

Association between Rheumatoid Factor Levels and Disease Activity in Patients with Rheumatoid Arthritis in Makassar: A Cross-Sectional Study

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ABSTRACT

Background: Rheumatoid factor (RF) is an important autoantibody in the classification and prognosis of rheumatoid arthritis (RA), but the relationship between RF level and contemporaneous disease activity remains inconsistent.

Objective: To evaluate the association between RF levels and disease activity measured by the Disease Activity Score 28-C-reactive protein (DAS28-CRP) in treated adult patients with RA in Makassar, Indonesia.

Methods: This analytical cross-sectional study included 73 adults with RA consecutively recruited between July 2025 and April 2026. All participants had received disease-modifying antirheumatic drug therapy for at least 6 months. RF was analyzed as a continuous variable and using a positivity threshold of ≥ 18 IU/mL. Values below the laboratory reporting limit were recorded as <10 IU/mL. Spearman correlation was the primary analysis because of the non-normal and left-censored RF distribution. Multivariable linear regression adjusted for age, sex, and disease duration; current corticosteroid use was added as a sensitivity analysis. For regression, RF values reported as <10 IU/mL were pragmatically substituted with 10 IU/mL, with 5 IU/mL used in a sensitivity analysis.

Results: Mean age was 36.77 ± 12.41 years and 62/73 (84.9%) participants were women. RF was positive in 46/73 (63.0%), whereas 26 (35.6%) had RF <10 IU/mL. Mean DAS28-CRP was 3.86 ± 1.17 , and moderate disease activity was the most frequent category (53.4%). RF correlated weakly and positively with DAS28-CRP (Spearman $\rho=0.258$; $p=0.028$). After adjustment for age, sex, and disease duration, each 10-IU/mL increase in RF was associated with a 0.126-point higher DAS28-CRP score (95% CI 0.016-0.236; $p=0.025$). After current corticosteroid use was added, the RF association was attenuated and was no longer statistically significant ($\beta=0.073$; 95% CI -0.042 to 0.189; $p=0.214$).

Conclusion: Higher RF levels were weakly associated with higher RA disease activity. The association persisted after adjustment for demographic factors and disease duration but was attenuated after accounting for current corticosteroid use. RF should therefore be interpreted as an adjunctive serological marker rather than a substitute for composite disease-activity assessment.

Keywords: rheumatoid arthritis; rheumatoid factor; DAS28-CRP; disease activity; corticosteroids

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INTRODUCTION

Rheumatoid arthritis (RA) is a chronic inflammatory autoimmune disease that primarily affects synovial joints and can lead to pain, swelling, cartilage destruction, bone erosion, deformity, functional impairment, and disability. Diagnosis and management require integration of clinical findings, serology, acute-phase reactants, and structured assessment of disease activity [1,2].

Treat-to-target strategies identify remission or low disease activity as the principal therapeutic goals. The Disease Activity Score 28 (DAS28) is widely used to monitor RA

because it combines joint assessment, an acute-phase reactant, and the patient global assessment [3,4]. Indonesian national data indicate that only a limited proportion of patients with RA achieve remission, underscoring the importance of regular disease-activity assessment in routine practice [5].

Rheumatoid factor (RF), particularly the IgM isotype, is an autoantibody directed against the Fc portion of immunoglobulin G. In addition to its role in RA classification, RF has been associated with more severe disease phenotypes and structural prognosis. However, RF

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levels do not consistently change in parallel with clinical activity. Aletaha and colleagues reported an association between RF and higher disease activity, whereas other studies suggest that the relationship between autoantibody levels and disease activity depends on the inflammatory context and patient characteristics [6,7].

Local data on the relationship between quantitative RF levels and RA disease activity in Makassar remain limited. This study therefore aimed to evaluate the association between RF level and DAS28-CRP, determine whether the association persisted after adjustment for age, sex, and disease duration, and explore the influence of current corticosteroid use on that relationship.

METHODS

Study design, setting, and participants

This was an analytical cross-sectional study conducted at the rheumatology outpatient clinics of Hasanuddin University Hospital and Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia. Adults with an established diagnosis of RA were consecutively recruited between July 2025 and April 2026. The diagnosis was documented by a rheumatologist and was based on the 2010 American College of Rheumatology/European League Against Rheumatism (ACR/EULAR) classification criteria when the required classification data were available, or on specialist rheumatologist assessment when complete classification variables were not available in the clinical record [2].

Eligible participants were aged ≥ 18 years and had received at least one disease-modifying antirheumatic drug (DMARD) for a minimum of 6 months. Patients with another autoimmune disease or a non-RA pain syndrome were excluded. Seventy-three participants were included in the final analysis. In the analytical dataset, all participants were receiving methotrexate (MTX): 44 were receiving MTX without corticosteroids and 29 were receiving MTX plus corticosteroids. Consequently, the study primarily represents treated RA outpatients rather than treatment-naïve or early untreated RA.

Clinical and laboratory measurements

Disease activity was assessed at the study visit using DAS28-CRP and categorized as remission, low, moderate, or high activity according to the prespecified study protocol. Assessment was performed by rheumatology clinicians to maintain consistency across participants.

C-reactive protein (CRP) was measured using a turbidimetric agglutination method. The laboratory reference range defined CRP <10.0 mg/L (<1.0 mg/dL) as normal. Rheumatoid factor was measured as IgM RF using an enzyme-linked immunosorbent assay (ELISA), with a positivity threshold of ≥ 18 IU/mL according to the study laboratory protocol. RF values below the laboratory reporting limit were recorded as <10 IU/mL. Because the actual concentrations below this limit were unknown, these observations were treated as tied values at the lower end of the distribution for rank-based analyses.

Statistical analysis

Categorical data are presented as frequencies and percentages. Numerical data are presented as mean $\pm$ standard deviation when approximately normally distributed and as median and interquartile range (IQR) when non-normally distributed. The primary association between RF and DAS28-CRP was evaluated using Spearman rank correlation because RF showed a non-normal distribution and because a substantial proportion of RF values were left-censored below the reporting limit. Differences in RF distribution across the four disease-activity categories were evaluated using the Kruskal-Wallis test. As a secondary analysis, mean DAS28-CRP was compared between RF-positive and RF-negative participants using Welch's t test.

Adjusted associations between RF and DAS28-CRP were explored using multivariable linear regression. The main model included age, sex, and disease duration as prespecified clinically relevant covariates. Covariate selection was deliberately parsimonious because of the modest sample size. Current corticosteroid use was added in a sensitivity model because treatment intensity may be closely related to contemporaneous disease activity; the corticosteroid coefficient was therefore interpreted as an association rather than a causal treatment effect because confounding by indication is likely in a cross-sectional design. For numerical regression analyses, RF values reported as <10 IU/mL were pragmatically substituted with 10 IU/mL in the main model; an additional sensitivity analysis substituted 5 IU/mL. These substitutions were used only to enable numerical modeling and do not imply that the true concentrations were 10 or 5 IU/mL. RF coefficients are reported per 10-IU/mL increase. A two-sided p value <0.05 was considered statistically significant.

Ethics

The study protocol received ethical approval from the Research Ethics Committee of Universitas Hasanuddin, Hasanuddin University Hospital, and Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia (Protocol No. UH26060694; Approval No. 885/UN4.6.4.5.31/PP36/2026; 20 July 2026). Written informed consent was obtained from all participants in accordance with the study protocol.

RESULTS

Participant characteristics

A total of 73 patients with RA were analyzed. Mean age was 36.77 ± 12.41 years (range 19-61 years), and most participants were women (62; 84.9%). Median disease duration was 2 years (IQR 1-5 years). The most frequently represented ethnic groups were Makassar (50.7%) and Bugis (41.1%). All participants were receiving MTX; 44 (60.3%) were receiving MTX without corticosteroids and 29 (39.7%) were receiving MTX plus corticosteroids. The main participant characteristics are summarized in Table 1.

Table 1. Participant characteristics (n=73)

Variable	Value
Age, years	36.77 ± 12.41
Women	62 (84.9%)
Men	11 (15.1%)
Disease duration, years	2 (1-5)*
Makassar ethnicity	37 (50.7%)
Bugis ethnicity	30 (41.1%)
Toraja ethnicity	5 (6.8%)
Javanese ethnicity	1 (1.4%)
MTX without corticosteroids	44 (60.3%)
MTX + corticosteroids	29 (39.7%)
Hypertension	22 (30.1%)
Joint deformity	7 (9.6%)
RF positive (≥18 IU/mL)	46 (63.0%)
RF <10 IU/mL	26 (35.6%)
RF, IU/mL	49.8 (<10-54.4)*
CRP, mg/L	27.0 (16.0-44.1)*
DAS28-CRP	3.86 ± 1.17

Data are presented as n (%), mean ± standard deviation, or median (IQR)*. RF values <10 IU/mL were below the laboratory reporting limit.

Disease activity and rheumatoid factor levels

Mean DAS28-CRP was 3.86 ± 1.17. Moderate disease activity was the most frequent category (39; 53.4%), followed by low disease activity (13; 17.8%), remission (11; 15.1%), and high disease activity (10; 13.7%).

Median RF increased numerically from the remission group to the high-activity group, although the overall difference across the four categories did not reach statistical significance (Kruskal-Wallis p=0.069).

Table 2. Distribution of RF according to disease-activity category

DAS28-CRP category	n (%)	Median RF (IU/mL)	RF positive, n (%)
Remission	11 (15.1%)	<10	4 (36.4%)
Low activity	13 (17.8%)	45.2	7 (53.8%)
Moderate activity	39 (53.4%)	49.8	26 (66.7%)
High activity	10 (13.7%)	51.0	9 (90.0%)

Kruskal-Wallis test for differences in RF across disease-activity categories: p=0.069. The proportion of RF-positive participants increased numerically across higher disease-activity categories.

Association between RF and DAS28-CRP

RF level showed a weak positive correlation with DAS28-CRP (Spearman rho=0.258; p=0.028). In a secondary analysis, RF-positive participants had a higher mean

DAS28-CRP score than RF-negative participants (4.12 vs 3.40; Welch p=0.009). The relationship between RF and DAS28-CRP is shown in Figure 1.

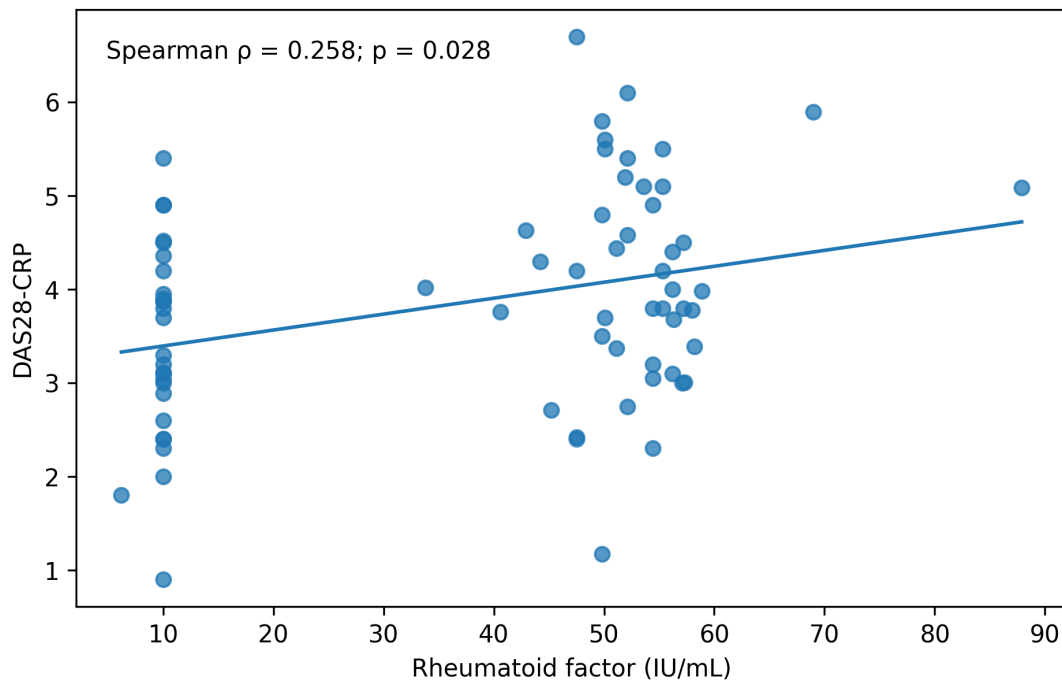


Figure 1. Relationship between rheumatoid factor level and DAS28-CRP. RF values reported as <10 IU/mL are displayed at the reporting limit of 10 IU/mL for visualization only. The line shows the linear regression fit; the primary correlation analysis used Spearman rank correlation ($\rho=0.258$; $p=0.028$).

Multivariable and sensitivity analyses

In the multivariable model adjusted for age, sex, and disease duration, each 10-IU/mL increase in RF was associated with a 0.126-point higher DAS28-CRP score (95% CI 0.016-0.236; $p=0.025$). This model explained approximately 23.0% of the variance in DAS28-CRP ($R^2=0.230$). When current corticosteroid use was added, the magnitude of the RF association decreased to 0.073 points per 10 IU/mL and was no longer statistically significant (95% CI -0.042 to 0.189; $p=0.214$). Current corticosteroid use itself was associated with a 0.994-point higher DAS28-CRP score (95% CI 0.519-1.470; $p<0.001$);

because of the cross-sectional design, this association is most plausibly interpreted in the context of confounding by indication rather than as evidence of a harmful corticosteroid effect.

The main-model result remained directionally consistent when RF values <10 IU/mL were substituted with 5 IU/mL: $\beta=0.112$ per 10 IU/mL (95% CI 0.011-0.212; $p=0.029$). This sensitivity analysis indicates that the observed RF-DAS28-CRP association in the main adjusted model was not wholly dependent on the selected substitution value, although simple substitution does not eliminate uncertainty caused by left-censoring.

Table 3. Association between RF level and DAS28-CRP in multivariable models

Model / variable	beta	95% CI	p	Interpretation
Model 1: RF per 10 IU/mL	0.126	0.016 to 0.236	0.025	Adjusted for age, sex, and disease duration; $R^2=0.230$
Model 2: RF per 10 IU/mL	0.073	-0.042 to 0.189	0.214	Model 1 + current corticosteroid use
Model 2: corticosteroid use	0.994	0.519 to 1.470	<0.001	Difference in DAS28-CRP for users vs non-users; non-causal interpretation
Sensitivity: RF per 10 IU/mL*	0.112	0.011 to 0.212	0.029	Model 1; RF <10 substituted with 5 IU/mL

*Sensitivity analysis addressing RF values below the laboratory reporting limit. The RF beta coefficient represents the change in DAS28-CRP per 10-IU/mL increase in RF.

Exploratory analysis of the acute-phase reactant

RF was not correlated with CRP as an acute-phase reactant (Spearman $\rho=-0.009$; $p=0.939$). This finding suggests that the association between RF and DAS28-CRP in this

dataset was not simply a reflection of variation in CRP. Because the individual DAS28-CRP components were not available separately in the final analytical dataset, the

study could not determine which component contributed most strongly to the observed association.

DISCUSSION

This study found a weak positive association between RF level and RA disease activity measured by DAS28-CRP. A Spearman correlation of 0.258 ($p=0.028$) indicates that higher RF levels tended to accompany higher disease activity, but the magnitude of the relationship was too small for RF to function as a stand-alone activity marker. In the adjusted model, each 10-IU/mL increase in RF was associated with a 0.126-point increase in DAS28-CRP after accounting for age, sex, and disease duration. The observed effect should therefore be considered clinically supplementary rather than dominant.

The characteristics of the study population are important for interpretation. Most participants were women (84.9%), the median disease duration was 2 years, and more than half had moderate disease activity. Remission, low, moderate, and high disease activity were observed in 15.1%, 17.8%, 53.4%, and 13.7% of participants, respectively. The remission proportion was lower and the moderate-activity proportion was higher than those reported in a nationwide Indonesian study, which reported remission in 24.5%, low activity in 18.5%, moderate activity in 44.6%, and high activity in 12.4% [5]. Direct comparison should nevertheless be made cautiously because patient characteristics, referral setting, disease duration, treatment exposure, and the disease-activity instrument can all affect category distributions.

Forty-six of 73 participants (63.0%) were RF positive at a threshold of ≥ 18 IU/mL. RF-positive participants had a higher mean DAS28-CRP score than RF-negative participants (4.12 vs 3.40; $p=0.009$). A numerical gradient was also observed across disease-activity categories: median RF increased from <10 IU/mL in remission to 45.2 IU/mL in low activity, 49.8 IU/mL in moderate activity, and 51.0 IU/mL in high activity. The proportion of RF-positive participants likewise increased across categories. Because the overall Kruskal-Wallis comparison did not reach statistical significance ($p=0.069$), these findings are best described as a consistent numerical trend rather than a definitive categorical difference.

The difference between the continuous and categorical analyses is statistically plausible. DAS28-CRP is intrinsically continuous; dividing it into remission, low, moderate, and high activity reduces numerical information and decreases the number of observations within each category. With only 73 participants, this loss of information is particularly relevant for the remission and high-activity groups, which contained 11 and 10 patients, respectively. This likely contributed to the significant continuous RF-DAS28-CRP correlation but non-significant four-group comparison.

The direction of the present findings is consistent with Aletaha and colleagues, who reported an association between RF and higher baseline disease activity in RA, whereas a similar relationship was not consistently

observed for antibodies against citrullinated proteins [6]. This supports the possibility that RF captures some biological characteristics associated with a more active disease phenotype. However, the low correlation in our study emphasizes that high RF does not necessarily imply high current activity in an individual patient. Patients with high RF may have low disease activity after treatment, whereas RF-negative patients may still have clinically important synovitis. RF should therefore not be used in isolation to judge treatment response.

A biological relationship between RF and RA activity is plausible. IgM RF recognizes the Fc portion of IgG and may participate in immune-complex formation, complement activation, and amplification of synovial inflammation. Nevertheless, RF is not an acute-phase reactant and its concentration does not necessarily change in parallel with day-to-day clinical inflammation. RF may therefore be better understood as a marker of an immunological phenotype associated with disease severity than as a rapidly responsive biomarker such as CRP.

Pongratz and colleagues reported that the relationship between autoantibody levels and RA disease activity depends on baseline inflammation and is not constant across clinical contexts [7]. This concept is relevant to the present findings. The RF-DAS28-CRP association persisted after adjustment for age, sex, and disease duration, but the main model explained only about 23% of DAS28-CRP variability. Most variability in disease activity therefore reflects other factors not represented in the model, including synovitis burden, adherence, DMARD dose and combination, comorbidities, psychosocial factors, non-inflammatory pain, and other biological variables.

An important exploratory finding was the absence of an RF-CRP correlation (Spearman $\rho=-0.009$; $p=0.939$). The association between RF and DAS28-CRP therefore did not appear to be driven simply by the acute-phase component. DAS28 is a composite index that integrates clinical and laboratory elements, so patients with similar CRP levels can have different DAS28 scores depending on tender-joint count, swollen-joint count, and patient global assessment [3]. This raises the possibility that the observed RF-DAS28-CRP relationship may relate more strongly to articular or patient-reported components than to the acute-phase response. That hypothesis cannot be tested in the present dataset because the individual DAS28 components were not retained separately.

The use of DAS28-CRP should also be considered when comparing these results with other studies. DAS28 can be calculated using ESR or CRP, and the two approaches do not always classify disease activity identically. Matsui and colleagues showed that DAS28-CRP may estimate lower activity and higher EULAR response rates than DAS28-ESR in a large observational RA cohort [8]. Differences between DAS28-CRP and DAS28-ESR may therefore account for part of the heterogeneity across national and international studies.

The RF association was attenuated after current corticosteroid use was added to the sensitivity model, with the coefficient decreasing to 0.073 points per 10 IU/mL and losing statistical significance. Conversely, corticosteroid use was associated with a higher DAS28-CRP score. This should not be interpreted as evidence that corticosteroids increase disease activity. In a cross-sectional clinical cohort, patients with more active disease are more likely to receive corticosteroids, creating confounding by indication. The direction of time between activity, treatment decisions, and measurement cannot be established; consequently, the corticosteroid-adjusted model is best regarded as a sensitivity analysis rather than a causal model.

Treatment context also limits generalizability. All participants had received MTX and 39.7% were also receiving corticosteroids. The study therefore describes treated RA outpatients rather than newly diagnosed, treatment-naïve patients. Therapy can reduce clinical activity and CRP more rapidly than it changes RF serostatus or titer, potentially weakening the contemporaneous RF-activity relationship. Requiring at least 6 months of DMARD exposure may also produce a disease-activity distribution that differs from inception or early-RA cohorts.

Clinically, these findings reinforce the principle that RF cannot replace composite disease-activity assessment. Treat-to-target management requires periodic evaluation and treatment adjustment based on the patient's overall clinical state [1,4]. A high RF level may provide supplementary information about serological phenotype but should not, by itself, trigger escalation of DMARD therapy when composite disease activity is low. Conversely, RF negativity does not exclude active disease when synovitis and high composite activity scores are present.

The left-censoring of RF values warrants specific methodological consideration. Twenty-six participants (35.6%) had RF reported as <10 IU/mL. Rank-based analysis reduces dependence on unknown absolute values by treating these observations as tied values at the lower end of the distribution. For adjusted numerical models, the study used pragmatic substitution at 10 IU/mL, with 5 IU/mL as a sensitivity analysis. The persistence of a similar direction and statistical significance in the main adjusted model under the alternative substitution supports robustness to this specific choice, but substitution cannot recover the true distribution below the detection limit. Future studies should use assays with lower quantitative reporting limits or statistical methods designed for censored biomarker data.

This study has several limitations. First, the cross-sectional design precludes temporal or causal inference. Second, the sample of 73 participants was smaller than the original proposal target, limiting power for small effects and subgroup comparisons. Third, all patients were treated with MTX and some were receiving corticosteroids, so the

results may not generalize to untreated RA. Fourth, detailed MTX dose, corticosteroid dose and duration, adherence, and recent treatment changes were not available, leaving the possibility of residual treatment-related confounding. Fifth, anti-CCP data were not available in the final analytical dataset, preventing direct comparison between RF and ACPA or assessment of double seropositivity. Sixth, individual DAS28-CRP components were unavailable for separate analysis. Seventh, the large proportion of RF measurements below the reporting limit reduced precision at low concentrations. Eighth, the linear-regression findings should be considered complementary to the rank-based primary analysis because the RF distribution was censored and non-normal. Finally, recruitment from a rheumatology outpatient service in Makassar limits generalizability to the broader Indonesian RA population, particularly settings with different ethnic composition, referral patterns, or treatment access.

The study also has strengths. RF was analyzed quantitatively rather than only dichotomized as positive or negative. Disease activity was assessed with DAS28-CRP, a clinically relevant composite measure. The analysis extended beyond simple correlation to an adjusted model incorporating age, sex, and disease duration, and sensitivity analyses examined both corticosteroid use and the handling of RF values below the reporting limit. The study also contributes local data from Makassar, an Indonesian setting that remains relatively underrepresented in published RA research and could provide a foundation for larger multicenter registries.

Future studies should use longitudinal designs with serial measurement of RF, CRP, individual DAS28 components, and treatment exposure to determine whether changes in RF track changes in activity or primarily reflect a longer-term serological phenotype. Adding anti-CCP, double-seropositivity status, joint imaging, and structural outcomes would help distinguish the role of RF as a marker of current activity from its prognostic role. Larger multicenter recruitment would also permit stratification by disease duration, treatment regimen, and ethnicity.

CONCLUSION

Higher RF levels were weakly associated with higher DAS28-CRP scores in patients with RA in Makassar. The association remained statistically significant after adjustment for age, sex, and disease duration, but it was attenuated and no longer significant when current corticosteroid use was added to the sensitivity model. RF should be considered an adjunctive serological biomarker rather than a replacement for composite assessment of disease activity. Longitudinal studies with larger samples, individual DAS28 components, anti-CCP measurements, and detailed treatment data are needed to clarify the independent and temporal relationship between autoantibody levels and RA activity.

DECLARATIONS

Ethics approval: The study protocol received ethical approval from the Research Ethics Committee of Universitas Hasanuddin, Hasanuddin University Hospital, and Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia (Protocol No. UH26060694; Approval No. 885/UN4.6.4.5.31/PP36/2026; 20 July 2026).

Consent to participate: Written informed consent was obtained from all participants in accordance with the study protocol.

Conflict of interest: The authors declare no conflicts of interest.

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Data availability: The study data are available from the corresponding author upon reasonable request and subject to the applicable ethics approval and institutional requirements.

Author contributions: Endy Adnan: Conceptualization, methodology, investigation, and writing - original draft and review & editing. Muhammad Rezky Juni: Investigation (data collection). Aditya Sandi: Investigation (data collection).

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