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Red-cell distribution width as a prognostic marker for aneurysmal subarachnoid hemorrhage: A systematic review and meta-analysis

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1. Introduction

Aneurysmal subarachnoid hemorrhage (aSAH) is a disease with a high mortality and morbidity rate. Amongst other factors, one of the highest contributors to the poor prognosis of this condition is a cerebral infarction.[1,2] Cerebral vasospasm has been thought as the sole cause of infarction and so the traditional therapy is to prevent cerebral vasospasm by induced hypertension, intraarterial balloon angioplasty, and infusion of vasodilator drugs such as nimodipine.[3] However, such rescue therapies are often administered after infarction had occurred, hence morbidity and mortality remain high. Fortunately, recent years have seen a surge in research trying to find a suitable prophylactic treatment with several drugs showing good potential, such as nimodipine, clazosentan, and heparin.[4–6] With a possible paradigm shift in the treatment strategy of aSAH, an excellent prognostic model is required to identify high-risk patients that would likely benefit from such a preventative treatment strategy.

Research has shown that inflammation and microthromboembolism are the emerging mechanisms behind an infarction. The current prognostic models such as the Hunt and Hess (HH) scale, the World Federation of Neurosurgical Societies (WFNS) scale, and the modified Fisher (mFisher) scale have good sensitivity and specificity. However, as they only evaluate admission clinical and radiographic characteristics, a suitable biomarker is required to address the biological processes behind an infarction. One of the promising biomarkers is red cell distribution width (RDW). It measures the heterogeneity of the erythrocytes' size by dividing the standard deviation of erythrocyte volume by the mean corpuscular volume (MCV). It has been shown to correlate with inflammatory level[7] and has a good prognostic value in many thrombotic and inflammatory conditions, including ischemic stroke and myocardial infarction.[8,9] RDW is relatively cheaper and easier to measure than other inflammatory markers, which could prove crucial, especially in many developing countries. Because of the promising potential of RDW, we aimed to evaluate the available evidence on its use in predicting the outcome of aSAH patients.

2. Methods

2.1. Database and literature search

A literature search was performed using PubMed, Scopus, Embase, and EuropePMC from database inception until May 31, 2022, with the following keywords: ["subarachnoid hemorrhage" or "aneurysmal subarachnoid hemorrhage" or "SAH" or "aSAH" and "erythrocyte indices" or "red blood cell distribution width" or "RDW"]. The bibliographies of

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Abbreviation list

aSAH	aneurysmal subarachnoid hemorrhage
CD	cluster of differentiation
CI	confidence interval
CSF	cerebrospinal fluid
ESR	erythrocyte sedimentation rate
GCS	Glasgow Coma Scale
GOS	Glasgow Outcome Scale
GRADE	the Grading of Recommendations Assessment, Development and Evaluation
HH	Hunt and Hess
hsCRP	high sensitivity C-reactive protein
ICAM	intercellular adhesion molecule
IL	interleukin
MCP	monocyte chemoattractant protein
MCV	mean corpuscular volume
MMP9	matrix metallopeptidase 9
mRS	modified Ranking Scale
NLR	neutrophil-to-lymphocyte ratio
OR	odds ratio
PLR	platelet-to-lymphocyte ratio
PROSPERO	The International Prospective Register of Systematic Reviews
RDW	red-cell distribution width
ROBINS-E	Risk Of Bias In Non-randomized Studies - of Exposures
ROC	receiver operating characteristic
SAPS	Simplified Acute Physiology Score
SIRI	systemic inflammatory response index
TNF	tumor necrosis factor
vWF	von Willebrand factor
WFNS	World Federation of Neurosurgical Societies

relevant studies were also reviewed to supplement the search. Only English-language literature was included. Two authors independently performed the initial search and screened the title and abstract of relevant studies. Any discrepancies were resolved by discussion with a third author.

2.2. Study selection

The criteria for selecting the study were as follows: 1) prospective and retrospective studies; 2) patients with acute aSAH; 3) showing an association between RDW and aSAH; and 4) reporting functional outcomes, cerebral infarction, or mortality. We expected the studies to use a different sampling time for RDW. Therefore, we included studies that measured RDW either once or serially. Studies that did not provide the raw data or results from the univariate or multivariate analysis were excluded. Review articles, editorials, correspondences, case reports, case series, and non–English-language articles were also excluded.

2.3. Data extraction

Two independent authors performed data extraction for this analysis using a standardized form that included the authors, publication year, study design, patient characteristics, and outcomes measured. Extracted data were then compared, with any discrepancies resolved by discussion.

The severity of aSAH was classified using the HH scale, WFNS scale, or mFisher scale. Functional outcome was measured using Glasgow Outcome Scale (GOS) or modified Rankin Scale (MRS). We expected the included studies would have used different cut-off values to define the functional outcome, so we did not specify a specific cut-off value. For cerebral infarction, we used the definitions suggested by Vergouwen et al.[10] Cerebral infarction was defined as the presence of cerebral infarction on CT or MRI of the brain within six weeks after aSAH, or on the latest CT or MRI study obtained before death within six weeks, or proven at autopsy but not present on CT or MRI study between 24 and 48 h after early aneurysm occlusion and not attributable to other causes such as surgical clipping or endovascular treatment. However, since not all studies followed these suggested definitions, other definitions were allowed, provided they did not differ significantly. Mortality was defined both as in-hospital mortality and long-term mortality. The data were then entered in Review Manager version 5.4 (The Cochrane Collaboration) software by one author and double-checked by another author.

2.4. Statistical analysis

Outcomes were compared between low RDW and high RDW using odds ratios (OR) and 95% confidence intervals (CI). Extracted OR and 95% CI were pooled and weighted using a generic inverse-variance method. Heterogeneity among studies was evaluated using the I^2 statistic and Cochrane *Q*-statistic test. An I^2 value higher than 50% or a P value higher than 0.10 indicated a significant presence of heterogeneity. In such heterogeneity, we used the random-effect model, while the fixed-effect model would be used in the absence of heterogeneity. We also expected the included studies to use different timeframes in evaluating the functional outcome and mortality. As such, we planned a subgroup analysis accordingly. Since this is not an interventional study, we expected the lack of randomized studies in our analysis. Therefore, we planned to conduct a sensitivity analysis by excluding studies with a risk of bias.

The risk of bias was assessed by one author at the study level with the Cochrane risk-of-bias tool (The Cochrane Collaboration) for randomized studies and the Risk Of Bias In Non-randomized Studies - of Exposures tool (ROBINS-E) for nonrandomized studies. Publication bias was assessed by one author using a funnel plot analysis. Certainty of evidence was assessed using the Grading of Recommendations Assessment, Development and Evaluation (GRADE) approach. Analysis was performed with Review Manager version 5.4 (The Cochrane Collaboration). This study was conducted following the 2015 PRISMA guidelines for a systematic review. This review has been registered at The International Prospective Register of Systematic Reviews (PROSPERO) under the registration number CRD42021254654.

3. Results

The initial search from the four databases resulted in 9726 studies; 3052 duplicate studies were excluded, and the remaining 6674 studies were screened. From the initial title and abstract screening, 53 full texts were evaluated for eligibility. 47 studies were excluded because they did not provide data on the outcomes we planned to evaluate, leaving six studies for the final analysis (Fig. 1).[11–16]

3.1. Study characteristic

The characteristics of the included studies are presented in Table 1. All were nonrandomized studies. Chugh et al, Fontana et al, and Siegler et al used serial RDW measurements.[11–13] Chugh et al and Fontana et al used the maximum recorded value over a serial measurement of 7–10 days, while Siegler et al used the average value from a 10-day serial measurement. On the other hand, Huang et al, Hong et al, and Ignacio et al only measured baseline RDW levels.[14–16] The cut-off value ranged from 13.4% to 16%, according to the respective studies' laboratory cut-off. Baseline characteristics (not shown in the table) differed significantly in the study by Chugh et al, Siegler et al, and Fontana et al, in

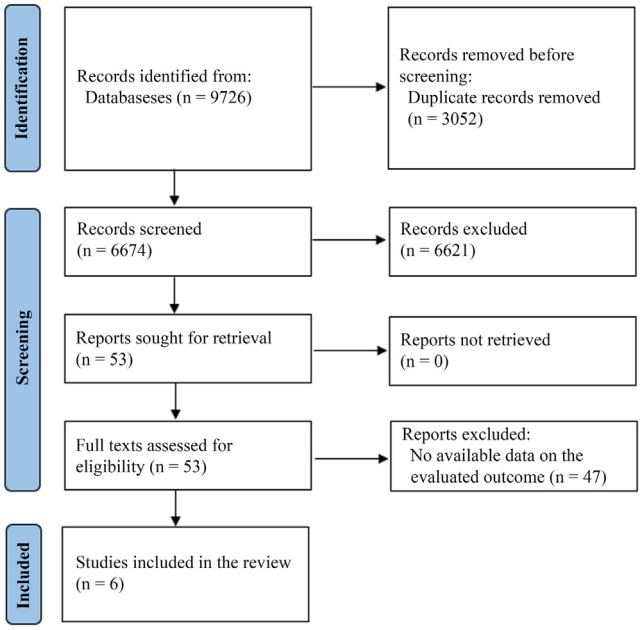

Fig. 1. Study flow diagram.

which group with a high RDW tended to be older and with more severe aSAH.[11–13] Regarding outcome, four studies reported functional outcome, five reported mortality, and three reported cerebral infarction. All included studies used multivariable logistic regression to analyze the outcome.

All the included studies were nonrandomized, so we used the ROBINS-E tool to assess the risk of bias. Table 2 gives a summary of the assessment. Four studies were assessed as having a low risk of bias, while two had some concerns and a high risk of bias. In the study by Chugh et al, the outcome evaluators were not blinded to the RDW level of the participant.[11] This could have introduced a potential bias in evaluating the functional outcome. The prospective study by Hong et al also did not use blinding, but since mortality was the evaluated outcome, the lack of blinding would not have introduced any bias.[15] Although the remaining studies did not use blinding, they were retrospective in nature, and since the evaluators at that time were not aware of any future studies, this would not have introduced any bias. Regarding confounding factors, all the included studies used multivariable logistic regression to analyze the outcome. However, the study by Huang et al was the only study that did not adequately control for confounding.[14] They only included RDW, neutrophil-to-lymphocyte ratio (NLR), and Simplified Acute Physiology Score (SAPS) in their multivariable regression model. Some of their samples' baseline characteristics, such as age and history of diabetes, differed significantly, which could have introduced significant confounding bias. The remaining five studies adequately controlled the confounding factors, where they included age, sex, comorbidities, Glasgow Coma Scale (GCS), HH scale, WFNS scale, mFisher scale, treatment, and other laboratory parameters in their logistic regression.

Publication bias was not analyzed since funnel plot analysis or other statistical analysis did not have enough power to detect publication bias in a small number of studies.

3.2. Severity and RDW value

Four out of the six studies provided a comparison of RDW value between patients' severity. However, a meta-analysis was not possible because the severity classification and reporting differed from one study to another. Chugh et al found that more patients with a high-grade aSAH (HH grade 4–5) had a high RDW value (5/12 [42%] vs 7/28 [25%]). However, the difference they observed was not significant (p 0.45). Fontana et al used median and interquartile ranges and found that patients with a high RDW value significantly had a worse WFNS scale (2 [1–5] vs 1 [1–3]; $p < 0.05$), but they had a better Fisher score (2 [1–5] vs 4 [3–4]; $p < 0.005$). On the other hand, Siegler et al reported patients with a high RDW value had a significantly worse Fisher score (4 [3–4] vs

Table 1
Characteristics of included studies.

Authors & Year	Study Design	Cohort Size (n)	Age (yrs)	Sex (M/F)	RDW Sampling Time	RDW cut-off (%)	Outcome measure
Chugh et al, 2015[11]	Prospective cohort	40	52.8[a]	10/30	Day 0, 1, 3, 5, 7, and 10; maximum value used for analysis	>14.5	Poor functional outcome (3-month mRS 3–6); 90-day mortality
Fontana et al, 2017[12]	Retrospective cohort	270	54[b]	121/149	Day 0 up to 7; maximum value used for analysis	>13.4	Poor functional outcome (3-month GOS 1–3); cerebral infarction; in-hospital mortality
Siegler et al, 2017[13]	Retrospective cohort	179	54[b]	43/136	Day 0–14; mean value used for analysis	>14.5	Poor functional outcome (discharge mRS >4); cerebral infarction
Huang et al, 2017[14]	Retrospective cohort	274	59[a]	110/164	Only initial test result used for analysis	>15	In-hospital mortality
Hong et al, 2018[15]	Prospective cohort	364	54.2[a]	148/216	Only initial test result used for analysis	>14.5	90-day mortality
Ignacio et al, 2022[16]	Retrospective cohort	222	51.7[a]	79/143	Only initial test result used for analysis	>16	Poor functional outcome (discharge mRS 3–6); cerebral infarction; in-hospital mortality

[a] Mean.
[b] Median.

Table 2
Risk of bias assessment.

Authors & Year	Confounding	Exposure Measurement	Selection	Post-exposure intervention	Missing data	Outcome measurement	Selection	Overall
Chugh et al, 2015	Low	Low	Low	Low	Low	Some concern	Low	Some concerns
Fontana et al, 2017	Low	Low	Low	Low	Low	Low	Low	Low risk of bias
Siegler et al, 2017	Low	Low	Low	Low	Low	Low	Low	Low risk of bias
Huang et al, 2017	High	Low	Low	Low	Low	Low	Low	High risk of bias
Hong et al, 2018	Low	Low	Low	Low	Low	Low	Low	Low risk of bias
Ignacio et al, 2022	Low	Low	Low	Low	Low	Low	Low	Low risk of bias

3 [3–4]; p 0.037). Hong et al found the median RDW value of patients with a severe aSAH (HH grade 4–5) was significantly higher than those with nonsevere HH grade (14.0 [10.1–17.7] vs 9.7 [7.6–12.7]; $p < 0.001$).

3.3. Functional outcome

Four studies with a total of 711 samples assessed the relationship between RDW value and functional outcome.[11–13,16] Ignacio et al and Siegler et al evaluated functional outcome at discharge, while Chugh et al and Fontana et al evaluated during a 3-month follow-up. Overall, higher RDW correlated with a worse functional outcome with an OR of 1.70 (95% CI 1.32–2.19; $p < 0.0001$). In the subgroup analysis, higher RDW resulted in a worse discharge and 3-month functional outcome, although the observed effect was not statistically significant during discharge (Fig. 2).

3.4. Mortality

Five studies with a total of 1170 samples assessed the relationship between RDW value and mortality.[11,12,14–16] Fontana et al, Huang et al, and Ignacio et al recorded hospital mortality, while Chugh et al and Hong et al recorded mortality over 90 days. Higher RDW increased the mortality rate with OR 2.16 (95% CI 1.25–3.72; $p = 0.006$). The subgroup analysis also showed a significant result for in-hospital and 90-day mortality (Fig. 3).

3.5. Cerebral infarction

Three studies with a total of 671 samples assessed the relationship between RDW and cerebral infarction.[12,13,16] They excluded other possible causes but did not elaborate further on the methods and time at which infarction occurred. High RDW translated to a higher risk of cerebral infarction with OR 2.74 (95% CI 1.71–4.40; $p < 0.0001$) (Fig. 4).

3.6. Sensitivity analysis

We evaluated the study by Chugh et al and Huang et al to have a risk of bias.[11,14] The lack of blinding in the study by Chugh et al could have introduced measurement bias, and therefore we excluded their study in the functional outcome analysis. We did not exclude that study from the mortality analysis because mortality is an objective outcome and would not be hindered by the lack of blinding. We also excluded the study by Huang et al that had a high risk of bias due to possible confounders. Our sensitivity analysis did not differ from the primary analysis in which RDW correlated significantly with functional outcome and mortality (Fig. 5, Fig. 6).

3.7. Certainty of evidence

The evidence was judged to be of very low to moderate certainty. Evidence regarding functional outcome did not have serious concerns, but the observational nature of the studies only produced low certainty. Evidence regarding mortality had a serious risk of bias and inconsistency. Therefore, despite its strong association, it was judged to be of very low certainty. Evidence regarding cerebral infarction was judged to be of moderate certainty due to its strong association. Table 3 gives a summary of the assessment.

4. Discussion

RDW is usually evaluated during a routine complete blood count examination. It reflects the state of erythropoiesis, and historically it has been used to help in differentiating iron-deficiency anemia, megaloblastic anemia, and thalassemia.[17] However, there has been a renewed interest in RDW as an inflammatory marker in recent years. RDW is now a known marker for autoimmune or inflammatory conditions such as systematic lupus erythematosus[18] and rheumatoid arthritis.[19] Inflammation itself causes impairment of erythropoiesis.[20] Moreover, it also strongly

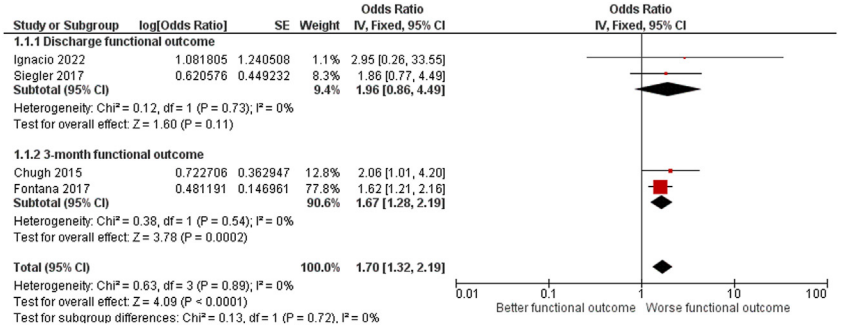

Fig. 2. Forest plot showing functional outcome between low and high RDW group.

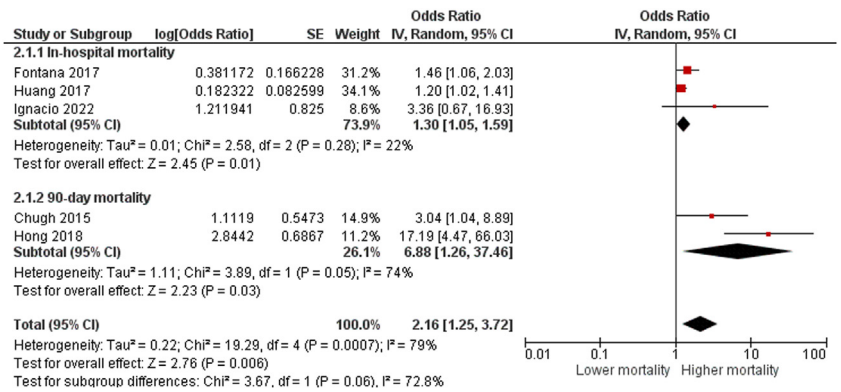

Fig. 3. Forest plot showing mortality between low and high RDW group.

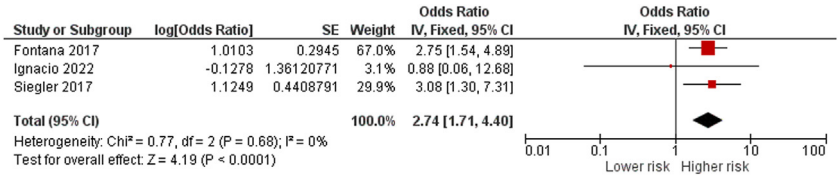

Fig. 4. Forest plot showing cerebral infarction between low and high RDW group.

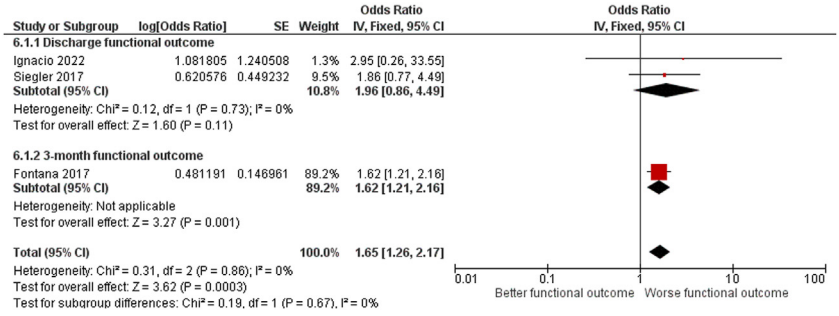

Fig. 5. Forest plot showing the sensitivity analysis for functional outcomes between low and high RDW group.

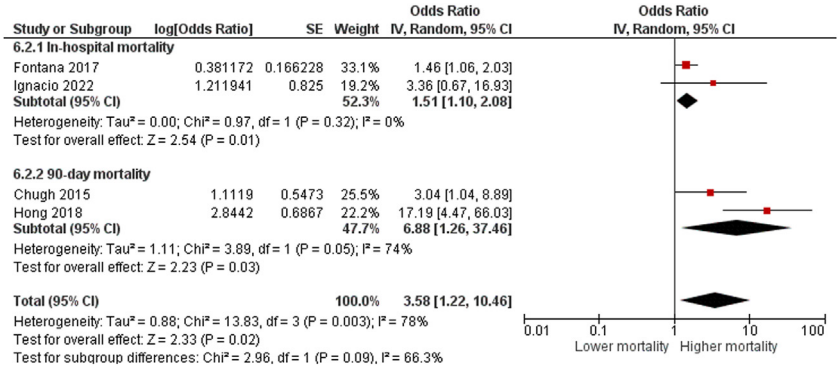

Fig. 6. Forest plot showing the sensitivity analysis for mortality between low and high RDW group.

Table 3
Certainty of evidence.

Outcome	No. of Studies	Study Design	Risk of Bias	Inconsistency	Indirectness	Imprecision	Other Consideration	OR (95% CI)	Overall
Functional outcome	4	Observational	Not serious	Not serious	Not serious	Not serious	None	1.70 (1.32–2.19)	Low
Mortality	4	Observational	Serious[a]	Serious[b]	Not serious	Not serious	Strong association[c]	2.16 (1.25–3.72)	Very low
Cerebral infarction	3	Observational	Not serious	Not serious	Not serious	Not serious	Strong association[c]	2.71 (1.71–4.40)	Moderate

[a] One study rated as having a serious risk of bias. Downgraded by one point.
[b] $I^2 > 40\%$. Downgraded by one point.
[c] OR > 2.0. Upgraded by one point.

correlates with prognosis in cardiovascular diseases and ischemic stroke, which both have a strong inflammatory and thromboembolic component.[8,21,22] One study by Patel et al showed that increased RDW is associated with decreased RBC deformability which could hinder microcirculation.[23]

Additionally, as anisocytosis usually results from chronic impairment of erythropoiesis,[24,25] it is logical to think that RDW reflects a chronic, rather than an acute, process. However, a higher RDW value also correlated with a higher acute-phase inflammatory marker such as hsCRP and ESR, independent of many confounding factors.[7] One interesting study by Kim et al demonstrated that an acute increase in RDW from baseline value was related to a higher mortality rate in patients with septic shock, showing that RDW could also increase in a short amount of time.[26] Therefore, we hypothesized that RDW has both a chronic and acute component, although the complete mechanism has not been elucidated. Another hypothesis is that RDW is a measure of the patient's hypoxic burden, proinflammatory state, and oxidative stresses caused by an intermittent, undiagnosed condition.[27] When the patient suffers from an acute condition, this impairment in their physiology could result in a worse outcome and would explain how RDW is related to the prognosis of many acute conditions, such as aSAH.

In this analysis, we found that a higher RDW value correlated to a higher risk of cerebral infarction with an OR of 2.74 (95% CI 1.71–4.40). Cerebral infarction is a known risk factor for a worse functional outcome and a higher mortality rate.[28,29] While it was believed before that cerebral vasospasm is the sole cause of infarction, recent studies have shown that its pathophysiology is much more complex. Neuroinflammation and microthromboembolism have been proposed as the other possible mechanism behind an infarction.[30] Several studies have found that high levels of nonspecific inflammatory markers such as lactate concentration, hsCRP

levels, ESR, leukocyte count, negative nitrogen balance, and neutrophil-lymphocyte ratio predicted infarction and poor outcome in aSAH.[31–34] Analysis of cerebrospinal fluid (CSF) had also shown that a higher concentration of proinflammatory cytokines IL-6, IL-8, IL-1β, TNF-α, and monocyte chemoattractant protein (MCP)-1 predicted cerebral infarction and poor outcomes.[35–37] Regarding microthromboembolism, several studies have shown that the presenvasce of micro clots correlates with cerebral infarction.[38,39] These micro clots are caused by platelet aggregation mediated by von Willebrand factor (vWF) and P-selectin.[40]

To summarize, inflammation and microthromboembolism have been established to be two of the emerging pathophysiological mechanisms behind cerebral infarction. There is also the possibility that a high RDW value is an epiphenomenon to the presence of systemic inflammation. Nevertheless, as RDW can predict outcomes in many inflammatory and thromboembolic disorders, this could explain its relationship with cerebral infarction.

We also found that a high RDW value correlated with a worse functional outcome with an OR of 1.70 (95% CI 1.32–2.19) and a higher mortality rate with an OR of 2.16 (95% CI 1.25–3.72). As mentioned before, cerebral infarction is the main contributor to poor outcomes. However, there are several other comorbidities with a strong correlation to poor outcomes in aSAH, such as diabetes mellitus, hypertension, chronic kidney disease, pulmonary infection, and anemia.[41–46] The relationship between RDW and those comorbidities have been well established. Two extensive cohort studies have found that a high RDW value correlates with an increased glycated hemoglobin value.[47,48] Several other papers have also pointed out the correlation between RDW and hypertension and chronic kidney disease.[49–51] A higher RDW value has also been found to correlate with the severity and outcome of community-acquired pneumonia.[52,53] In the case of anemia, the most common type of anemia, such as iron deficiency anemia, anemia of chronic disease, and vitamin B_{12} deficiency, results in a higher RDW value.[54] We admitted that the relationship between those comorbidities with RDW might reduce RDW specificity. However, we proposed that this should be seen as an advantage where RDW could reflect the condition of many comorbidities. In some patients, such comorbidities might not be obvious as they could be subclinical or undiagnosed and RDW could help reflect the overall proinflammatory state and link it with the prognosis of an aSAH patient.

An excellent prognostic model is required to accurately determine the severity of aSAH patients and administer treatment accordingly. Several prophylactic treatments for cerebral infarction are currently under research, including but not limited to nimodipine, clazosentan, and heparin.[4–6] The availability of an accurate prognostic model can provide tremendous help in risk stratification before beginning treatment. The currently established predictors for aSAH patients are the HH scale, WFNS scale, and modified Fisher scale.[55,56] These predictors only evaluate the patients' admission clinical and radiological characteristics. Considering that the pathophysiology of aSAH comprises a wide array of biomolecular processes, it is appropriate to find a suitable biomarker that is cheap and easy to examine. Regarding this issue, other new biomarkers have been studied, such as neutrophil-to-lymphocyte ratio (NLR), platelet-to-lymphocyte ratio (PLR), and systemic inflammatory response index (SIRI).[57–59] Unfortunately, only a few studied these parameters alongside RDW, making a comparison between them difficult. One study by Huang et al compared RDW directly with NLR in aSAH patients. They found that RDW had a stronger correlation with in-hospital mortality than NLR (OR 1.39 [95% CI 1.06–1.82] vs OR 1.04 [95% CI 1.00–1.08]).[14] In the study by Ignacio et al, they found through receiver operating characteristic (ROC) curves analysis that RDW had a better ability in predicting cerebral infarction while NLR performed better for functional outcome.[16] Further research is still required to conclusively determine which routine laboratory examination has the best prognostic capability. Nevertheless, we hope that the result of our analysis could prove that RDW also has a promising potential to be included in a prognostic model. Several other prognostic models incorporating laboratory values, baseline characteristics, and comorbidities were currently being developed.[60–62] We suggest future research to incorporate RDW in devising a prognostic model.

Although we have shown that RDW was associated with a higher rate of mortality, cerebral infarction, and worse functional outcome, there are several concerns that need to be discussed. One of the significant challenges in using RDW as a biomarker is the absence of a universal reference range due to the lack of harmonization between manufacturers and laboratories.[63] In the studies included in our analysis, they used the cut-off value set by their respective laboratories' the cut-off value and they varied between 13.4% and 16%. Nevertheless, as our analysis showed that a high RDW value correlated significantly with the poor outcomes of aSAH patients, a cut-off value set independently by laboratories still holds an excellent prognostic capability. Another concern is the lack of agreement regarding the sampling time. Three studies in our analysis used the initial test result on admission, two used serial measurement and used the maximum value over a period of 7–10 days, and one used the mean value over a period of 14 days.[11–16] Two of the three studies that used a serial measurement provided day-to-day data.[11,12] Chugh et al found that day-1, 3, 5, and 7 RDW values correlated significantly to a worse functional outcome.[11] In the study by Fontana et al, they found that admission RDW values were significantly higher in those that developed a cerebral infarction, had a worse functional outcome, and had a higher mortality rate.[12] However, they only included the maximum value from their serial sampling in their logistic regression. Cerebral infarction in aSAH likely develops over a period of days after the ictus. Therefore, although the studies of Chugh et al, Fontana et al, Huang et al, and Hong et al showed that admission RDW had a significant correlation with prognosis,[11,12,14,15] we hypothesized that a serial measurement time is more appropriate. The cheap cost of RDW would not hinder serial measurement, as in the case of other more expensive biomarkers.

Some limitations of our analysis need to be addressed. One limitation is the small number of included studies. Moreover, only single-center studies were available for analysis. However, since the studies' location spanned the United States of America, Philippines, Belgium, Israel, and Belgium, the result of our analysis should have encompassed a wide range of populations. Another limitation is that some of our studies have a potential risk of confounding bias. This bias is especially evident in the study by Huang et al.[14] However, excluding that said study in our sensitivity analysis did not produce a different result. Lastly, regarding cerebral infarction, the included studies did not mention the time window in which it occurred and did not elaborate on how they excluded iatrogenic infarct. This could lead to under or over-reporting of infarction. In the future, more prospective, multi-center, and large-sample studies are needed to investigate this issue further. Additionally, we suggested future studies should adhere to the proposed definition by Vergouwen et al.[10]

5. Conclusion

We found that a high RDW value significantly correlated with the rate of poor functional outcome, mortality, and cerebral infarction. RDW also has the benefit of being cheap and easy to measure. Therefore, RDW examination has the potential to be used in determining the severity and prognosis of aSAH patients. Future studies should incorporate RDW alongside other biomarkers to find the most suitable combination for a prognostic model.

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Declaration of competing interest

The authors declared that there were no commercial or financial relationships that could be construed as a potential conflict of interest.

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