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Association between Platelet-Derived Growth Factor-BB (PDGF-BB) and Platelet-Lymphocyte Ratio (PLR) with the Severity of Acute Ischemic Stroke Patients

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KEYWORDS

Acute Ischemic Stroke; Platelet Derived Growth Factor-BB; PDGF-BB; Platelet-Lymphocyte Ratio; PLR; NIHSS, Angiogenesis

ABSTRACT:

Introduction: Acute ischemic stroke is a leading cause of mortality and disability worldwide. Platelet activation and inflammatory processes contribute significantly to the pathophysiology of ischemic stroke. Platelet-Derived Growth Factor-BB (PDGF-BB) plays a role in angiogenesis and neuroprotection, whereas Platelet-to-Lymphocyte Ratio (PLR) reflects thrombotic activity and inflammation. This study aimed to analyze the relationship between Platelet-Derived Growth Factor-BB (PDGF-BB) levels and Platelet-Lymphocyte Ratio (PLR) values with the severity of acute ischemic stroke.

Objectives: To determine the correlation between PDGF-BB levels and PLR values and the severity of acute ischemic stroke in patients.

Methods: This observational analytical study used a cross-sectional design. Patients with acute ischemic stroke (onset 1–7 days) aged 18–80 years were recruited from Dr. Wahidin Sudirohusodo General Hospital and affiliated hospitals in Makassar. Serum PDGF-BB levels were measured using ELISA, while PLR was calculated from absolute platelet and lymphocyte counts. Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS). Correlation analysis was performed using Spearman's test. Receiver Operating Characteristic (ROC) analysis was used to determine the diagnostic performance of PDGF-BB and PLR for stroke severity.

Results: Forty-one patients were included in the final analysis. PDGF-BB levels showed a weak negative correlation with stroke severity ($r = -0.311$, $p = 0.048$), indicating that higher PDGF-BB levels were associated with lower NIHSS scores. In contrast, PLR showed a moderate positive correlation with stroke severity ($r = 0.436$, $p = 0.004$). ROC analysis demonstrated that PLR had good discriminative ability for stroke severity (AUC = 0.821, sensitivity 75%, specificity 76.5%), whereas PDGF-BB showed poor discriminative ability (AUC = 0.586).

Conclusions: PDGF-BB levels were inversely correlated with stroke severity, while PLR was positively correlated with stroke severity. Compared with PDGF-BB, PLR demonstrated better performance in distinguishing stroke severity and may serve as a practical biomarker in clinical settings. Further large-scale studies are required to confirm these findings.

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1. Introduction

Stroke is a global health issue and a leading cause of death and disability. The Global Burden of Disease (GBD) 2019 report states that stroke is the second and third leading cause of death and disability based on disability-adjusted life-years (DALY). Ischemic stroke accounted for the most new stroke cases in 2019, followed by intracerebral hemorrhage and subarachnoid hemorrhage [1]. Most ischemic strokes are caused by atherosclerosis in large arteries or cardiac embolism. Approximately 45% of ischemic stroke cases are caused by thrombi forming in large or small arteries. Meanwhile, approximately 14–30% of cases result from cardiac embolism [1,2].

Atherosclerosis is a chronic inflammatory disease of the blood vessels triggered by various factors, including interactions between blood cells (e.g., platelets and monocytes) and blood plasma components (e.g., lipoproteins). Environmental factors (e.g., hypertension, hyperglycemia, and hyperlipidemia) can accelerate the process of atherosclerosis, increasing the risk of ischemic stroke. Platelets play a vital role in the process of atherosclerosis. They form plaque in injured blood vessels and release bioactive molecules that strengthen plaque and promote inflammation, thrombosis, and angiogenesis [2,3].

Platelets contain three types of granules: 1) electron-dense, 2) α-granules, and 3) lysosomes. Each contains active substances essential for their role in various pathophysiological processes. PDGF-BB is a growth factor molecule secreted by α-granules. During an ischemic stroke, platelets become activated, degranulate, and release PDGF-BB. To exert its effects, PDGF-BB must bind to its receptor, PDGFR-β, which is expressed by pericytes. The secretion of PDGF-BB plays a role in angiogenesis, neuroprotection, and maintaining cell survival following an acute ischemic stroke [4,5].

PLR (Platelet-Lymphocyte Ratio) is a ratio that indicates a link between inflammation and atherosclerosis development. PLR is a relatively easy-to-measure biomarker. Previous studies have demonstrated that elevated platelet concentrations and reduced lymphocyte counts can exacerbate atherosclerosis, increase the risk of restenosis, and affect the stability of atherosclerotic plaques [6]. Therefore, this study aims to investigate the relationship between serum PDGF-BB levels, PLR values, and stroke severity in acute ischemic stroke patients. This research may provide new insights into prognosis assessment and treatment strategies for stroke patients

2. Objectives

This study aims to evaluate the relationship between platelet-derived growth factor-BB (PDGF-BB) levels, platelet-to-lymphocyte ratio (PLR) values, and the severity of acute ischaemic stroke, as measured by the National Institutes of Health Stroke Scale (NIHSS) score.

3. Methods

Study Design and Settings

This was a cross-sectional study conducted at Dr. Wahidin Sudirohusodo Makassar General Hospital and affiliated hospitals within the hasanuddin University medical network in Makassar. Data collection was conducted between December 2025 and March 2026.

Participants

Patients diagnosed with acute ischemic stroke and admitted to participating hospitals were screened for eligibility. Ischemic stroke was diagnosed based on clinical neurological examination and confirmed through neuroimaging studies. Participants were recruited using a consecutive sampling method. This involved including all eligible patients who met the inclusion criteria. This was continued until the required sample size was achieved.

The inclusion criteria for this study: Acute ischemic stroke patients with an onset of 1-7 days, aged 18-80 years, and experiencing a first ischemic stroke. Patients who are willing to participate in the study must sign a consent form. Exclusion criteria for this study: Patients with chronic kidney disease, chronic heart failure, malignant disease, infectious disease, severe liver disease, and autoimmune disease.

Data Collection and Clinical Assessment

Stroke severity was assessed using the National Institutes of Health Stroke Scale (NIHSS) within the first 24 hours of hospital admission. For analytical purposes, stroke severity was categorized into two groups: mild stroke (NIHSS < 5) and moderate-to-severe stroke (NIHSS ≥ 5) [7,8]. Demographic characteristics and vascular risk factors, including hypertension, diabetes mellitus, dyslipidemia, and smoking history, were recorded from medical records and patient interviews.

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Laboratory Measurement

Venous blood samples were obtained from all participants at the time of hospital admission. Routine hematological parameters, including platelet and lymphocyte counts, were analyzed using an automated hematology analyzer in the hospital laboratory. The platelet-to-lymphocyte ratio (PLR) was calculated by dividing the absolute platelet count by the absolute lymphocyte count obtained from routine hematological analysis

Serum PDGF-BB levels were measured at the Hasanuddin University Medical Research Center (HUMRC) laboratory using an enzyme-linked immunosorbent assay (ELISA) kit (Invitrogen, Thermo Fisher Scientific, Vienna, Austria) according to the manufacturer's instructions.

Data and Statistics Analysis

Statistical analysis was performed using SPSS software (IBM Corp., Armonk, NY, USA). Continuous variables were presented as median with minimum–maximum values, while categorical variables were expressed as frequencies and percentages. The normality of data distribution was assessed using the Shapiro–Wilk test. Since the data were not normally distributed, correlations between PDGF-BB levels, PLR values, and NIHSS scores were analyzed using Spearman's rank correlation test.

A Receiver Operating Characteristic (ROC) curve analysis was performed to evaluate the ability of PDGF-BB and PLR to discriminate stroke severity, and the Youden's Index was used to determine optimal cutoff values. Diagnostic performance was assessed based on the area under the curve (AUC), sensitivity, and specificity. A p-value of less than 0.05 was considered statistically significant.

Ethical Approval

This study was approved by the institutional review board and conducted in accordance with the principles of the Declaration of Helsinki. Ethical clearance was obtained from the Health Research Ethics Committee, Faculty of Medicine, Hasanuddin University, under protocol number 265/UN4.6.4.5.31/PP36/2026.

4. Results

Characteristics of Research Subjects

Table 1 presents the baseline characteristics of the research subjects. A total of 41 patients with ischemic stroke were recruited. Of these, 22 patients (53.7%) were male, and 19 patients (46.3%) were female. The median age of the participants was 57.9 years. The largest age group was 46–60 years (22 patients, 53.7%), followed by 61–75 years (15 patients, 36.6%), 31–45 years (3 patients, 7.3%), and >75 years (1 patient, 2.4%). Based on stroke onset, the majority of patients presented on the first day after symptom onset (19 patients, 46.3%), followed by the second day (9 patients, 22.0%), the third, fourth, and fifth days (each 4 patients, 9.8%), and the sixth day (1 patient, 2.4%). Regarding vascular risk factors, dyslipidemia was the most common comorbidity, affecting 26 patients (63.4%), followed by hypertension (24 patients, 58.5%), smoking (12 patients, 29.3%), and diabetes mellitus (6 patients, 14.6%).

The median PDGF-BB level among the study participants was 511.2 pg/mL (range 106.6–1216.06), while the median platelet-to-lymphocyte ratio (PLR) was 181.6 (range 68.69–2852.9). Stroke severity, assessed using NIHSS, showed a median score of 5 (range 1–23). Based on severity classification, 17 patients (41.5%) had mild stroke, whereas 24 patients (58.5%) had moderate-to-severe stroke.

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Variable	Total (n=41) n(%) Median (min-max)
Gender	
• Male	22 (53.7%)
• Female	19 (46.3%)
Onset	
• 1	19 (46.3%)
• 2	9 (22.0%)
• 3	4 (9.8%)
• 4	4 (9.8%)
• 5	4 (9.8%)
• 6	1 (2.4%)
Age	56 (34-80)
• 31-45 years	3 (7.3%)
• 46-60 years	22 (53.7%)
• 61-75 years	15 (36.6%)
• > 75 years	1 (2.4%)
Risk Factor	
• Hypertension	24 (58.5%)
• Dyslipidemia	26 (63.4%)
• Diabetes mellitus	6 (14.6%)
• Smoking	12 (29.3%)
Platelet Derived Growth Factor – BB (PDGF-BB)	511.2 (106.6-1216.06)
Platelet-Lymphocyte Ratio (PLR)	181.6 (68.69-2852.9)
NIHSS Score	5 (1-23)
• Mild	17 (41.5%)
• Moderate-Severe	24 (58.5%)

Table1. Baseline characteristics of research subjects

Correlation Between Platelet-Derived Growth Factor-BB (PDGF-BB) Levels and Stroke Severity

The relationship between PDGF-BB levels and stroke severity was analyzed using Spearman's correlation test, revealing a weak negative correlation between PDGF-BB levels and NIHSS scores ($r = -0.311$, $p = 0.048$). This indicates an inverse relationship between PDGF-BB levels and NIHSS score, suggesting that higher PDGF-BB levels are associated with lower NIHSS scores. The relationship is depicted within the scatter plot (Figure 1), which illustrates a downward trend, where increasing PDGF-BB levels correspond to decreasing NIHSS scores.

Figure 1. Correlation Plot of the Platelet-Derived Growth Factor-BB (PDGF-BB) with the Severity of Acute Ischemic Stroke

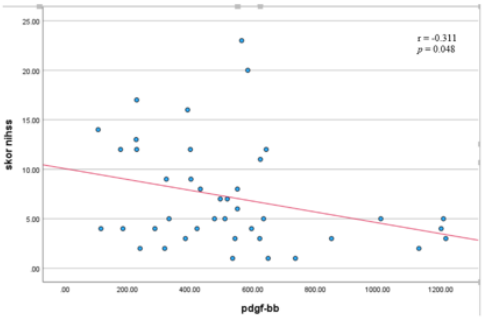

Table 2. Correlation Between Platelet-Derived Growth Factor-BB (PDGF-BB) Levels and the Severity of Acute Ischemic Stroke

Variabel	NIHSS		
	r	95% CI	*p*
***Platelet-Derived Growth Factor – BB* (PDGF-BB)**	-0.311	-0.571 – 0.05	0.048

Correlation Between Platelet-Lymphocyte Ratio (PLR) and Stroke Severity

Table 3 shows that the Spearman correlation analysis demonstrated a moderate positive correlation between PLR and NIHSS scores ($r = 0.436$, $p = 0.004$). This indicates that higher PLR is associated with greater stroke severity. The scatter plot (Figure 2) demonstrates an upward trend, showing that NIHSS scores tend to increase as PLR values increase.

Figure 2. Correlation Plot of the Platelet-Lymphocyte Ratio (PLR) with the Severity of Acute Ischemic Stroke

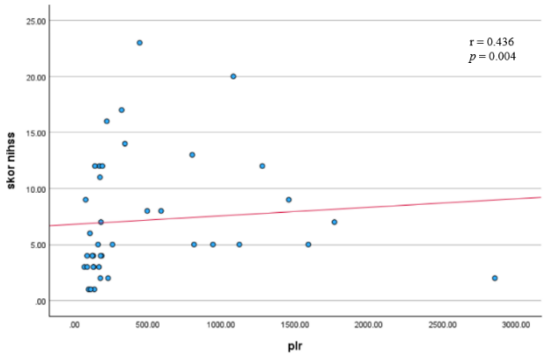

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Table 3. Correlation Between Platelet-Lymphocyte Ratio (PLR) and the Severity of Acute Ischemic Stroke

Variable	NIHSS		
	r	95% CI	*p*
***Platelet-Lymphocyte Ratio* (PLR)**	0.436	0.139 – 0.661	0.004

Receiver Operating Characteristic (ROC) Curve Analysis

A ROC curve analysis was performed to assess the ability of PDGF-BB levels and PLR values to distinguish the severity of acute ischemic stroke based on the NIHSS score. For PDGF-BB, the ROC analysis showed an area under the curve (AUC) of 0.586 (Figure 3), indicating poor discriminative ability for identifying moderate-to-severe stroke. The optimal cutoff value for PDGF-BB was <359 pg/mL, with a sensitivity of 70.6% and a specificity of 29.2% (Table 4).

Figure 3. ROC Curve for PDGF-BB Levels and Acute Ischemic Stroke Severity

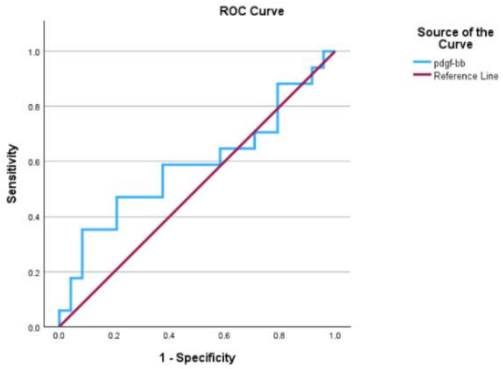

Table 4. Sensitivity and Specificity of PDGF-BB Levels for Acute Ischemic Stroke Severity

Variable			Severity Level (NIHSS Score) Mild (<5)	Moderate Severe (≥5)	*p*	Sensitivity	Specificity	AUC
PDGF-BB	≤ 356	n	6	6	0,146	70.6	29.2	0.586
		%	50.0%	50.0%				
	> 356	n	22	7				
		%	74.9%	24.1%				
Total		n	28	13				
		%	68.3%	31,7%				

In contrast, PLR demonstrated better diagnostic performance (Figure 4). The ROC analysis showed an AUC of 0.821, indicating good discriminative ability for differentiating stroke severity. The optimal PLR cutoff value was 178.3, which yielded a sensitivity of 75.0% and a specificity of 76.5% (Table 5).

Figure 4. ROC Curve for PLR Values and Acute Ischemic Stroke Severity

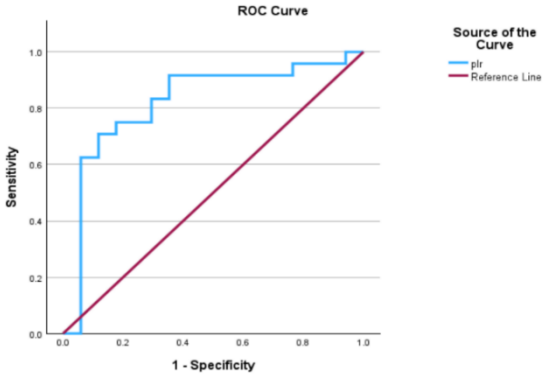

Table 5. Sensitivity and Specificity of PLR Values for Acute Ischemic Stroke Severity

Variable			Severity Level (NIHSS Score) Mild (<5)	Moderate -Severe (≥5)	*P*	Sensitivity	Specificity	AUC
PLR	≤ 178.3	n	13	6	0,002	75.0	76.5	0.821
		%	68.4%	31.6%				
	> 178.3	n	4	18				
		%	18.2%	81.8%				
	Total	n	17	24				
		%	41.5%	58.5%				

5. Discussion

This was an observational study designed to examine the relationship between PDGF-BB levels and PLR values and the severity of acute ischemic stroke in patients. The baseline characteristics of the study participants showed that the majority were male and in the middle-aged to older adult range. This aligns with previous studies indicating higher stroke incidence in men than in women and increases with age [9]. These findings suggest that age and sex play a role in the incidence of ischemic stroke, which is important for the epidemiology of acute ischemic stroke. The increased incidence among women has been extensively studied, particularly among postmenopausal women, who have declining estrogen

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levels. Estrogen exerts neuroprotective effects and promotes vasodilation [10].

Among the vascular risk factors identified in this study, dyslipidemia and hypertension were the most prevalent comorbidities. Both are major risk factors for ischemic stroke due to atherosclerosis, vascular stiffness, and impaired autoregulation. Akbar et al. (2018) show that hypertension and dyslipidemia exacerbate endothelial damage and impair cerebral perfusion in older adults [9]. Wang et al. (2022) found that combined hypertension and dyslipidemia significantly increase the risk of stroke. [11].

The median PDGF-BB level in this study was 511.2 (106.6–1216.06) pg/mL. A previous study showed PDGF-BB levels in patients with acute ischemic stroke of 250.1 (171.9–588.1) pg/mL, higher than in control patients at 93 (49.1–203) pg/mL. Another study showed PDGF-BB levels in patients with acute ischemic stroke to be 7.0 (2.40–2.22) ng/mL, twice as high as in healthy individuals [12,13]. These findings indicate greater secretion and activity of PDGF-BB following ischemic stroke. Statistical analysis revealed a weak negative correlation between PDGF-BB levels and the severity of acute ischemic stroke, as measured by the NIHSS score. This implies that higher PDGF-BB levels are associated with milder stroke severity, reflected by a lower NIHSS score. The PDGF-BB level cutoff value in this study demonstrated very weak predictive ability and very low specificity.

Research shows that PDGF-BB has a protective role during ischemic stroke. PDGF-BB is involved in angiogenesis, promoting cell proliferation, cell-to-cell interactions, and the migration of vascular smooth muscle cells (VSMCs). Additionally, PDGF-BB plays a neuroprotective role by stimulating nerve growth factor (NGF) and neurotrophin-3 [4,5]. This process protects brain cells from ischemic stroke-induced damage. Narsimhalu et al. (2014) demonstrated that high baseline PDGF-BB levels are not associated with the risk of death or disability following ischemic stroke and attributed the findings to the role of PDGF-BB in the angiogenesis process [5,14]. Iihara et al. (1994) showed that PDGF-BB expression occurs within 24 hours after stroke [15]. However, several processes must occur before PDGF-BB can exert its effects. Platelet activation and degranulation release PDGF-BB, which then binds to the PDGFR-β receptor. The signaling initiated by PDGF-BB binding to its receptor promotes angiogenesis and neuroprotection [4,5,15].

The median PLR value in this study was 181.6 (68.7–2853). Consistent with Luo H et al. (2019), the PLR values in the subjects of this study were higher than those in adults [16]. The statistical test showed a moderate positive correlation between PLR values and the severity of acute ischemic stroke. This indicates that higher values indicate more severe acute ischemic stroke, as reflected by high NIHSS scores. The PLR cutoff value in this study shows that PLR is better at distinguishing the severity of acute ischemic stroke. Previous studies have shown that higher PLR values after stroke are associated with poorer outcomes. Felix Adrian et al. (2022) reported higher values in the acute ischemic stroke group (188.46) than in the control group (108.29). Study subjects with a PLR value > 148 were significantly linked to poor outcomes. Bagaskara et al. (2025) found higher PLR values in patients with an NIHSS score > 4 [17,18].

PLR reflects platelet function in thrombosis and neuroinflammation, while lymphocytes reflect adaptive immunity following a stroke. Platelet activation and function occur immediately after ischemia. Activated platelets interact with leukocytes and endothelial cells at the site of vascular occlusion. This triggers an inflammatory cascade that can exacerbate ischaemic injury and contribute to the severity of the stroke (19,20). Lymphocytes, particularly T cells, will infiltrate the peri-infarct area within 3 days after an ischemic stroke and release any pro-inflammatory cytokine. Elevated PLR values in the acute phase may reflect lymphocyte depletion. Secondary lymphoid organs atrophy following a cerebral ischemic event, followed by lymphocyte apoptosis within 6–12 hours after stroke (2). This leads to a decrease in lymphocytes. A decrease in lymphocyte count can exacerbate neuronal injury and neurological deficits. Regulatory T cells (Tregs), a subtype of lymphocytes, play a protective role in the brain by producing anti-inflammatory cytokines during the acute phase of stroke. Decreased Treg counts are known to significantly worsen neurological deficits (19,20).

The results of this study revealed an inverse relationship between PDGF-BB and PLR and acute ischemic stroke severity. The ability to distinguish ischemic stroke severity was greater in PLR. In addition, in clinical practice, PLR is easier and simpler to perform and does not involve high costs. PLR value can be recommended

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as a single biomarker for determining the severity of acute ischaemic stroke in patients.

The strengths of this study are as follows: First, it investigates the relationship between two biomarkers with different mechanisms of action and the severity of acute ischaemic stroke. Second, stroke severity was assessed using the NIHSS, a sensitive, user-friendly, and validated tool. Third, this study uses clinical data from Indonesia, where similar studies are still limited.

Several limitations should be considered. First, the relatively small sample size may limit the generalizability of the results. Second, the cross-sectional design of the study prevents the evaluation of dynamic changes in biomarker levels during stroke recovery. Third, blood samples were collected within 1–7 days of stroke onset, and inflammatory responses may vary across different stages of the acute phase. Finally, potential confounding factors, such as comorbidities, medication use, and other inflammatory markers, were not fully controlled for in the analysis.

Future studies involving larger populations and prospective longitudinal designs are needed to better understand the role of PDGF-BB and PLR in stroke pathophysiology and prognosis. Additional research exploring the combined use of multiple biomarkers may also improve the prediction of stroke severity and clinical outcomes.

6. Conclusion

The results of this study suggest that PDGF-BB levels and the PLR ratio are involved in various mechanisms following a stroke. High PDGF-BB levels are associated with milder severity, whereas high PLR values are associated with greater severity. This study proves that PLR is more effective at discriminating between the severity of acute ischaemic stroke.

Author Contributions: Conceptualization, ND, AKB, and AB, GVS, MA, HB; Methodology, AB, AKB and GVS; Software, ND.; Validation, ND, AKB, AB, GVS, MA, and HB; Formal analysis, ND, AKB, AB, GVS, MA and HB; Investigation, ND, AKB, AB and GVS; Resources, ND; Data Curation, ND and AB; Writing—Original Draft Preparation, ND, AKB, AB and GVS; Writing—Review and Editing, ND, AKB, AB, GVS, MA and HB; Visualization, ND, AKB, AB, GVS, MA and HB; Supervision, AKB, AB, GVS, MA and HB; Project Administration, ND; Funding Acquisition, ND, AKB, AB and GVS. All authors have read and agreed to the published version of the manuscript.

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Data Availability Statement: The data supporting the findings of this study are available from the corresponding author upon reasonable request.

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Conflicts of Interest: The authors declare no conflict of interest.

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