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Admission Red Blood Cell Distribution Width as a Marker of Severe Acute Exacerbation in Patients With Bronchiectasis: A Retrospective Cohort Study

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Abstract

Background: Red cell distribution width (RDW) has been proposed as a marker of systemic inflammation and physiological reserve, but its value in acute exacerbation of bronchiectasis (AEB) remains unclear. This study examined whether admission RDW and RDW-derived indices, including the RDW-to-albumin ratio (RAR) and hemoglobin-to-RDW ratio (HRR), were associated with severe AEB. We also compared their adjusted associations and discriminative performance and developed an RDW-based nomogram.

Methods: We retrospectively included 413 patients hospitalized for AEB at the Fourth Affiliated Hospital of Zhejiang University School of Medicine between September 2021 and September 2024. Severe AEB was defined by in-hospital clinical events and occurred in 122 patients; 291 patients were classified as non-severe. Demographic characteristics, comorbidities, laboratory findings, microbiological results and imaging features were extracted from electronic medical records. Univariable logistic regression and least absolute shrinkage and selection operator (LASSO) regression were used to screen candidate predictors. Marker-specific multivariable logistic models estimated the associations of RDW, RAR and HRR with severe AEB. An RDW-based nomogram was then evaluated with receiver operating characteristic (ROC) curves, calibration analysis and decision curve analysis.

Results: RDW was higher in patients with severe AEB than in those with non-severe AEB. In the adjusted RDW model, RDW remained associated with severe AEB after adjustment for age, chronic obstructive pulmonary disease (COPD), congestive heart failure (CHF), number of involved lung lobes, C-reactive protein and white blood cell count (OR=1.31, 95% CI 1.03-1.71, P=0.036). Single-marker AUCs were 0.650 for RDW, 0.681 for RAR and 0.511 for HRR. RAR showed slightly higher single-marker discrimination, but only RDW retained statistical significance after multivariable adjustment. The RDW-based nomogram showed good internal discrimination (AUC=0.875, 95% CI 0.842-0.908), acceptable calibration and favorable net benefit in internal evaluation.

Conclusions: Admission RDW was independently associated with severe AEB and performed more consistently than RAR or HRR after adjustment. RDW alone had limited discrimination, but its combination with routine clinical variables may support early inpatient risk stratification. External validation is required before clinical implementation.

Background

Bronchiectasis is a chronic airway disease marked by irreversible bronchial dilatation, persistent airway inflammation and recurrent infection.(1, 2) Acute exacerbations shape much of its clinical burden.(3, 4) Severe episodes may lead to hospitalization, ventilatory support or intensive care and can accelerate functional decline.(5) Identifying patients at risk of early deterioration is therefore a practical priority in AEB management.(6)

AEB severity varies widely.(7–9) Some patients present with symptom worsening that stabilizes quickly, whereas others develop respiratory failure, altered mental status or hemodynamic instability. Bedside

assessment still depends heavily on clinical judgement. Routine laboratory markers available soon after admission may therefore add value, particularly where airway biomarkers are not available.(10–12) Sputum neutrophil elastase and neutrophil serine protease activity capture important exacerbation biology and are now therapeutically targetable, but most hospitals do not yet use them for routine triage.(13, 14)

Red cell distribution width (RDW), a routinely reported complete blood count parameter, reflects heterogeneity in erythrocyte size.(15) Although RDW was developed for hematologic assessment, it is now widely studied as a marker of systemic illness.

Higher RDW has been linked to inflammation, oxidative stress, nutritional impairment, iron deficiency and disordered erythropoiesis, all of which may accompany acute respiratory deterioration.(16) Elevated RDW has also been associated with adverse outcomes in COPD, community-acquired pneumonia, sepsis, heart failure and viral respiratory disease.

RDW-derived indices combine erythrocyte heterogeneity with related physiological domains. RAR integrates RDW with serum albumin and may capture inflammatory and nutritional stress.(17) HRR combines hemoglobin concentration with erythrocyte size variability and has been evaluated as a prognostic marker in acute and chronic diseases.(18) Evidence for these markers in bronchiectasis remains sparse.(19) The independent value of RAR and HRR in severe AEB is unclear, and few studies have translated RDW-related markers into a clinically interpretable early risk model.

We investigated the associations of RDW, RAR and HRR with severe AEB in hospitalized patients with bronchiectasis. We then identified independent predictors of severe AEB and developed an RDW-based nomogram for individualized risk estimation.

Methods

Study design

This single-center retrospective cohort study used electronic medical record data from the Fourth Affiliated Hospital of Zhejiang University School of Medicine. Consecutive patients hospitalized for AEB between September 1, 2021, and September 1, 2024, were screened. If a patient had more than one admission during the study period, only the first hospitalization was analyzed. The study evaluated RDW and two RDW-derived indices, RAR and HRR, in relation to severe AEB.

Ethics statement

This study was approved by the Ethics Committee of the Fourth Affiliated Hospital of Zhejiang University School of Medicine (approval No. [insert approval number]). Because of the retrospective design and the

use of anonymized clinical data, the requirement for written informed consent was waived. The study was conducted in accordance with the Declaration of Helsinki.

Participants

Patients were eligible if they were aged 18 years or older, had non-cystic fibrosis bronchiectasis confirmed by high-resolution computed tomography, were hospitalized for AEB, and underwent routine blood testing and serum albumin measurement within 24 h of admission. Outcome data and admission RDW, hemoglobin and serum albumin values were required for the primary analyses. Exclusion criteria were immune-related connective tissue disease, immunodeficiency or ongoing immunosuppressive therapy; severe coexisting lung disease, including lung cancer, interstitial lung disease, active tuberculosis, destroyed lung, pulmonary cavities or previous lung surgery; severe liver or renal insufficiency; malignant tumor history; pregnancy; severe cognitive impairment or inability to cooperate with treatment; self-discharge or transfer within 24 h of admission; genetic defects or chromosomal abnormalities; blood transfusion within 3 months before admission; and known hematologic disease.

Data collection

Demographic characteristics, clinical features, comorbidities, laboratory findings, microbiological results and imaging data were extracted from electronic medical records. Demographic and clinical variables included age, sex, body mass index, smoking status, previous exacerbation or hospitalization history, and comorbidities including COPD, asthma, heart failure, diabetes, chronic kidney disease and liver disease. Laboratory variables came from the first test performed within 0–24 h after admission and included white blood cell count, neutrophil count, lymphocyte count, hemoglobin, RDW, serum albumin, CRP, procalcitonin and arterial blood gas parameters. Microbiological data were based on sputum or blood culture results, with particular attention to Pseudomonas aeruginosa detection.(20) Imaging variables included the number of involved lung lobes and bilateral involvement on high-resolution chest CT. RDW was expressed as RDW-CV. RAR was calculated as RDW-CV divided by serum albumin, and HRR was calculated as hemoglobin divided by RDW-CV.

Outcomes

The primary endpoint was severe AEB, defined as the occurrence of at least one of the following events during hospitalization: ICU admission, non-invasive or invasive mechanical ventilation, respiratory failure, or in-hospital death. Patients who did not meet any of these criteria were classified as having non-severe AEB.

Statistical analysis

Continuous variables were summarized as mean +/- standard deviation or median with interquartile range according to distribution. Categorical variables were reported as counts and percentages. Between-group comparisons used the t test, Wilcoxon rank-sum test, chi-square test or Fisher's exact test, as appropriate. Univariable logistic regression identified candidate variables associated with severe AEB. Variables with $P < 0.10$ entered the candidate pool for subsequent modelling.

The extent of missing data was assessed before modelling. Variables with substantial missingness or limited clinical availability were not used as core predictors in the final nomogram. The main analyses were performed using patients with available data for the variables included in each model. Because RDW, hemoglobin and serum albumin were required to calculate the exposure variables, patients without these measurements within 24 h of admission were not included. Missingness was reported descriptively where relevant, and no imputation was performed for the primary analysis.

RDW, RAR and HRR were not entered into the same multivariable model because they are mathematically related. Three marker-specific models were built, each using RDW, RAR or HRR as the core exposure. Candidate variables identified in univariable analysis were further screened with LASSO regression. In each marker-specific model, the RDW-related marker was retained as the exposure of interest, while clinical covariates were selected with the lambda.1se criterion to obtain a parsimonious covariate set. Variance inflation factors were used to assess collinearity. Smoking status and Pseudomonas aeruginosa detection were forced into the RDW model in sensitivity analyses because they were clinically plausible confounders. Model discrimination was evaluated with ROC curves and AUCs. Calibration of the final RDW-based nomogram was assessed with calibration curves and 1,000 bootstrap resamples.(21) Clinical usefulness was examined with decision curve analysis.(22) All analyses were performed using R software (version [specify version]), and two-sided $P < 0.05$ was considered statistically significant.

Results

Baseline characteristics

The cohort included 413 hospitalized patients with AEB, including 291 with non-severe AEB and 122 with severe AEB. Patients in the severe AEB group were older [72.00 (66.00, 78.00) vs. 64.00 (53.00, 71.00) years, $P < 0.001$] and more often male (54% vs. 42%, $P = 0.036$). Former smoking was more common in the severe group (24% vs. 14%; overall smoking-status $P = 0.045$). COPD and CHF also occurred more often in the severe group (COPD: 65% vs. 19%, $P < 0.001$; CHF: 44% vs. 8.6%, $P < 0.001$). Baseline characteristics are summarized in Table 1.

Laboratory and imaging profiles differed between groups. Patients with severe AEB had lower serum albumin [3.72 (3.34, 4.04) vs. 3.86 (3.60, 4.07), $P < 0.001$], higher RDW [13.30 (12.60, 14.40) vs. 12.90 (12.40, 13.30), $P < 0.001$] and higher RAR [3.68 (3.33, 4.16) vs. 3.35 (3.13, 3.63), $P < 0.001$]. HRR did not differ significantly [0.98 (0.86, 1.10) vs. 0.99 (0.89, 1.08), $P = 0.7$]. WBC, neutrophil count, CRP and

procalcitonin were higher in the severe group, whereas lymphocyte count was lower. Arterial blood gas results showed lower PaO2 and PaO2/FiO2 and higher PaCO2. Pseudomonas aeruginosa detection was more frequent in severe AEB (25% vs. 11%, P = 0.001). Severe AEB was also associated with more involved lung lobes and a higher frequency of bilateral disease (87% vs. 64%, P < 0.001). Marker distributions are shown in Fig. 1.

All 122 patients with severe AEB had respiratory failure. ICU admission occurred in 17 patients, 35 received mechanical ventilation and no in-hospital deaths were recorded.

Univariate analysis

In univariable logistic regression, COPD (OR = 7.88, 95% CI 4.94–12.76, P < 0.001), CHF (OR = 8.45, 95% CI 4.96–14.76, P < 0.001), number of involved lung lobes (OR = 1.75, 95% CI 1.48–2.09, P < 0.001) and age (OR = 1.07, 95% CI 1.05–1.09, P < 0.001) were associated with higher odds of severe AEB.

RAR showed the strongest univariable association among the RDW-related indices (OR = 3.56, 95% CI 2.36–5.56, P < 0.001). RDW was also associated with higher odds of severe AEB (OR = 1.76, 95% CI 1.44–2.21, P < 0.001), whereas HRR was not (OR = 0.53, 95% CI 0.15–1.92, P = 0.331). CRP, WBC, Pseudomonas aeruginosa detection, former smoking and male sex were also associated with severe AEB. Key univariable results are shown in Table 2.

LASSO analysis

RDW, RAR and HRR were not entered into the same multivariable model because they are mathematically related. Three marker-specific models were built, each using RDW, RAR or HRR as the core exposure. Candidate variables identified in univariable analysis were further screened with LASSO regression. In each marker-specific model, the RDW-related marker was retained as the exposure of interest, while clinical covariates were selected with the lambda.1se criterion to obtain a parsimonious covariate set. Variance inflation factors were used to assess collinearity. Smoking status and Pseudomonas aeruginosa detection were forced into the RDW model in sensitivity analyses because they were clinically plausible confounders. Model discrimination was evaluated with ROC curves and AUCs. Calibration of the final RDW-based nomogram was assessed with calibration curves and 1,000 bootstrap resamples.(21) Clinical usefulness was examined with decision curve analysis.(22) All analyses were performed using R software (version [specify version]), and two-sided P < 0.05 was considered statistically significant.

Multivariate analysis

After adjustment for age, COPD, CHF, number of involved lung lobes, CRP and WBC, RDW remained associated with severe AEB (OR = 1.31, 95% CI 1.03–1.71, P = 0.036). Age, COPD, CHF and number of involved lung lobes were also independently associated with severe AEB in the RDW model.

RAR was not independently associated with severe AEB in the multivariable RAR model (OR = 1.38, 95% CI 0.82–2.38, P = 0.238). HRR also showed no independent association in the HRR model (OR = 3.08, 95% CI 0.55–17.76, P = 0.204). The adjusted association with severe AEB was therefore more stable for RDW than for the two derived indices. Marker-specific multivariable models are summarized in Table 3 and Fig. 2.

Sensitivity analyses for smoking status and Pseudomonas aeruginosa

Sensitivity analyses examined whether smoking status or Pseudomonas aeruginosa detection influenced the RDW association. Median RDW was 13.2 (12.6, 14.1) in patients with Pseudomonas aeruginosa detection and 12.9 (12.5, 13.5) in those without detection. By smoking status, median RDW was 12.9 (12.5, 13.5) in never smokers, 12.8 (12.5, 13.3) in current smokers and 13.1 (12.4, 13.7) in former smokers. In linear regression, smoking status and Pseudomonas aeruginosa detection were not independently associated with RDW, whereas COPD and CHF were associated with higher RDW.

When smoking status and Pseudomonas aeruginosa detection were added to the multivariable RDW model, RDW remained independently associated with severe AEB (OR = 1.31, 95% CI 1.03–1.71, P = 0.037). Current smoking, former smoking and Pseudomonas aeruginosa detection were not independently associated with severe AEB in this model. All adjusted VIF values were low (maximum adjusted VIF, 1.22), indicating no substantial multicollinearity.

ROC analysis

Single-marker ROC analysis showed modest discrimination for RDW and RAR and weak discrimination for HRR. AUCs were 0.650 (95% CI 0.589–0.711) for RDW, 0.681 (95% CI 0.620–0.741) for RAR and 0.511 (95% CI 0.445–0.577) for HRR. Discrimination improved after clinical variables were incorporated. AUCs were 0.875 (95% CI 0.842–0.908) for the RDW model, 0.872 (95% CI 0.839–0.906) for the RAR model and 0.874 (95% CI 0.842–0.907) for the HRR model. The three multivariable models showed similar discrimination, with only small AUC differences. ROC curves are shown in Fig. 3.

Nomogram analysis

An RDW-based nomogram was developed from the multivariable logistic regression model. The model included RDW, age, COPD, CHF, number of involved lung lobes, CRP and WBC. Each predictor was assigned a point value, and the total score corresponded to the predicted probability of severe AEB. This format provided a visual way to combine RDW with routine clinical variables. The nomogram is shown in Fig. 4.

Calibration analysis

Calibration analysis was used to compare the predicted probabilities from the RDW nomogram with the observed risk of severe AEB. The calibration curve was generally close to the ideal reference line. After bootstrap internal validation, the model maintained acceptable calibration, with a mean absolute error of 0.041.

Decision curve analysis

Decision curve analysis assessed the potential clinical utility of the RDW-based nomogram. Across a range of threshold probabilities, the model provided greater net benefit than treat-all or treat-none strategies within this retrospective cohort. Calibration and decision curve analyses are shown in Fig. 5.

Discussion

In this retrospective cohort of 413 patients hospitalized for AEB, admission RDW was higher in severe AEB and remained associated with severity after adjustment for age, COPD, CHF, number of involved lung lobes, CRP and WBC. The association persisted after further adjustment for smoking status and Pseudomonas aeruginosa detection. RAR showed slightly higher single-marker discrimination but lost independent significance after adjustment. HRR showed limited discriminatory and adjusted prognostic value. These findings position RDW as a simple severity-associated marker rather than a stand-alone predictor.

The association between RDW and severe AEB may reflect systemic inflammatory burden, oxidative stress and impaired cardiopulmonary reserve during acute deterioration. RDW increases when erythropoiesis is disturbed by inflammatory cytokines, oxidative injury, nutritional impairment or chronic disease burden. In bronchiectasis, exacerbations often intensify airway inflammation, bacterial infection, hypoxaemia and systemic inflammatory responses.(23, 24) These processes can affect red blood cell maturation and survival, providing a plausible link between AEB severity and RDW.

This interpretation fits the biology of bronchiectasis. Acute deterioration occurs on a background of chronic airway infection, mucus retention, impaired mucociliary clearance and recurrent neutrophil-dominant inflammation. Recent studies show that bronchiectasis exacerbations are biologically heterogeneous events, with inflammatory and microbial endotypes that may differ from stable-state disease.(8, 10, 23, 24) Neutrophil serine proteases, airway microbiome instability and radiological extent may amplify epithelial injury and systemic inflammatory signalling. RDW may therefore capture part of this disease-specific inflammatory load rather than nonspecific inflammation alone.(12, 14, 25)

These findings align with broader evidence linking RDW to adverse outcomes in inflammatory, infectious and cardiopulmonary diseases.(26) Elevated RDW has been associated with severity and poor prognosis in COPD, pneumonia, sepsis and heart failure, conditions in which inflammation, oxidative stress and impaired oxygen transport often coexist.(27) The present study extends this concept to hospitalized AEB, where the outcome of interest was severe in-hospital deterioration rather than long-term prognosis. The persistence of the RDW association after adjustment for comorbidities, inflammatory markers and

radiological extent suggests that RDW carries information not fully captured by conventional clinical variables.

RDW showed a more stable adjusted association with severe AEB than the derived indices. RAR was higher in severe AEB and had the highest single-marker AUC, a pattern consistent with the combined information from RDW and albumin. After adjustment for clinical severity factors and inflammatory markers, however, RAR no longer remained significant. Its apparent prognostic value may therefore overlap with systemic inflammation, nutritional impairment and comorbidity burden.(28, 29) HRR performed least well, possibly because hemoglobin was not the dominant severity signal in this cohort and because the HRR denominator reintroduces RDW in an inverse mathematical form.

The weaker adjusted performance of RAR also has a clinical explanation. Albumin is influenced by acute inflammation, fluid status, hepatic synthesis, nutrition and chronic comorbidity. In an acute hospitalization cohort, this denominator may add context-sensitive variability and may duplicate information already represented by CRP, WBC, comorbid disease and radiological extent. RDW, in contrast, is a direct complete-blood-count parameter and may summarize cumulative inflammatory and oxidative stress more consistently.

RDW alone had modest predictive performance (AUC = 0.650), so it should not be used as an isolated triage test. Its value emerged when combined with age, COPD, CHF, number of involved lung lobes, CRP and WBC. The final RDW-based model showed good internal discrimination (AUC = 0.875), acceptable calibration after internal validation and potential net benefit across clinically relevant thresholds. Because RDW is inexpensive, routinely measured and available shortly after admission, it may help flag patients who need closer observation or earlier respiratory assessment.

The model should be viewed alongside established bronchiectasis severity tools. The Bronchiectasis Severity Index, FACED and E-FACED scores stratify longer-term risks such as mortality, hospitalization and future exacerbations and include relatively stable clinical, functional, microbiological and radiological variables.(30–32) Our model addresses a narrower inpatient question: whether admission data can help identify patients at risk of severe in-hospital AEB events. RDW is therefore best framed as an acute, low-cost adjunct to existing severity frameworks, not as a replacement for them.

The clinical use case is admission triage. RDW could contribute to an early warning profile when complete blood count, CRP and basic comorbidity information are available before specialist bronchiectasis assessment. Patients with elevated RDW together with older age, COPD, CHF, multilobar involvement and increased inflammatory markers may need closer monitoring, earlier arterial blood gas assessment, proactive oxygen or ventilatory planning and timely senior respiratory review. A normal RDW should not reassure clinicians on its own, because severe AEB is multifactorial and single-marker discrimination was modest.

Former smoking and Pseudomonas aeruginosa detection were associated with severe AEB in univariable analysis, but neither variable was retained by LASSO selection or remained significant after

forced inclusion in the sensitivity model. This is clinically plausible. Smoking history may partly reflect older age, COPD and reduced respiratory reserve, while Pseudomonas aeruginosa detection may mark advanced airway structural disease, recurrent infection or greater inflammatory burden.(33–35) After COPD, CHF, lobar involvement, CRP and WBC entered the model, smoking status and Pseudomonas aeruginosa detection added little independent information. Their lack of selection should not be read as clinical irrelevance; it indicates limited incremental value beyond the core severity-related variables in this cohort. RDW remained associated with severe AEB after these variables were forced into the model, supporting the robustness of the finding.

This study has several strengths. The endpoint captured clinically meaningful severe AEB events, including ICU admission, mechanical ventilation, respiratory failure and in-hospital death. RDW, RAR and HRR were evaluated in separate models to avoid collinearity from their mathematical relationships. The analysis combined variable selection, multivariable regression, sensitivity analysis, ROC analysis, calibration assessment and decision curve analysis, allowing RDW to be assessed as both an associated marker and a candidate risk-stratification feature.

The study does not establish RDW as a causal mediator of severe AEB. RDW may integrate inflammatory stress, oxidative injury, hypoxaemia, malnutrition, anaemia biology and comorbidity burden rather than drive acute respiratory deterioration. Experimental work supports oxidative stress as a contributor to anisocytosis, but this retrospective study cannot separate causal pathways from disease-load signalling.(15) RDW should therefore be described as a severity-associated biomarker and risk-stratification feature, not as a therapeutic target.

Several limitations warrant attention. The single-center retrospective design leaves room for residual confounding. Although the severe AEB definition included several clinically important events, all severe cases in this cohort had respiratory failure and no in-hospital deaths occurred. The model should therefore not be interpreted as a mortality or ICU-admission prediction tool. Some variables, including blood gas parameters, procalcitonin, BMI and hospitalization cost, had missing data, which may introduce information bias. The nomogram underwent internal validation only.(36) Its clinical performance should be confirmed in multicenter prospective cohorts.(37) Mechanistic biomarkers, including iron metabolism indices, oxidative stress markers and inflammatory cytokines, were not available, so the biological link between RDW and severe AEB remains inferential.

Future directions

Future work should move beyond internal statistical validation. Multicenter prospective cohorts should externally validate the RDW-based model, compare it directly with BSI, FACED and E-FACED in acute-care settings, and test whether RDW improves calibration, net benefit and clinical decisions beyond established variables. Mechanistic studies could pair RDW with longitudinal inflammatory markers, neutrophil protease activity, microbiome profiling, oxygenation indices and nutritional measures. This would help clarify whether RDW reflects airway endotype, systemic inflammation, hypoxaemic stress or

overall disease burden. Impact studies are also needed to test whether RDW-informed triage changes monitoring intensity, escalation timing or patient outcomes.

In conclusion, elevated admission RDW was independently associated with severe AEB and remained stable after adjustment for major clinical confounders. RDW alone had limited discriminatory ability, but an RDW-based multivariable nomogram may support early inpatient risk stratification. Prospective external validation and mechanistic studies are needed before RDW-based risk assessment can be adopted in routine care.

Conclusions

In this retrospective cohort of hospitalized patients with AEB, admission RDW was independently associated with severe disease after adjustment for clinical and inflammatory factors. RDW showed a more stable adjusted association than RAR or HRR. A nomogram combining RDW with age, COPD, CHF, lobar involvement, CRP and WBC showed good apparent discrimination, acceptable internal calibration and potential net benefit. These findings support RDW as part of a multivariable risk-stratification approach rather than as a stand-alone severity marker. Because the study was single-center and retrospective, prospective multicenter validation is needed before clinical implementation.

Abbreviations

AEB, acute exacerbation of bronchiectasis;

AUC, area under the curve;

CHF, congestive heart failure;

COPD, chronic obstructive pulmonary disease;

CRP, C-reactive protein;

DCA, decision curve analysis;

HRR, hemoglobin-to-RDW ratio;

LASSO, least absolute shrinkage and selection operator;

PA, Pseudomonas aeruginosa;

RAR, RDW-to-albumin ratio;

RDW, red cell distribution width;

ROC, receiver operating characteristic;

WBC, white blood cell count.

Declarations

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Authors' contributions

DDC, YL and ZHX designed the study. DDC drafted the manuscript and developed the statistical analysis plan. YL, KKZ and YG collected the clinical data. ZHX obtained funding and revised the manuscript. All authors read and approved the final manuscript.

Data availability

The original data supporting this study are stored in the hospital electronic medical record system. Further inquiries may be directed to the corresponding author. Aggregated data supporting the baseline characteristics, regression analyses, model performance analyses and sensitivity analyses are provided in the supplementary source tables. The individual-level clinical dataset and model-predicted probabilities contain potentially identifiable clinical information and are not publicly available because of patient privacy and institutional data governance restrictions.

Clinical trial number

Not applicable.

Ethics approval and consent to participate

This study was approved by the Ethics Committee of the Fourth Affiliated Hospital of Zhejiang University School of Medicine (approval No. [YJ260610144069]). Because of the retrospective design and the use of anonymized clinical data, the requirement for written informed consent was waived.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing interests.

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Tables

Tables 1 to 3 are available in the supplementary files section

Figures

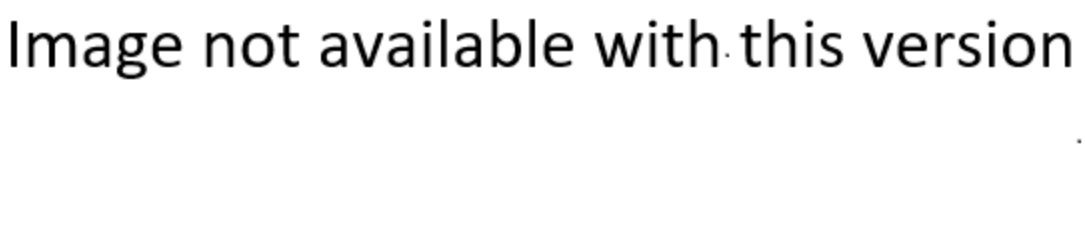


Figure 1

Distribution of admission RDW-related markers by AEB severity. Violin plots show marker distributions, box plots show median and interquartile range, and points represent individual patients.

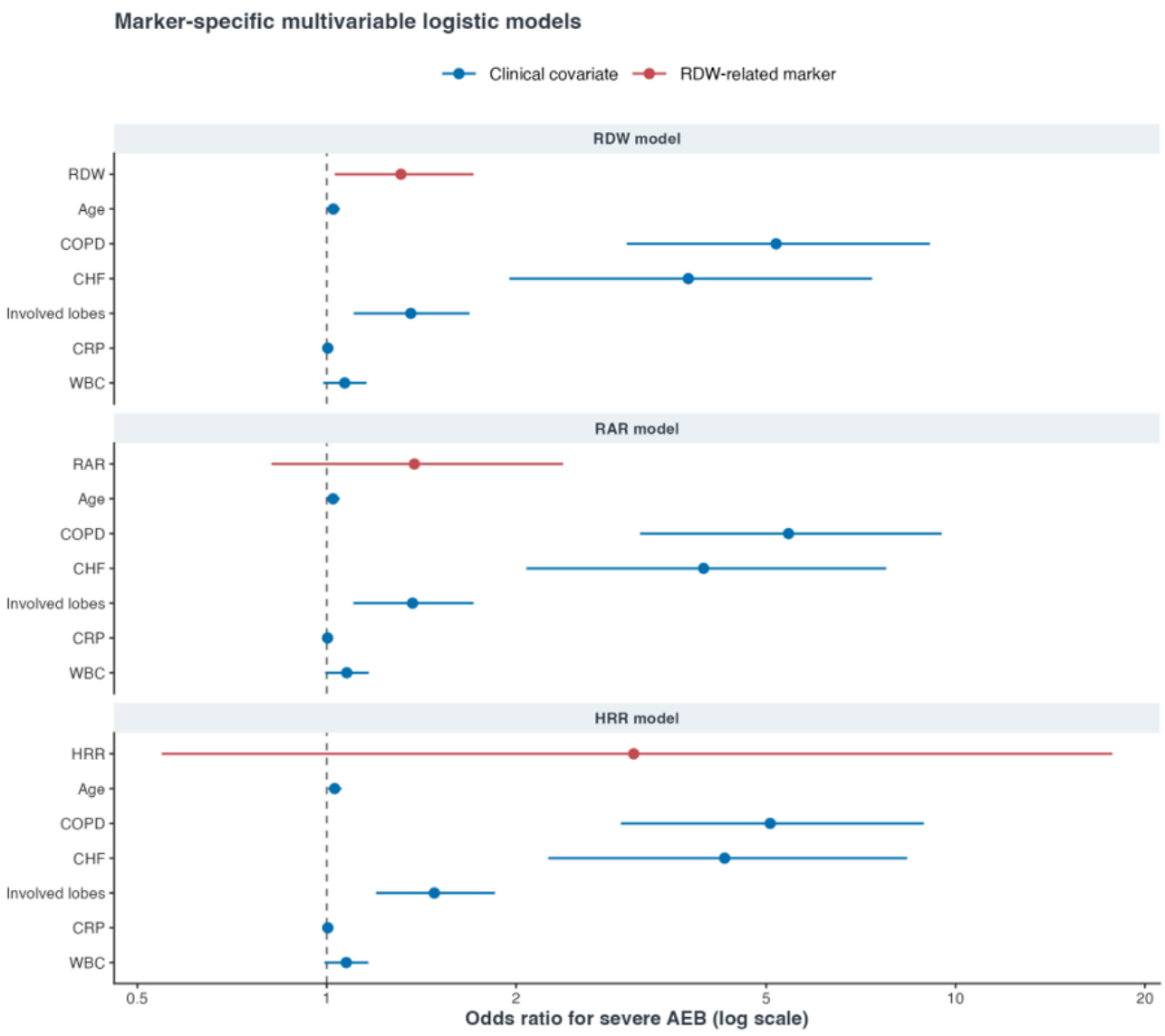


Figure 2

Marker-specific multivariable logistic regression models for severe AEB. Odds ratios are shown on a logarithmic scale. Red points indicate the RDW-related marker in each model; blue points indicate clinical covariates.

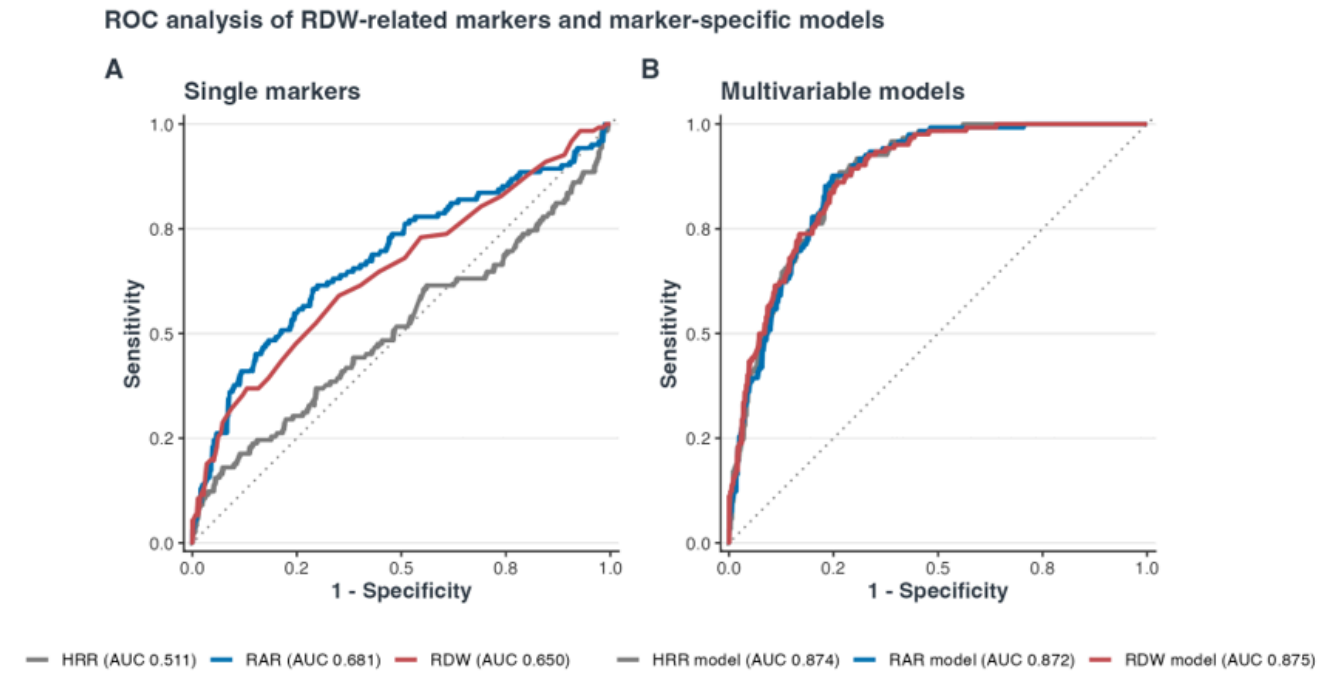


Figure 3

Receiver operating characteristic curves for RDW-related markers and models. RAR showed the highest single-marker AUC, whereas the three multivariable models showed similar discrimination.

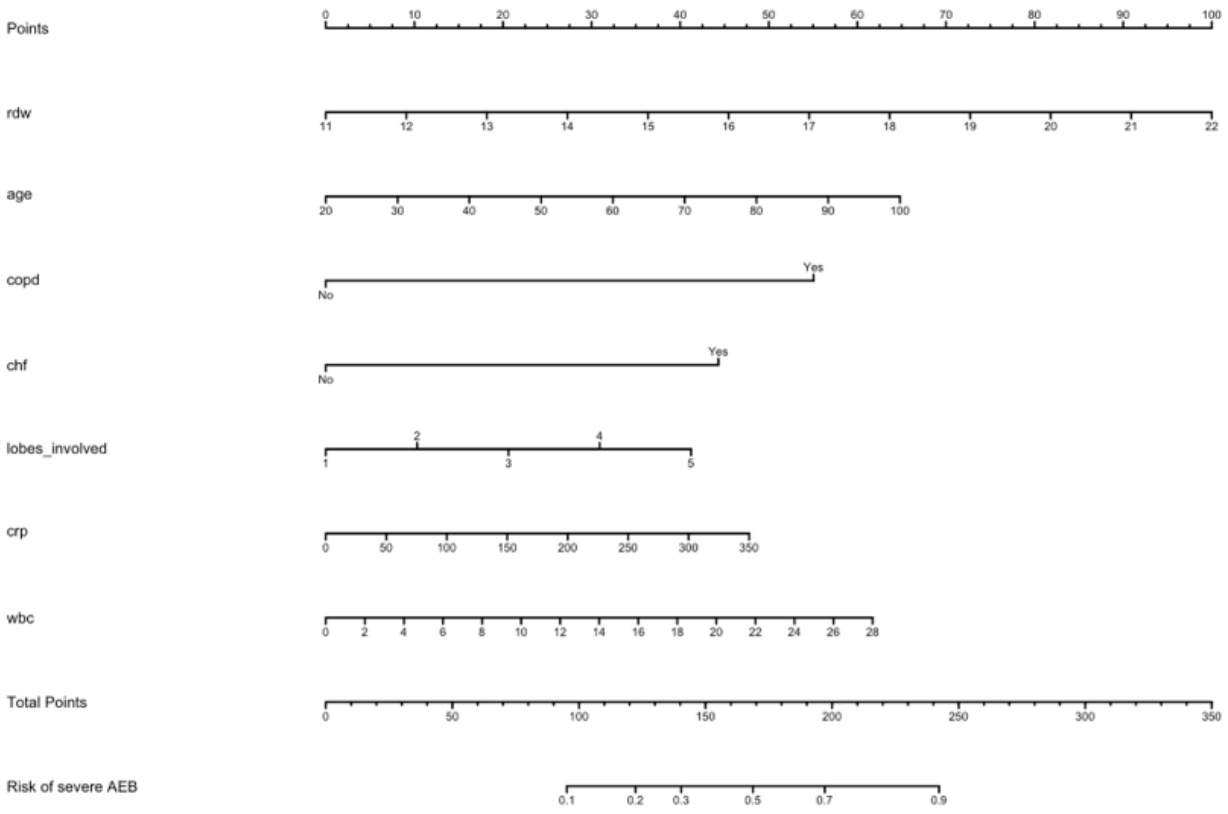


Figure 4

Nomogram for estimating the probability of severe AEB. The model incorporated RDW, age, COPD, CHF, number of involved lung lobes, CRP and WBC.

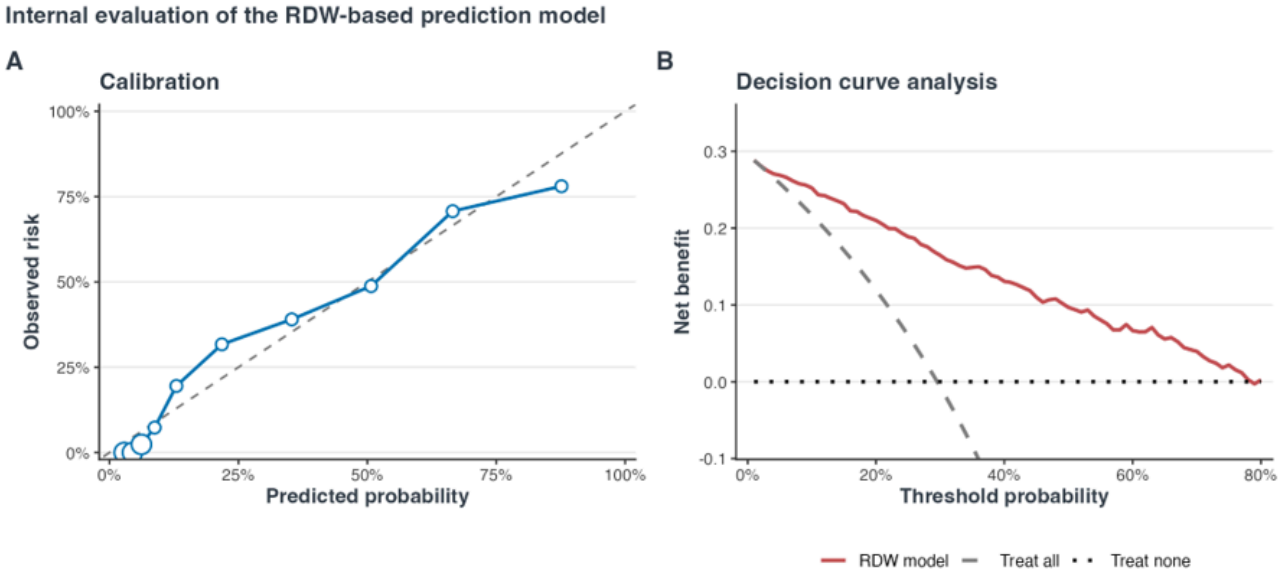


Figure 5

Internal evaluation of the RDW-based prediction model. Panel A shows calibration by deciles of predicted probability. Panel B shows decision curve analysis for the RDW model compared with treat-all and treat-none strategies.

Supplementary Files

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- Tables.docx