another included study from the same centre,[27] and two did not provide an extractable MPV-specific estimate.[28,29] Study characteristics are detailed in Table 1 and outcome definitions in Table 2.

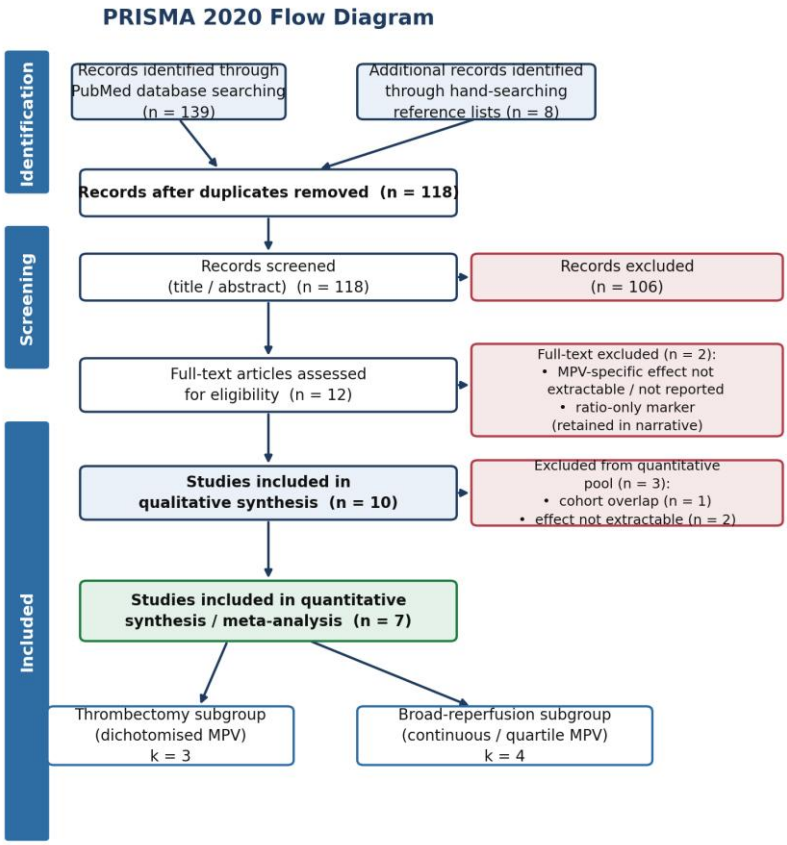

Figure 1. PRISMA 2020 flow diagram. Ten studies entered the qualitative synthesis and seven the quantitative meta-analysis (thrombectomy subgroup, k = 3; broad-reperfusion subgroup, k = 4).

Table 1. Characteristics of the included studies.

Study	Country	Design	n	Modality	MPV metric	OR (95% CI)
Peng 2018	China	Prospective, 2-centre	153	Thrombectomy	≥10.4 fL	3.93 (1.73–8.94)
Tian 2025	China	Retrospective	247	Thrombectomy	>10.75 fL	2.75 (1.79–4.22)
Sabença 2020	Portugal	Prospective	129	Thrombectomy	Cut-off / tertile	1.45 (0.65–3.23)*
Chen 2021	China	Retrospective	257	Thrombectomy (EVT)	Continuous	1.37 (0.86–2.20)*
Xie 2019	China	Retrospective	183	Thrombolysis	Continuous / tertile	1.52 (1.01–2.29)
Dourado Sotero 2021	Portugal	Retrospective	267	Thrombolysis	Continuous	1.36 (1.00–1.83)
Dębiec 2021	Poland	Retrospective	361	tPA ± thrombectomy	Quartile	1.12 (0.89–1.43)†
Staszewski 2019‡	Poland	Retrospective	237	Thrombolysis	Tertile (>8.8 fL)	1.90 (1.01–4.00)
Li (ATTENTION) 2023‡	China	Registry	1044	Thrombectomy (basilar)	Quartile	Not extractable
Wang 2021‡	China	Retrospective	267	Thrombolysis	Continuous	Not extractable

*OR, odds ratio for poor functional outcome (mRS 3–6); fL, femtolitres; EVT, endovascular thrombectomy. *Reconstructed estimate. †Inverted from the reported favourable-outcome OR. ‡Included in the qualitative synthesis but not pooled.*

Table 2. Outcome definition and assessment time-point for the seven pooled studies.

Study	Outcome definition	Time-point	Estimate type
Peng 2018	mRS 3–6	3 months	Adjusted OR
Tian 2025	mRS 3–6	90 days	Adjusted OR
Sabença 2020	mRS 3–6	3 months	Crude 2×2 (reconstructed)
Chen 2021	mRS 3–6	3 months	Median→SMD (reconstructed)
Xie 2019	mRS 3–6	90 days	Adjusted OR
Dourado Sotero 2021	mRS 3–6	Discharge (90-day trend)	Adjusted OR
Dębiec 2021	mRS 3–6 (inverted from mRS 0–2)	In-hospital era	Adjusted OR, inverted

mRS, modified Rankin Scale; SMD, standardised mean difference. Most studies assessed the outcome at 90 days; two at discharge.

3.2. Risk of bias

On the Newcastle–Ottawa Scale, the two thrombectomy cohorts by Peng and by Tian and the large ATTENTION registry were judged to be at low overall risk of bias, reflecting well-defined cohorts, adjustment for major confounders and complete outcome ascertainment.[13,14,28] The remaining studies were judged to be at moderate risk, most commonly because of retrospective single-centre design, incomplete adjustment, or outcome assessment at discharge rather than at 90 days, as presented in Figure 2.

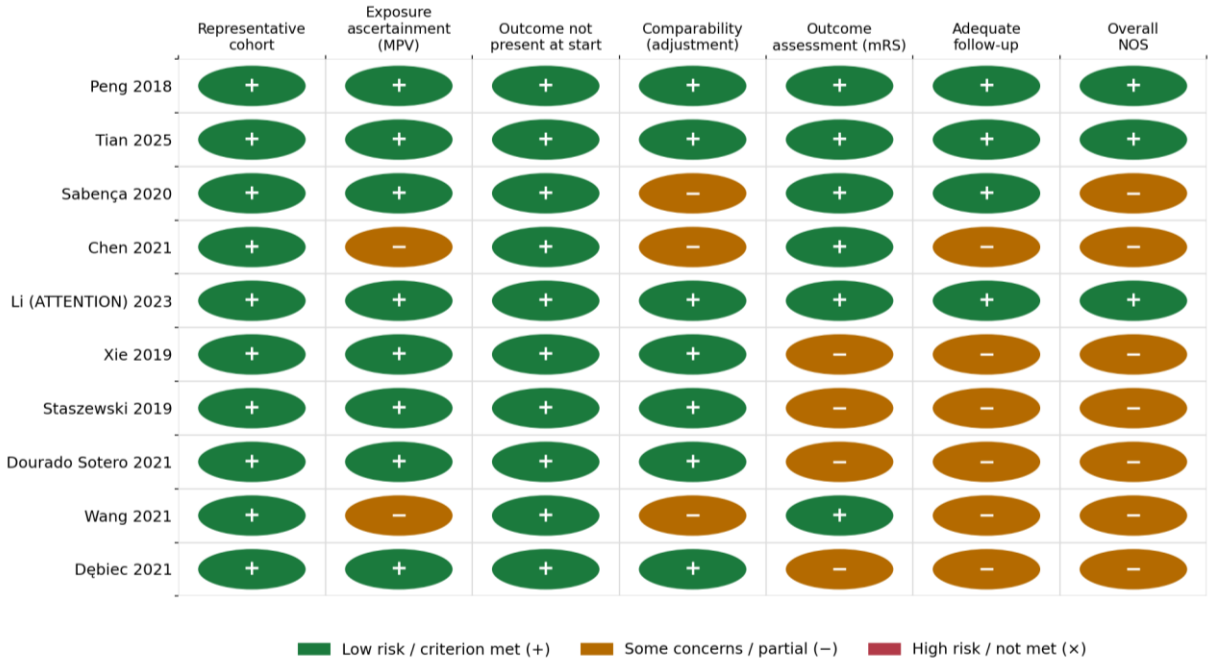

Figure 2. Newcastle–Ottawa risk-of-bias assessment across the selection, comparability and outcome domains for the included cohort studies.

3.3. Primary synthesis and subgroups

Pooling the seven studies showed that a higher admission MPV was associated with an increased odds of poor functional outcome: pooled OR 1.62 (95% CI 1.22–2.16; p = 0.0009), with substantial heterogeneity (I^2 = 68.5%; τ^2 = 0.092; Q = 19.05, df = 6, p = 0.004). The 95% prediction interval was wide (OR 0.83–3.19) and included unity, so a null or trivially protective true effect cannot be excluded in some settings. The forest plot is shown in Figure 3. The pre-specified subgroup analysis explained much of the heterogeneity: in the mechanical thrombectomy subgroup with dichotomised MPV (Peng, Tian and Sabença; 529 patients) the association was strong and homogeneous (OR 2.56, 95% CI 1.60–4.08; p < 0.001; I^2 = 35.0%),[13–15] whereas in the broad-reperfusion subgroup with continuous or quartile MPV (Chen, Xie, Dourado Sotero and Dębiec; 1,068 patients) it was weaker but significant (OR 1.27, 95% CI 1.08–1.49; p = 0.004; I^2 = 0%).[16,17,25,26] The between-subgroup difference was significant (Q_between = 7.73, df = 1, p = 0.005; I^2_between = 87.1%) but should be regarded as exploratory, since it did not survive removal of the most heavily weighted study in each arm and part of it is likely methodological, arising from ROC-based dichotomisation.

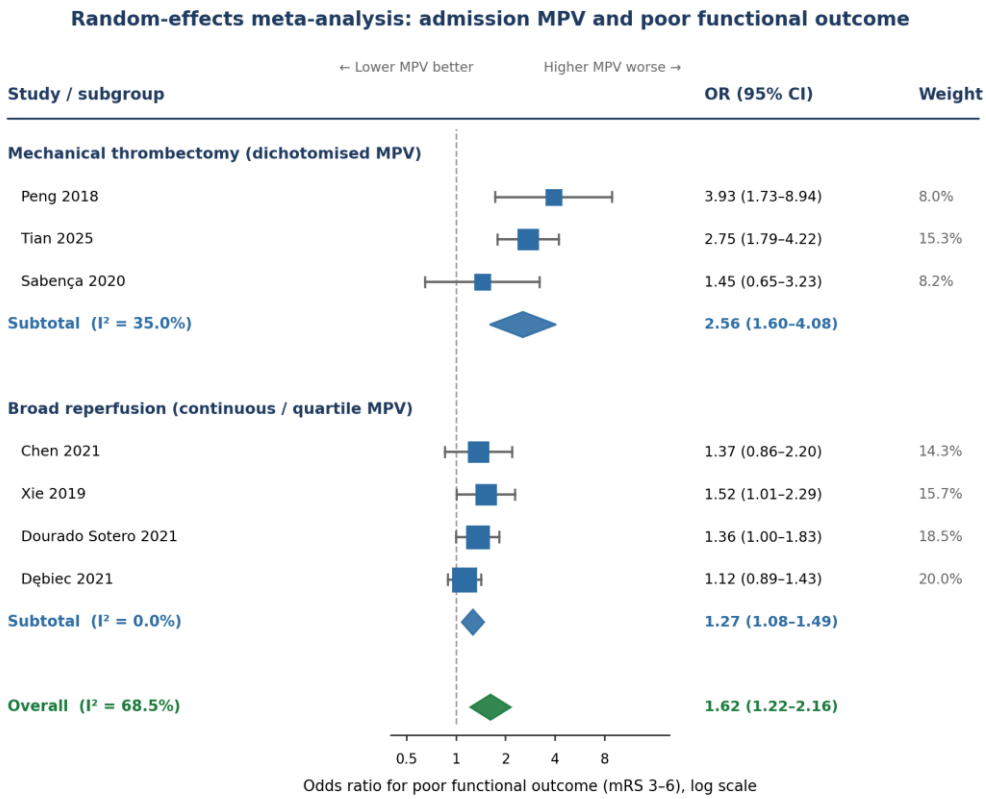

Figure 3. Random-effects forest plot stratified by the pre-specified subgroups. Squares represent individual study odds ratios (size proportional to weight); diamonds represent pooled estimates.

3.4. Sensitivity analyses and certainty of evidence

Leave-one-out analysis confirmed a robust pooled estimate (OR range 1.42–1.78, all confidence intervals above unity; Figure 4). The additional analyses are summarised in Table 3. Restricting the pool to the five adjusted-OR studies gave a similar point estimate (OR 1.73) but, under the Hartung–Knapp adjustment, an interval that crossed unity. The Hartung–Knapp adjustment applied to the primary analysis widened the overall interval (OR 1.62, 95% CI 1.11–2.37) while preserving significance; applied to the thrombectomy subgroup it produced a wide interval that included unity (OR 2.56, 95% CI 0.87–7.54), underscoring the fragility of a three-study subgroup. The broad-reperfusion subgroup remained significant (OR 1.27, 95% CI 1.02–1.58). Removing the most heavily weighted study in each arm abolished the between-subgroup difference (2.38 versus 1.40; p = 0.30). Because fewer than ten studies were pooled, the Egger test was not performed, the funnel plot in Figure 4 is descriptive only, and publication bias could not be excluded. Applying GRADE, the certainty of evidence was judged to be low, limited by inconsistency and imprecision (Table 4).

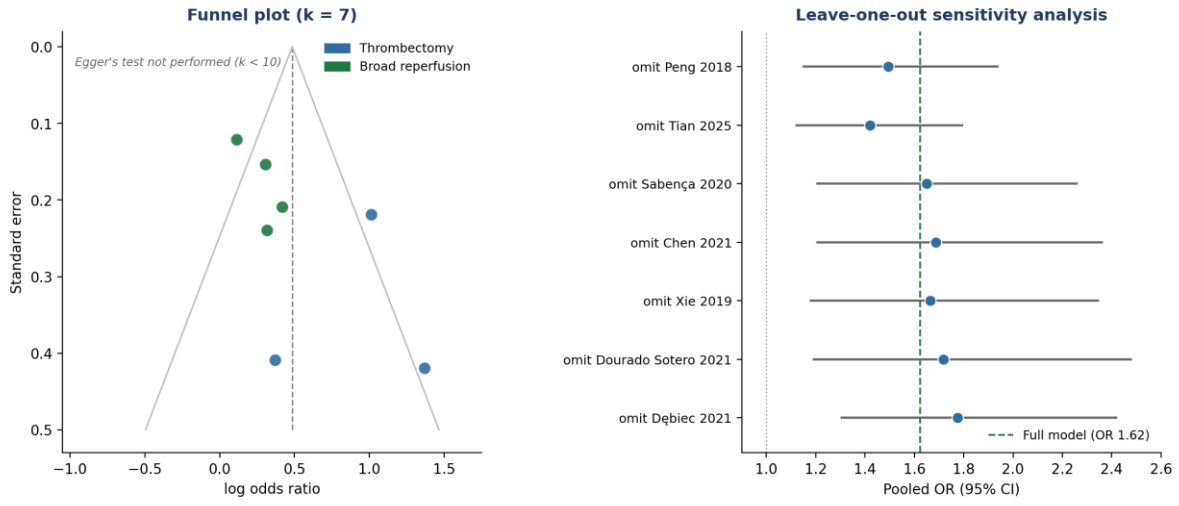

Figure 4. Descriptive funnel plot (left) and leave-one-out sensitivity analysis (right). The Egger test was not performed because fewer than ten studies were pooled.

Table 3. Summary of the primary and sensitivity analyses.

Analysis	k	Pooled OR (95% CI)	I^2	Comment
Primary (DerSimonian–Laird)	7	1.62 (1.22–2.16)	68.5%	Significant
Hartung–Knapp adjustment	7	1.62 (1.11–2.37)	68.5%	Significant, wider
Adjusted-OR studies only	5	1.73 (1.19–2.51)	79.0%	Significant (DL)
— same, Hartung–Knapp	5	1.73 (0.95–3.14)	79.0%	Crosses unity
Thrombectomy subgroup	3	2.56 (1.60–4.08)	35.0%	Significant (DL)
— same, Hartung–Knapp	3	2.56 (0.87–7.54)	35.0%	Crosses unity (fragile)
Broad-reperfusion subgroup	4	1.27 (1.08–1.49)	0%	Robust
Between-subgroup, omit heaviest each arm	5	2.38 vs 1.40	—	Difference NS (p = 0.30)
Leave-one-out range	6	1.42–1.78	—	All CIs exclude 1

Notes: DL, DerSimonian–Laird; NS, not significant. Primary DerSimonian–Laird estimates are unchanged; the remaining rows are sensitivity analyses.

Table 4. GRADE certainty-of-evidence assessment.

Domain	Judgement	Rationale
Study design	Observational cohorts	Starts at low certainty
Risk of bias	Not serious	Mostly moderate NOS; low for anchor studies
Inconsistency	Serious	I^2 = 68.5%; prediction interval crosses unity
Imprecision	Serious	Only 7 studies; HK crosses unity in subgroups
Indirectness	Not serious	Direct population, exposure and outcome
Publication bias	Not assessable	k < 10; cannot be excluded
Overall certainty	**LOW**	**Positive association, but confidence limited**

Notes: NOS, Newcastle–Ottawa Scale; HK, Hartung–Knapp.

4. Discussion

In this meta-analysis of seven cohort studies comprising 1,597 pooled patients, a higher admission MPV was associated with an increased odds of poor functional outcome after reperfusion therapy (pooled OR 1.62). The most important finding was that the magnitude of the association depended strongly on treatment modality and on how MPV was measured (Figure 3, Table 3): strong and consistent with dichotomised MPV after thrombectomy (OR 2.56) yet substantially weaker with continuous or quartile MPV in broad reperfusion (OR 1.27). Conservative sensitivity analyses temper this message — the between-subgroup contrast did not survive removal of the most influential study in each arm, and the thrombectomy interval crossed unity under Hartung–Knapp adjustment — so we present the modality-dependence of MPV as a testable hypothesis rather than an established fact.

The direction of our findings is consistent with a previous meta-analysis in unselected stroke, which reported higher MPV in patients with poor outcome and found the effect most consistent in non-thrombolytic patients[12] — an observation that dovetails with our attenuated signal once a broad reperfusion-treated population and continuous metrics are considered. Our thrombectomy estimate is concordant with individual positive cohorts,[13,14] whereas the weaker continuous-metric estimate reconciles the apparently null primary studies with the overall positive signal.[15,16] The added value of this review is its explicit focus on the reperfusion-treated population and the separation of the effect by modality, which has not previously been undertaken.

4.1. Explaining the between-subgroup difference

At least three complementary factors explain the between-subgroup difference. First, MPV operationalisation: the positive thrombectomy studies dichotomised MPV at data-derived ROC thresholds, which tend to inflate the effect size relative to a per-unit continuous odds ratio — an interpretation reinforced by the loss of between-subgroup significance in our robustness analysis.[13,14] Second, aetiological composition: cohorts dominated by cardioembolic stroke may dilute the value of MPV, because platelet-rich thrombi are more characteristic of atherothrombotic mechanisms and clot composition influences both outcome and recanalisation;[30,31] an MPV effect more evident in large-artery atherosclerosis has been reported.[32] This remains a hypothesis because included studies did not consistently report outcomes by TOAST subtype. Third, loss of independent signal under adjustment: when modelled continuously alongside inflammatory markers such as the neutrophil-to-lymphocyte ratio, MPV may partly index stroke severity, and several cohorts reported that an inflammatory ratio outperformed MPV.[16,33–35]

4.2. Biological plausibility

There is a coherent biological basis for the association. Larger platelets release greater quantities of thromboxane, P-selectin and glycoprotein IIb/IIIa, and elevated MPV may reflect more reticulated, immature platelets associated with antiplatelet resistance.[10,11] In the thrombectomy context, this heightened pro-thrombotic tendency, together with platelet-driven

thrombo-inflammation and blood–brain-barrier disruption, could facilitate re-thrombosis and ischaemia–reperfusion injury despite successful reperfusion.[7,8] This connects MPV to futile recanalisation, in which a patient recanalises yet fails to recover;[2,36] a marker flagging such patients would be more specifically useful, but no pooled study conditioned outcomes on recanalisation status.

4.3. Clinical implications

MPV is inexpensive, routinely measured and rapidly available, making it an attractive supplementary marker, particularly among thrombectomy candidates and in resource-conscious settings.[37] Its discriminative ability is nonetheless modest (areas under the ROC curve around 0.6–0.65),[38] so a statistically detectable association is not a clinically discriminating test. A high admission MPV may reasonably be regarded as one of several markers flagging higher risk and prompting closer observation; it should not, on current evidence, alter the decision to offer reperfusion, and it is best integrated into multivariable models together with established clinical, imaging and inflammatory variables, where even a small incremental value could be worthwhile at scale.[38,39]

4.4. Generalisability

Generalisability warrants caution. Most cohorts originated from East-Asian centres, and MPV varies with ethnicity, analyser type and local practice; the thresholds used by positive thrombectomy studies differ and correspond to no universal cut-point,[13,14] so a clinician elsewhere could not simply transplant a threshold. For the international and substantially South-East Asian readership of this journal, where a full blood count is universally available even when advanced imaging is not, MPV holds particular appeal, but this raises the importance of local validation.

4.5. Strengths and limitations

The strengths of this review include a focused question, a-priori subgroup definition, a robust primary estimate on leave-one-out analysis, transparent handling of reconstructed and inverted estimates, exclusion of overlapping cohorts, and a comprehensive set of sensitivity analyses with a formal GRADE appraisal. Limitations must be emphasised. First, the number of pooled studies was small, and the thrombectomy subgroup comprised only three studies, so its estimate is fragile.[13–15] Second, two of seven estimates were reconstructed, mixing adjusted with unadjusted estimates.[15,16] Third, heterogeneous MPV operationalisation and differing outcome definitions precluded a single homogeneous metric. Fourth, pre-analytical variability is a concern: MPV rises with storage time in EDTA, and the sampling-to-measurement interval was rarely reported.[9] Fifth, most cohorts were retrospective, single-centre and East-Asian, and the English-language restriction may have introduced selection bias. Finally, publication bias could not be assessed. Future work should comprise adequately powered, prospective, multicentre thrombectomy cohorts with a pre-registered plan, a standardised pre-analytical protocol, a single continuous MPV metric reported per standard deviation, stratification by TOAST aetiology, conditioning on recanalisation status,[3–6] and evaluation of MPV alongside competing markers within validated prediction models.[39–44]

5. Conclusion

A higher admission mean platelet volume was associated with a poor functional outcome in patients with acute ischaemic stroke treated with reperfusion therapy, but the strength of the association depended markedly on treatment modality and measurement scale — strong with dichotomised MPV after mechanical thrombectomy (OR 2.56) and weaker, though still significant, with continuous or quartile MPV in broad reperfusion (OR 1.27). The overall estimate (OR 1.62) was robust to leave-one-out removal, but the wide prediction interval, the fragile between-subgroup difference and the widening of the thrombectomy interval under a conservative estimator indicate low certainty of evidence. MPV should be regarded as a promising but not yet definitive supplementary marker whose apparent value in thrombectomy candidates is hypothesis-generating. Because it is inexpensive and universally available from the routine admission full blood count, it is well positioned to complement — rather than replace — established predictors within multivariable models. Confirmation requires adequately powered, prospective, multicentre studies that standardise the MPV metric and pre-analytical interval, stratify by aetiology, condition on recanalisation status, and evaluate incremental prognostic value within integrated models.

6. Declarations

6.1. Ethics approval and consent to participate

Ethical approval and patient consent were not required for this systematic review and meta-analysis, which synthesised previously published, aggregate, de-identified data and used no individual patient data or identifiable material (including neuro-imaging).

6.2. Consent for publication

Not applicable; no identifiable individual patient data or images are presented.

6.3. Availability of data and materials

All data analysed in this review were extracted from previously published studies, each of which is cited in the reference list. The extraction table and the reconstructed and inverted effect-size derivations that support the findings are available from the corresponding author on reasonable request.

6.4. Competing interests

The authors declare no financial or personal conflict of interest.

6.5. Funding

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6.6. Author contributions

Conception and design: IADK and KT. Literature search, study selection and data extraction: IADK, IASW and KW. Statistical analysis and interpretation: KT, AAAPL and NPAPM. Drafting of the manuscript: IADK. Critical revision for important intellectual content: all authors. All authors reviewed and approved the final version and agree to be accountable for all aspects of the work.

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