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Original Article

Changes in N-Terminal Pro-B-Type Natriuretic Peptide Levels According to Estimated Plasma Volume Variation in Patients with Acute Decompensated Heart Failure

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Abstract

Background: Acute Decompensated Heart Failure (ADHF), often caused by fluid retention and elevated left ventricular filling pressure, is the most common form of acute heart failure, accounting for 50-70% of cases. It carries high morbidity and mortality, with in-hospital death up to 11% and one-year post-discharge mortality reaching 36%. Rehospitalization rates range from 22-30% within 1-3 months to 65% within one year. In Indonesia, the 2013 prevalence of physician-diagnosed heart failure was 0.13%, affecting approximately 230,000 people.

Methods: This prospective observational study was conducted on ADHF patients admitted to the Cardiology Department of Dr. M. Djamil General Hospital, Padang, during July-August 2025. Blood and N-Terminal pro-B-type Natriuretic Peptide (NT-proBNP) assessments were obtained at admission and upon discharge. Participants were divided into two categories based on their estimated Plasma Volume Variation (ePVV) results (<0% and ≥0%). The Mann-Whitney test was used to compare NT-proBNP variations between these groups.

Results: A total of 33 participants were included, the majority being male, with pneumonia as the most prevalent comorbidity and ischemic heart disease as the primary underlying cause. Patients with ePVV <0% showed a median change in NT-proBNP of 21.06%, whereas those with ePVV ≥0% showed a median change of 9.58%. The Mann-Whitney test indicated that this difference between groups was statistically significant ($p = 0.012$).

Conclusions: The observed variation in NT-proBNP levels across ePVV categories suggests that ePVV may be a valuable non-invasive marker for assessing congestion status in ADHF. Combining ePVV measurements with NT-proBNP monitoring could enhance clinical assessment and management strategies for patients with heart failure.

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Introduction

Acute Decompensated Heart Failure (ADHF) is the most common presentation of acute heart failure, accounting for roughly 50–70% of cases. It involves a rapid or gradual worsening of heart failure symptoms and clinical status, often necessitating urgent medical intervention or hospitalization.[1] According to the Acute Heart Failure Global Registry of Standard Treatment (ALARM-HF), in-hospital mortality among patients hospitalized for acute heart failure is approximately 11%, with post-discharge mortality remaining substantial, reaching around 13% at three months and nearly 30% at one year, underscoring the persistent vulnerability of this population after hospital discharge.[2] The median duration of hospitalization differs by region, averaging about 5 days in the United States, 6-11 days in European countries, and extending up to 21 days in Japan.[3] Hospital readmission following discharge continues to pose a significant global challenge, with rates ranging from 22-30% within the first 1-3 months and reaching up to 65% within a year of the initial hospitalization. In Indonesia, available data are scarce; however, the 2013 National Health Survey reported a prevalence of physician-diagnosed heart failure of 0.13%, corresponding to approximately 229,696 affected individuals.[4-5]

A key pathophysiological hallmark of ADHF is congestion, which results from excessive fluid accumulation and elevated left ventricular filling pressure. This condition serves as a critical predictor of adverse outcomes in heart failure. Failure to detect or adequately manage congestion increases the likelihood of rehospitalization and death. Hence, precise evaluation and continuous monitoring of congestion are vital for optimizing treatment. Current international guidelines emphasize the importance of assessing volume status to inform diuretic therapy. Nevertheless, evaluating congestion remains challenging, particularly when extrapulmonary manifestations are subtle or when residual fluid overload persists at the time of discharge.[6] Plasma Volume (PV) represents the intravascular fluid component and serves as an indicator of volume overload, playing a crucial role in maintaining fluid balance between the intravascular and interstitial spaces. While radioisotope-based techniques are considered the gold standard for PV measurement, they are expensive, invasive, and unsuitable for routine clinical use because they require repeated venous sampling and lengthy laboratory processing.[7] Various approaches have been developed to assess intravascular volume status, including clinical examination, ultrasonography, and biomarker use. One promising parameter, the estimated Plasma Volume Status (ePVS), is calculated using hemoglobin and hematocrit levels. This indicator has been correlated with congestion, cardiorenal syndrome, and unfavorable clinical outcomes. A reduction in ePVS generally signifies effective decongestion and better prognosis. Nevertheless, most available evidence originates from retrospective studies and primarily involves patients with chronic heart failure rather than those with ADHF.[6]

Estimated Plasma Volume Variation (ePVV) provides a non-invasive means of evaluating PV changes and acts as an indirect indicator of congestion. It is derived from sequential hemoglobin and hematocrit measurements, offering a practical and less invasive alternative to direct PV assessment. This approach is based on the principle that changes in hemoglobin concentration are inversely related to fluctuations in total intravascular volume.[8] N-Terminal pro-B-type Natriuretic Peptide (NT-proBNP) is a well-established biomarker used to diagnose and stratify the risk of heart failure. Released in response to ventricular wall stretch, it reflects intravascular volume and cardiac filling pressure, serving as both a marker of congestion and a predictor of poor outcomes. A meta-analysis of 12 clinical studies by Savarese et al. (2014) demonstrated that NT-proBNP-guided therapy was associated with reduced all-cause mortality and heart failure–related hospitalizations. However, it did not significantly affect overall readmission rates.[9]

Few studies have specifically explored the relationship between ePVV and NT-proBNP. Grigore et al. reported that patients with ePVV values below 0% experienced a significant decline in NT-proBNP following intravenous diuretic therapy, with ePVV being the only independent predictor of NT-proBNP reduction at discharge.[10-12] Similarly, Kobayashi et al. demonstrated that ePVS at discharge independently predicted post-discharge outcomes in hospitalized ADHF patients.[13] However, the direct association between dynamic intravascular volume changes (ePVV) and short-term NT-proBNP response during hospitalization remains insufficiently characterized, particularly in real-world clinical settings.

Therefore, the present study aimed to evaluate the relationship between ePVV and changes in NT-proBNP levels during hospitalization in patients with ADHF, and to assess whether ePVV could serve as a practical non-invasive marker of decongestion response.

Methods

Study Design

This prospective observational study was carried out at the Integrated Cardiac Care Unit of Dr. M. Djamil General Hospital, Padang, and the Biomedical Laboratory of the Faculty of Medicine, Andalas University. Independent and dependent variables were measured at two time points, namely at hospital admission and before discharge, to evaluate changes during hospitalization. Data were collected between July and August 2025, following ethical approval from the Health Research Ethics Committee of Dr. M. Djamil General Hospital, Padang (Approval No. DP.04.03/D.XVI.10.1/289/2025).

Study Population and Sample

The study population included all patients diagnosed with ADHF who were admitted to the Integrated Cardiac Care Unit of Dr. M. Djamil General Hospital, Padang. Patients who met the predefined inclusion and exclusion criteria were recruited consecutively until the target sample size was reached. Using a two-proportion formula, the minimum required sample size was determined to be 33 participants, based on a 95% confidence level ($Z\alpha/2 = 1.96$), 70% study power ($Z\beta = 0.52$), and mortality proportions from previous studies ($p_1 = 0.378$; $p_2 = 0.118$).

Inclusion Criteria

Patients diagnosed with ADHF who were admitted to Dr. M. Djamil General Hospital, Padang.

Exclusion Criteria

Patients were excluded if they presented with any of the following conditions: severe anemia, received a blood transfusion during hospitalization, had hyponatremia requiring correction, septic shock, stage V chronic kidney disease, or were undergoing hemodialysis, acute coronary syndrome as the primary reason for admission, atrial fibrillation, or significant valvular stenosis.

Data Collection

Patients who met the inclusion criteria were enrolled at the time of admission. A total of 6 mL of venous blood was collected, 3 mL in an EDTA tube for hematology testing and 3 mL in a serum tube for NT-proBNP analysis. Hematology samples were processed immediately in the hospital laboratory. For NT-proBNP analysis, samples were centrifuged at 4000 rpm for 15 minutes, and the plasma was separated, labeled, and stored at −80°C until analysis at the Biomedical Laboratory, Faculty of Medicine, Universitas Andalas.

A follow-up venous blood sample was collected 24 hours before discharge to assess dynamic changes in hematological parameters and NT-proBNP levels during hospitalization. Hemoglobin and hematocrit values obtained at admission and discharge were used to calculate ePVV with the Strauss formula, and patients were categorized into two groups (ePVV $< 0\%$ and ePVV $\geq 0\%$). NT-proBNP levels were then compared between admission and discharge to determine the percentage change.

Statistical Analysis

Univariate analysis was performed to describe patient characteristics. Categorical variables were expressed as frequencies and percentages, while continuous variables were presented as mean ± standard deviation or median (minimum–maximum), depending on data distribution. Patients were categorized into two groups based on ePVV calculated using the Strauss formula: a negative ePVV group (ePVV $< 0\%$), indicating hemoconcentration and intravascular volume reduction, and a non-negative ePVV group (ePVV $\geq 0\%$), indicating the absence of effective volume contraction.

Changes in NT-proBNP levels and ePVV were calculated by comparing values obtained at hospital admission and 24 hours before discharge, reflecting longitudinal changes during hospitalization.

The Shapiro–Wilk test was applied to assess normality for samples with $n < 50$. Differences in NT-proBNP percentage changes between the two ePVV groups were analyzed using the independent t-test for normally distributed data and the Mann–Whitney U test for non-normally distributed data. A p-value < 0.05 was considered statistically significant. All statistical analyses were conducted using SPSS for Windows.

Results

A total of 42 patients diagnosed with ADHF met the inclusion criteria for this prospective observational study. Following the application of exclusion criteria, including hemodialysis (n = 1), cardiogenic shock (n = 1), septic shock (n = 2), valvular heart disease (n = 3), and in-hospital mortality (n = 2), a total of 33 patients remained eligible for longitudinal analysis. All of these participants were included in the subsequent statistical evaluation.

Baseline Characteristics

Baseline characteristics of the study population are summarized in Table 1. Of the 33 participants, 25 (75.8%) were male. Patients were divided into two groups based on ePVV: Group A (ePVV <0%, n = 18) and Group B (ePVV ≥0%, n = 15). No

significant differences were observed between the groups regarding demographic factors such as age, sex, Body Mass Index (BMI), or admission vital signs, including systolic and diastolic blood pressure, heart rate, and respiratory rate. Comorbidities, including hypertension, diabetes mellitus, pneumonia, chronic obstructive pulmonary disease, and chronic kidney disease, were similarly distributed between groups. However, the length of hospital stay was significantly longer in Group B (6.0 ± 2.0 days) than in Group A (4.72 ± 1.40 days; p = 0.039). Echocardiographic assessment revealed a significant difference in Tricuspid Annular Plane Systolic Excursion (TAPSE), with Group B showing lower TAPSE values (15.53 ± 2.77 mm) than Group A (20.33 ± 5.32 mm, p = 0.003). Other echocardiographic parameters, including Ejection Fraction (EF), Stroke Volume (SV), Cardiac Output (CO), and Systemic Vascular Resistance (SVR), did not differ significantly between the groups.

Table 1. Baseline characteristics of the study population.

Variable	Group A (ePVV <0%, n=18)	Group B (ePVV ≥0%, n=15)	p-value
Age, median (range), years	61.5 (32-69)	57 (40-75)	0.630[1]
Male sex, n (%)	14 (77.8)	11 (73.3)	0.767[2]
BMI, mean ± SD (kg/m^2)	23.08 ± 3.36	24.11 ± 3.53	0.397[1]
Systolic BP, mean ± SD (mmHg)	122.61 ± 17.82	122.00 ± 20.80	0.928[1]
Diastolic BP, mean ± SD (mmHg)	74.67 ± 17.05	74.40 ± 14.42	0.962[1]
HR, median (range), bpm	78 (62–112)	83 (70–107)	0.556[3]
RR, mean ± SD (breaths/min)	24.61 ± 3.09	23.60 ± 1.99	0.284[1]
Hypertension, n (%)	8 (44.4)	8 (53.3)	0.611[2]
Diabetes mellitus, n (%)	6 (33.3)	8 (53.3)	0.247[2]
Pneumonia, n (%)	16 (88.9)	12 (86.7)	1.000[2]
COPD, n (%)	3 (16.7)	3 (20.0)	1.000[2]
CKD, n (%)	1 (5.6)	2 (13.3)	0.579[2]
HF etiology (Ischemic), n (%)	13 (72.2)	14 (93.3)	0.186[2]
Length of stay, mean ± SD (days)	4.72 ± 1.40	6.0 ± 2.0	0.039[1]
EF, mean ± SD (%)	32.22 ± 10.34	30.53 ± 8.88	0.622[1]
TAPSE, mean ± SD (mm)	20.33 ± 5.32	15.53 ± 2.77	0.003[1]
Stroke volume, mean ± SD (mL)	41.11 ± 12.08	39.60 ± 10.12	0.703[1]
Cardiac output, mean ± SD (L/min)	3.36 ± 1.14	3.73 ± 1.03	0.343[1]
SVR, median (range), dyne·sec/cm^5	1439 (1022–2400)	1478 (1021–3275)	0.957[3]

BMI: Body Mass Index; BP: Blood Pressure; CKD: Chronic Kidney Disease; COPD: Chronic Obstructive Pulmonary Disease; EF: Ejection Fraction; ePVV: estimated Plasma Volume Variation ; HF: Heart Failure; HR: Heart Rate; RR: Respiratory Rate; SD: Standard Deviation; SVR: Systemic Vascular Resistance; TAPSE: Tricuspid Annular Plane Systolic Excursion.

Table 2. Hemoglobin and hematocrit values at admission and pre-discharge.

Parameter	Group A (ePVV <0%)	Group B (ePVV ≥0%)	p-value
Hemoglobin, g/dL			
Admission	12.33 ± 2.27	13.07 ± 1.49	0.292[1]
Discharge	13.67 ± 2.48	11.82 ± 1.32	0.394[1]
Hematocrit, %			
Admission	37.61 ± 7.48	39.53 ± 4.64	0.014[1]
Discharge	42.89 ± 7.58	36.20 ± 3.65	0.003[1]

ePVV: estimated Plasma Volume Variation.

Laboratory Parameters

Hematology findings are presented in Table 2. Significant differences in hematocrit levels were observed between the groups. At admission, Group B had a higher hematocrit (39.53 ± 4.64%) than Group A (37.61 ± 7.48%; p = 0.014). By discharge, this pattern was reversed, with Group A showing higher hematocrit levels (42.89 ± 7.58%) than Group B (36.20 ± 3.65%, p = 0.003), indicating greater hemoconcentration in Group A during hospitalization. Hemoglobin levels, however, did not differ significantly between the groups at either time point.

Congestion Assessment

Clinical and ultrasonographic congestion parameters are summarized in Table 3. At admission, all patients in both groups were classified as congested based on EVEREST scores (≥2). By discharge, decongestion (score <2) was observed in 44.4% of patients in Group A and 40% in Group B, with no significant difference between groups (p = 0.797). Sonographic evaluation using SAFE scores also revealed no significant differences in hypervolemia status at admission or discharge. Furthermore, other parameters, including the E/e' ratio, estimated Right Atrial Pressure (eRAP), B-lines, and Internal Jugular Vein (IJV) respiratory variation, did not differ significantly between the groups.

NT-proBNP Levels and Changes

Comparison of NT-proBNP levels and their percentage changes between groups is presented in Table 4. At admission, Group A demonstrated significantly higher baseline NT-proBNP levels (median 14,350.5 pg/mL) compared to Group B (median 10,438 pg/mL, p = 0.015), indicating a greater initial congestion burden. By discharge, NT-proBNP levels were similar between the two groups (p = 0.486).

Despite higher baseline NT-proBNP levels, Group A exhibited a significantly greater percentage reduction in NT-proBNP during hospitalization (median 21.06%) compared to Group B (median 9.58%, p = 0.012). This finding suggests that patients who achieved negative ePVV experienced a more pronounced biomarker response to decongestive therapy.

Table 3. Congestion Assessment Based on Clinical and Ultrasound Scores at Admission.

Parameter	Group A (ePVV <0%)	Group B (ePVV ≥0%)	p-value
EVEREST admission, n (%)			-
Decongested (<2)	0 (0%)	0 (0%)	-
Congested (≥2)	18 (100%)	15 (100%)	-
EVEREST discharge, n (%)			0.797[1]
Decongested (<2)	8 (44.4%)	6 (40%)	
Congested (≥2)	10 (55.6%)	9 (60%)	
SAFE admission, n (%)			0.455[2]
Non-hypervolemia (-4 to +1)	1 (5.6%)	0 (0%)	
Hypervolemia (+2 to +4)	17 (94.4%)	15 (100%)	
SAFE discharge, n (%)			0.152[2]
Non-hypervolemia (-4 to +1)	17 (94.4%)	11 (73.3%)	
Hypervolemia (+2 to +4)	1 (5.6%)	4 (26.7%)	

Table 4. NT-proBNP values and percentage change during hospitalization based on ePVV groups.

Parameter	Group A (ePVV <0%)	Group B (ePVV ≥0%)	p-value
NT-proBNP, pg/mL			
Admission, median (range)	14,350.5 (8,716–22,855)	10,438 (7,093–18,761)	0.015[1]
Discharge, median (range)	9,969.5 (6,495–18,640)	9,713 (6,133–16,918)	0.486[1]
Percentage change, median (range), %	21.06 (3.67–58.85)	9.58 (0.5–52.68)	0.012[1]

ePVV: estimated Plasma Volume Variation; NT-proBNP: N-Terminal pro-B-type Natriuretic Peptide.

Discussion

This study underscores variations in patient characteristics and therapeutic responses in ADHF according to ePVV.[13] The predominance of male patients in both groups is consistent with global trends, which report a higher incidence of heart failure among men. This disparity is likely attributable to a greater burden of cardiovascular risk factors in men, particularly ischemic heart disease, which was the leading etiology in our cohort.[14] These observations align with findings from the REPORT-HF registry and other epidemiological studies, confirming that ischemic heart disease is more common in men and contributes to their increased susceptibility to acute decompensation.[15]

The mean age of patients in this study indicates that most cases occur in individuals over 50 years old, which aligns with global cohort analyses and data from tertiary care centers. This finding emphasizes that ADHF predominantly affects middle-aged and older populations, where age-related cardiac structural changes and comorbidities increase vulnerability to decompensation.[16-17] In addition to chronic comorbid conditions such as hypertension, pneumonia in this cohort was more appropriately identified as a precipitating factor rather than a comorbidity. Pneumonia, the most common non-cardiac trigger observed in this study, can worsen hemodynamic status by increasing systemic inflammation, metabolic demand, and hypoxemia, and has been associated with higher in-hospital mortality and prolonged length of stay. Hypertension, although the second most prevalent chronic condition in this cohort, should be interpreted primarily as a major risk factor for the development and progression of heart failure rather than a direct cause of acute decompensation. However, episodes of uncontrolled blood pressure or hypertensive crisis may act as precipitating factors for ADHF by acutely increasing left ventricular afterload and myocardial oxygen demand. These observations underscore the need for comprehensive therapeutic approaches addressing both chronic cardiovascular risk factors and acute non-cardiac or hemodynamic triggers.[18]

Echocardiographic findings, particularly TAPSE differences, indicate more severe right ventricular dysfunction in patients with ePVV ≥ 0%. This association may reflect the impact of persistent hemodynamic congestion and volume overload on right ventricular performance, without implying a direct pulmonary-specific mechanism. Although other hemodynamic parameters such as SV, CO, and SVR showed no significant differences, assessing right ventricular function provides additional prognostic information and may guide therapy. The longer hospital stay observed in patients with ePVV ≥0% may reflect a more complex clinical course, including slower decongestion response, persistent volume overload, and the need for prolonged diuretic titration or monitoring, rather than a direct effect of ePVV itself. This observation supports the notion that patients achieving hemoconcentration tend to demonstrate more efficient decongestive response, whereas patients with persistent hemodilution may require longer hospitalization to reach clinical stability.[19-21]

Hemoglobin and hematocrit analyses reflect differences in intravascular volume response. In the present study, patients in the ePVV <0% group demonstrated a significant increase in hematocrit from admission to discharge. In contrast, the ePVV ≥0% group showed lower discharge hematocrit values and minimal dynamic change. The increase in hematocrit observed in the ePVV <0% group indicates effective decongestion and beneficial hemoconcentration, consistent with findings from the ESCAPE trial. In contrast, minimal or declining hematocrit in the ePVV ≥0% group suggests a suboptimal diuretic response. This highlights the importance of dynamic volume monitoring, especially in patients with potential confounding factors such as severe hyperglycemia.[5,22]

Residual congestion, assessed using EVEREST scores, E/e' ratio, B-lines, and SAFE scores, demonstrated that many patients still experienced congestion at discharge. This pattern aligns with findings from the DOSE-AHF and CARESS-HF trials, indicating that residual congestion persists even when clinical signs appear improved.[23-26] SAFE scoring, which is sensitive to real-time hemodynamic changes, confirmed that some patients appeared clinically non-hypervolemic yet continued to have elevated filling pressures. Meanwhile, ePVV <0% was correlated with hemoconcentration and predicted a greater decrease in NT-proBNP. These findings underscore the need to combine clinical, imaging, and biomarker assessments of accurately evaluate volume status.[8,27]

Importantly, the higher baseline NT-proBNP levels in the ePVV <0% group may reflect greater initial volume overload and neurohormonal activation, which could partly explain the larger absolute and percentage reductions during hospitalization. This baseline difference represents a potential confounding factor that should be

considered when interpreting treatment response.[28]

However, despite presenting with more severe congestion at admission, patients in the ePVV <0% group achieved effective hemoconcentration, suggesting a favorable decongestive response to intravenous diuretic therapy. In contrast, patients in the ePVV ≥0% group, although starting with lower NT-proBNP levels, demonstrated limited volume contraction and less pronounced biomarker improvement. These findings indicate that ePVV may reflect treatment responsiveness and intravascular volume dynamics rather than merely baseline disease severity.[29-30]

A greater reduction in NT-proBNP in the ePVV <0% group indicates that hemoconcentration correlates with a better response to intravenous diuretic therapy in the present cohort. Conversely, patients with ePVV ≥0% and persistent hemodilution demonstrated a smaller reduction in NT-proBNP and a longer duration of hospitalization, reflecting a slower in-hospital decongestion response rather than post-discharge outcome. Dual NT-proBNP measurements both at admission and pre-discharge may be useful for guiding discharge readiness and short-term treatment optimization in hospitalized patients with acute decompensated heart failure.

Overall, these findings highlight the utility of ePVV as a practical marker of intravascular volume status and treatment response in patients with acute decompensated heart failure. Combining clinical evaluation, hemodynamic assessment, ultrasonography, and biomarkers such as NT-proBNP provides a more comprehensive approach to patient management, allowing early identification of high-risk individuals and optimization of decongestive therapy.

Limitations of the Study

This study has several limitations. Although this study employed a prospective observational design, the relatively small sample size and single-center setting may limit the generalizability of the findings to broader heart failure populations. Variability in baseline clinical severity and comorbid conditions among participants may have influenced changes in ePVV and NT-proBNP during hospitalization. It may not have been fully controlled in this observational setting. In addition, baseline differences in NT-proBNP levels between study groups may have introduced residual confounding that could not be fully adjusted due to the observational design and limited sample size.

Additionally, osmolarity was not assessed in this study. Osmolarity can affect ePVV calculations, particularly in patients with diabetes mellitus, as fluctuations in glucose and sodium levels can alter fluid distribution.

Conclusion

This study demonstrates that patients with ePVV ≥0% tend to have longer hospital stays and lower TAPSE values compared to those with ePVV <0%. Moreover, the ePVV <0% group exhibited greater dynamic changes in hematocrit, with significantly lower values at admission and higher values at discharge than the ePVV ≥0% group. Although there were no significant differences in congestion parameters by EVEREST or SAFE scores at admission or discharge, the ePVV <0% group showed a greater reduction in NT-proBNP, indicating a better therapeutic response.

List of Abbreviations

ADHF	Acute Decompensated Heart Failure
ALARM-HF	Acute Heart Failure Global Registry of Standard Treatment
BMI	Body Mass Index
CO	Cardiac Output
COPD	Chronic Obstructive Pulmonary Disease
eRAP	estimated Right Atrial Pressure
PV	Plasma Volume
EF	Ejection Fraction
ePVS	estimated Plasma Volume Status
ePVV	estimated Plasma Volume Variation
HF	Heart Failure
HR	Heart Rate
IJV	Internal Jugular Vein
NT-proBNP	N-Terminal pro-B-type Natriuretic Peptide
SV	Stroke Volume
SVR	Systemic Vascular Resistance
TAPSE	Tricuspid Annular Plane Systolic Excursion

Ethical Clearance

Ethical approval was obtained from the Health Research Ethics Committee of Dr. M. Djamil General Hospital, Padang (Approval No. DP.04.03/D.XVI.10.1/289/2025).

Publication Approval

All authors consent to the publication of this manuscript.

Authors' Contributions

All authors have made significant intellectual contributions to this manuscript according to the criteria, and each has participated sufficiently in the work to take public responsibility for its content. WP and YRI contributed to the conception and design of the study; WP, YRI, CKK, and HA contributed to the analysis and interpretation of data; WP drafted the manuscript; WP, YRI, CKK, and HA were involved in critically revising the manuscript for important intellectual content; and all authors (WP, YRI, CKK, and HA) gave final approval of the version to be published. All authors agreed to be accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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Conflict of Interest

The authors declares no conflict of interest.

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