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Atherogenic Lipid Indices and In-Hospital Mortality in Patients with Heart Failure and LVEF <50%: A Retrospective Cohort Study

Sukuria Usman[1,2*], Suryani As'ad[1,2], Nurpudji Astuti Daud[1,2], Agussalim Bukhari[1,2], Aminuddin[1], A. Yasmin Syauki[1,2]

[1]Department of Clinical Nutrition, Faculty of Medicine, Hasanuddin University, Makassar, South Sulawesi, Indonesia

[2]Department of Clinical Nutrition, Dr. Wahidin Sudirohusodo General Hospital, Makassar, South Sulawesi, Indonesia.

ABSTRACT: Atherogenic lipid indices may provide complementary prognostic information in heart failure (HF), but their association with short-term mortality remains uncertain. We evaluated the associations of the atherogenic index of plasma (AIP) and total cholesterol-to-high-density lipoprotein cholesterol ratio (TC/HDL-C) with in-hospital mortality among adults hospitalized with HF and left ventricular ejection fraction <50%. This single-center retrospective cohort included 105 patients admitted between January 2023 and December 2025; 24 patients (22.9%) died during hospitalization. AIP was calculated as log10 of the molar triglyceride-to-HDL-C ratio, and TC/HDL-C was calculated from routine lipid measurements. Patients who died had higher AIP than survivors (0.448 ± 0.373 vs 0.270 ± 0.315; P=0.022) and lower HDL-C (21.5 [14.5–34.8] vs 28.0 [21.5–39.5] mg/dL; P=0.039). Each 0.1-unit increase in AIP was associated with higher odds of in-hospital mortality in crude analysis (OR 1.168, 95% CI 1.018–1.340; P=0.026), after multivariable adjustment (aOR 1.165, 95% CI 1.012–1.341; P=0.034), and in Firth penalized sensitivity analysis (aOR 1.151, 95% CI 1.003–1.320; P=0.046). TC/HDL-C was not significantly associated with mortality. Higher AIP was associated with in-hospital mortality and warrants validation in larger prospective multicenter cohorts.

1. INTRODUCTION

Heart failure (HF) remains a major global health burden, affecting more than 64 million people worldwide and contributing substantially to hospitalization, mortality, and healthcare expenditure [1]. Current guidelines classify HF according to left ventricular ejection fraction (LVEF), including HF with reduced ejection fraction (HFrEF; LVEF ≤40%) and HF with mildly reduced ejection fraction (HFmrEF; LVEF 41–49%) [2,3]. Hospitalization represents a particularly vulnerable phase, with clinically important short-term mortality despite advances in guideline-directed therapy. Contemporary Indonesian multicenter data similarly demonstrate substantial in-hospital risk among patients admitted with acute HF [4].

The prognostic interpretation of lipid parameters in established HF is complex. A recent meta-analysis of 52 studies involving 93,286 patients found inverse associations between several conventional lipid parameters and mortality, consistent with the so-called lipid paradox in HF; however, these observational findings do not imply a protective effect of higher lipid concentrations [5]. Composite lipid indices may therefore provide complementary prognostic information beyond isolated lipid measurements.

The atherogenic index of plasma (AIP), calculated as log10 of the molar triglyceride-to-high-density lipoprotein cholesterol ratio, has been associated with cardiovascular events in population-based studies and adverse outcomes in coronary artery

disease [6,7]. More directly, Yu et al. [8] reported an association between higher AIP and 30-day mortality in 1,248 patients with acute decompensated HF, although the relationship was nonlinear.

The total cholesterol-to-high-density lipoprotein cholesterol (TC/HDL-C) ratio has also demonstrated nonlinear associations with mortality in the general population [9], while population-based findings for AIP remain heterogeneous [10]. However, evidence directly evaluating both lipid indices in hospitalized patients with HF remains limited, particularly among patients with LVEF <50%. Therefore, this study examined the associations of AIP and TC/HDL-C with in-hospital mortality among hospitalized patients with HF and LVEF <50%.

2. MATERIALS AND METHODS

2.1 Study Design, Setting, and Participants

This retrospective cohort study was conducted at Dr. Wahidin Sudirohusodo General Hospital, Makassar, Indonesia. Medical records of patients hospitalized with heart failure (HF) between January 2023 and December 2025 were reviewed. Data access and extraction were performed from May to June 2026 after ethical approval had been obtained.

Adults aged ≥18 years who were hospitalized with HF and had a left ventricular ejection fraction (LVEF) <50% were eligible for inclusion. Patients were required to have an available lipid profile during hospitalization, with triglycerides, total cholesterol, and high-density lipoprotein cholesterol (HDL-C) measurements obtained within the same laboratory testing episode used to calculate the lipid-derived indices.

Patients were excluded if they had incomplete clinical or laboratory data required for the study, severe infection or sepsis, advanced chronic kidney disease requiring hemodialysis, chronic liver disease, autoimmune disease, terminal malignancy or ongoing chemotherapy, or documented use of lipid-lowering medication before hospitalization. All eligible records during the study period were included using total sampling. Of 650 records assessed, 545 were excluded and 105 patients comprised the final analytic cohort.

2.2 Data Collection and Clinical Variables

Demographic, clinical, echocardiographic, and laboratory data were extracted from the medical records. Age, sex, body weight, height, LVEF, comorbidities, lipid parameters, and vital status at hospital discharge were recorded. Body mass index (BMI) was calculated as body weight in kilograms divided by height in meters squared.

LVEF was analyzed as a continuous variable and was also used descriptively to classify patients as having HF with reduced ejection fraction (HFrEF; LVEF ≤40%) or HF with mildly reduced ejection fraction (HFmrEF; LVEF 41–49%). Comorbidity burden was represented by the total number of four documented comorbidity domains—other cardiovascular disease, hypertension, diabetes mellitus, and renal disease—yielding a count ranging from 0 to 4. Individual comorbidities were additionally reported descriptively.

The primary outcome was all-cause in-hospital mortality, defined as death from any cause during the index hospitalization. Patients discharged alive were classified as survivors.

2.3 Lipid Measurements and Atherogenic Indices

Total cholesterol, HDL-C, low-density lipoprotein cholesterol (LDL-C), and triglyceride concentrations were obtained from routine hospital laboratory records and expressed in mg/dL. The atherogenic index of plasma (AIP) and total cholesterol-to-HDL cholesterol ratio (TC/HDL-C) were independently recalculated from the original lipid values for analysis.

For calculation of AIP, triglycerides were converted to mmol/L by dividing triglyceride concentrations in mg/dL by 88.57, whereas HDL-C was converted to mmol/L by dividing HDL-C concentrations in mg/dL by 38.67. AIP was then calculated as log10 [triglycerides (mmol/L)/HDL-C (mmol/L)]. The TC/HDL-C ratio was calculated by dividing total cholesterol by HDL-C; because both measurements were expressed in the same units, no additional unit conversion was required.

The primary analyses treated both indices as continuous exposures. AIP was modeled per 0.1-unit increase and TC/HDL-C per 1-unit increase. For descriptive and supportive analyses, AIP was categorized as low (<0.11), intermediate (0.11–0.21), or high (>0.21), whereas TC/HDL-C was categorized as <5 or ≥5. These categories were not used to derive data-dependent prognostic cutoffs.

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2.4 Statistical Analysis

Continuous variables were assessed for distribution using histograms, Q–Q plots, skewness, and the Shapiro–Wilk test. Approximately normally distributed variables were summarized as mean ± standard deviation and compared between survivors and patients who died using the independent-samples *t* test. Non-normally distributed variables were summarized as median with interquartile range and compared using the Mann–Whitney U test. Categorical variables were presented as frequencies and percentages and compared using Pearson's chi-square test.

Associations of AIP and TC/HDL-C with in-hospital mortality were examined using binary logistic regression. Each lipid-derived index was evaluated in a separate model to avoid unnecessary overlap between indices sharing HDL-C as a component. Crude odds ratios (ORs) with 95% confidence intervals (CIs) were first estimated. Prespecified multivariable models were then adjusted for age, sex, BMI, total comorbidity count, and LVEF. Age was modeled per 10-year increase, BMI per 5-kg/m² increase, LVEF per 5-percentage-point increase, and total comorbidity count per additional documented comorbidity. AIP was modeled per 0.1-unit increase, whereas TC/HDL-C was modeled per 1-unit increase. Covariates were entered simultaneously without automated variable-selection procedures.

Model calibration was assessed using the Hosmer–Lemeshow goodness-of-fit test. Multicollinearity among predictors was evaluated using tolerance and variance inflation factor statistics. No receiver operating characteristic-derived cutoffs, stepwise variable selection, or data-driven threshold optimization was performed.

Because only 24 in-hospital deaths occurred and the multivariable models contained six regression terms, Firth bias-reduced logistic regression was additionally performed as a sensitivity analysis using the same covariate set. Firth estimates were obtained using the Jeffreys-prior adjusted-score approach, with Wald-type 95% CIs and two-sided *P* values. Conventional analyses were performed using IBM SPSS Statistics version 30 (IBM Corp., Armonk, NY, USA), and Firth sensitivity analyses were implemented in R version 4.4.1 through the SPSS R Integration environment. No missing-value imputation was performed. All statistical tests were two-sided, and $P<0.05$ was considered statistically significant.

3. RESULTS AND DISCUSSION

3.1 Results

A total of 650 medical records were assessed for eligibility. After exclusion of 545 records, 105 patients with HF and LVEF <50% were included in the final analysis. Of these, 81 (77.1%) survived to hospital discharge and 24 (22.9%) died during hospitalization (Figure 1).

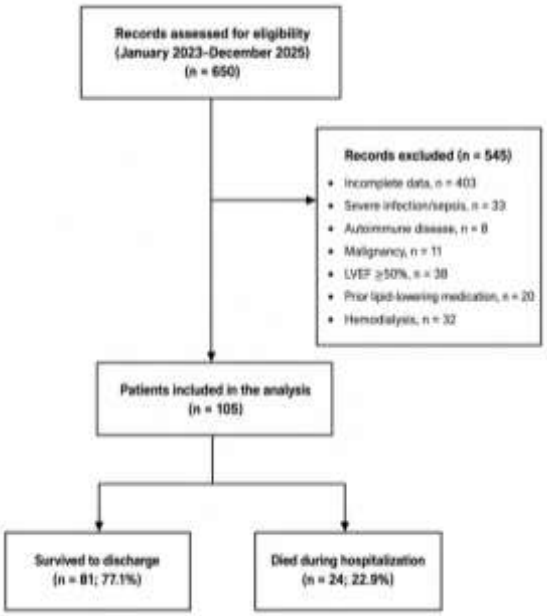

Figure 1. Study flow diagram

The mean age of the study population was 58.73 ± 13.42 years, 57.1% were male, and 76.2% had HFrEF. No baseline demographic or clinical characteristic differed significantly between survivors and patients who died (all *P*>0.05) (Table 1).

Table 1. Baseline Characteristics According to In-Hospital Mortality

Characteristic	Overall (n=105)	Survived (n=81)	Died (n=24)	*P* value
Age, years	58.73 ± 13.42	58.75 ± 13.77	58.67 ± 12.45	0.978[a]
Male sex, n (%)	60 (57.1)	45 (55.6)	15 (62.5)	0.546[b]
BMI, kg/m²	22.83 (20.25–26.17)	23.31 (20.38–27.37)	21.67 (18.46–24.85)	0.143[c]
LVEF, %	33.17 ± 9.21	32.75 ± 9.07	34.59 ± 9.72	0.392[a]
HF phenotype, n (%)				0.212[b]
HFrEF	80 (76.2)	64 (79.0)	16 (66.7)	
HFmrEF	25 (23.8)	17 (21.0)	8 (33.3)	
Total comorbidities	2 (1–2.5)	2 (1–3)	2 (0–2)	0.221[c]
Other cardiovascular disease, n (%)	71 (67.6)	58 (71.6)	13 (54.2)	0.109[b]
Hypertension, n (%)	44 (41.9)	35 (43.2)	9 (37.5)	0.619[b]
Diabetes mellitus, n (%)	38 (36.2)	33 (40.7)	5 (20.8)	0.075[b]
Renal disease, n (%)	34 (32.4)	25 (30.9)	9 (37.5)	0.542[b]

Note: Data are presented as mean ± SD, median (IQR), or n (%). [a]Independent-samples t test; [b]Pearson chi-square test; [c]Mann–Whitney U test. BMI, body mass index; LVEF, left ventricular ejection fraction; HFrEF, heart failure with reduced ejection fraction; HFmrEF, heart failure with mildly reduced ejection fraction.

Patients who died had lower HDL-C concentrations than survivors (21.50 [14.50–34.75] vs 28.00 [21.50–39.50] mg/dL; *P*=0.039) and higher AIP (0.448 ± 0.373 vs 0.270 ± 0.315; *P*=0.022). Total cholesterol, LDL-C, triglycerides, and TC/HDL-C did not differ significantly between the groups. The predefined AIP and TC/HDL-C categories were also not significantly associated with in-hospital mortality (Table 2).

Table 2. Lipid Profile and Atherogenic Indices According to In-Hospital Mortality

Lipid parameter	Overall (n=105)	Survived (n=81)	Died (n=24)	P value
Total cholesterol, mg/dL	181.64 ± 62.54	182.88 ± 60.94	177.46 ± 68.87	0.711[a]
HDL-C, mg/dL	27.00 (20.00–37.50)	28.00 (21.50–39.50)	21.50 (14.50–34.75)	0.039*[b]
LDL-C, mg/dL	109.00 (70.00–160.00)	109.00 (75.00–161.00)	106.00 (54.75–157.75)	0.321[b]
Triglycerides, mg/dL	117.00 (89.50–159.50)	114.00 (85.00–157.50)	140.50 (107.25–168.50)	0.102[b]
AIP	0.311 ± 0.336	0.270 ± 0.315	0.448 ± 0.373	0.022*[a]
TC/HDL-C ratio	6.32 (4.75–8.44)	6.25 (4.56–8.15)	7.26 (5.53–9.38)	0.149[b]
AIP category, n (%)				0.086[c]
Low (<0.11)	32 (30.5)	28 (34.6)	4 (16.7)	
Intermediate (0.11–0.21)	15 (14.3)	13 (16.0)	2 (8.3)	
High (>0.21)	58 (55.2)	40 (49.4)	18 (75.0)	

TC/HDL-C category, n (%)				0.207[c]
<5	28 (26.7)	24 (29.6)	4 (16.7)	
≥5	77 (73.3)	57 (70.4)	20 (83.3)	

Note: Data are presented as mean ± SD, median (IQR), or n (%). [a]Independent-samples t test; [b]Mann–Whitney U test; [c]Pearson chi-square test. AIP, atherogenic index of plasma; HDL-C, high-density lipoprotein cholesterol; LDL-C, low-density lipoprotein cholesterol; TC/HDL-C, total cholesterol-to-HDL cholesterol ratio. *Significant at p<0.05.

In crude logistic regression, each 0.1-unit increase in AIP was associated with higher odds of in-hospital mortality (OR 1.168, 95% CI 1.018–1.340; *P*=0.026). The association remained significant after adjustment for age, sex, BMI, total comorbidity count, and LVEF (aOR 1.165, 95% CI 1.012–1.341; *P*=0.034). The Firth penalized sensitivity analysis yielded a similar result (aOR 1.151, 95% CI 1.003–1.320; *P*=0.046). TC/HDL-C was not significantly associated with in-hospital mortality in the crude, adjusted, or Firth models (all *P*>0.05) (Table 3).

Table 3. Associations of Atherogenic Lipid Indices with In-Hospital Mortality

Exposure	Crude OR (95% CI)	*P* value	Adjusted OR (95% CI)	*P* value	Firth-adjusted OR (95% CI)	*P* value
AIP, per 0.1-unit increase	1.168 (1.018–1.340)	0.026*[a]	1.165 (1.012–1.341)	0.034*[a]	1.151 (1.003–1.320)	0.046*[b]
TC/HDL-C, per 1-unit increase	1.108 (0.986–1.246)	0.085[a]	1.112 (0.989–1.250)	0.076[a]	1.098 (0.980–1.231)	0.108[b]

Note: [a]Binary logistic regression; [b]Firth penalized logistic regression. Adjusted models included age, sex, BMI, total comorbidity count, and LVEF. AIP, atherogenic index of plasma; CI, confidence interval; OR, odds ratio; TC/HDL-C, total cholesterol-to-HDL cholesterol ratio. *Significant at p<0.05.

3.2 Discussion

In this retrospective cohort of hospitalized patients with HF and LVEF <50%, higher atherogenic index of plasma (AIP) was associated with greater odds of in-hospital mortality. The association remained statistically significant after adjustment for age, sex, BMI, total comorbidity count, and LVEF and was retained in the Firth bias-reduced sensitivity analysis. In contrast, TC/HDL-C was not significantly associated with mortality in any model. Patients who died also had lower HDL-C concentrations, whereas total cholesterol, LDL-C, and triglyceride concentrations did not differ significantly between groups. Among the lipid-derived indices evaluated, AIP therefore showed the most consistent association with in-hospital mortality in this cohort, a pattern directionally consistent with previous evidence in acute decompensated HF [8].

The closest comparable study is that of Yu et al. [8], which included 1,248 patients with acute decompensated HF and found that higher AIP was associated with 30-day mortality after multivariable adjustment. Their restricted cubic spline analysis demonstrated a nonlinear relationship, with the lowest estimated risk around an AIP of −0.1. Because the present study assessed in-hospital mortality and was restricted to patients with LVEF <50%, our findings should not be interpreted as validation of the same threshold. Broader cardiovascular evidence nevertheless supports the relevance of AIP, with higher values associated with cardiovascular events in population-based cohorts and major adverse cardiovascular outcomes among patients with coronary artery disease [6,7].

Mortality associations with AIP are not uniform across populations. Studies in general populations have reported U- or J-shaped relationships and, in some analyses, no significant adjusted association with cardiovascular mortality [10,11]. Persistently elevated AIP trajectories have also been associated with incident HF among adults with hypertension, although this finding concerns development of HF rather than prognosis after hospitalization [12]. These differences emphasize that the prognostic meaning of AIP may depend on population characteristics, disease stage, and clinical context.

AIP combines triglyceride and HDL-C information and may capture aspects of atherogenic dyslipidemia that are not represented by either measurement alone. It has been associated with cardiometabolic risk factors and with smaller, denser

LDL particles [13]. Triglyceride-rich lipoproteins and their cholesterol-enriched remnants also have established roles in atherogenic lipid metabolism, providing biological context for the triglyceride component of AIP [14]. In HF, however, lipid measurements are influenced by processes beyond atherosclerosis. Reduced cardiac output, congestion, inflammation, metabolic stress, and alterations in hepatic, renal, and gastrointestinal function may affect lipoprotein homeostasis [15]. Consistent with this complexity, Yu et al. [8] reported that inflammatory and nutritional pathways partially mediated the association between AIP and 30-day mortality. AIP should therefore be interpreted as a composite marker reflecting several interrelated metabolic and systemic processes rather than as a direct causal determinant of mortality.

The lower HDL-C concentration observed among patients who died is consistent with evidence that HDL biology in HF extends beyond circulating HDL-C concentration. In BIOSTAT-CHF, higher HDL cholesterol efflux capacity was associated with lower mortality independently of HDL-C concentration [16]. Teis et al. [17] further reported associations of HDL particle size and cholesterol content with cardiovascular death in chronic HF, whereas Klobučar et al. [18] found that lower HDL-apoA-II and its subfractions were associated with increased 1-year mortality in acute HF. These studies indicate that HDL composition and function may provide prognostic information not captured by conventional HDL-C measurement alone [15]. Accordingly, the lower HDL-C observed among non-survivors in the present study should not be interpreted as evidence that therapeutic elevation of HDL-C would improve clinical outcomes.

In contrast to AIP, TC/HDL-C was not significantly associated with in-hospital mortality. In the general population, TC/HDL-C has shown nonlinear associations with mortality, but direct evidence for this ratio in hospitalized HF remains limited [9]. Interpretation is further complicated by the unusual relationship between conventional lipid concentrations and prognosis in established HF. A meta-analysis of 52 studies involving 93,286 patients reported inverse observational associations between several lipid parameters and mortality, consistent with the phenomenon commonly described as the lipid paradox [5]. These observational associations should not be interpreted as evidence that higher lipid concentrations are inherently protective.

Nutritional status provides an important clinical context for this paradox. A meta-analysis found that malnutrition was associated with substantially higher all-cause mortality in HF, and a large retrospective cohort similarly demonstrated poorer survival among malnourished patients [19,20]. Low cholesterol and HDL-C concentrations may therefore reflect advanced disease, inflammation, cachexia, or malnutrition rather than directly contribute to worse outcomes. These factors may partly explain why a ratio based on total cholesterol and HDL-C did not demonstrate a consistent prognostic association in the present cohort.

The predefined AIP categories were not significantly associated with mortality despite the significant association observed when AIP was analyzed as a continuous variable. With only 24 in-hospital deaths, categorization produced relatively small subgroups and reduced the information retained from the continuous exposure. This loss of information may have reduced statistical power to detect differences across categories.

Recent studies evaluating AIP have increasingly modeled the index continuously and, when sample size permits, used restricted cubic splines to assess potential nonlinear associations [8,10,11]. Given the limited number of events in the present cohort, retaining AIP as a continuous exposure was preferable to deriving a new sample-specific prognostic cutoff. The present findings therefore support further evaluation of AIP as a continuous marker rather than establishing categorical risk thresholds for clinical use.

This study evaluated two routinely calculable lipid-derived indices in a clinically relevant hospitalized HF population and used prespecified multivariable adjustment. The consistency of the AIP association across conventional logistic regression and Firth bias-reduced regression provides additional support for the robustness of the observed direction of association in a dataset with a limited number of mortality events.

Several limitations should nevertheless be considered. The retrospective, single-center design limits external validity, and requiring an available lipid profile may have introduced selection bias. Patients receiving lipid-lowering therapy before hospitalization were excluded, further limiting generalizability. Only 24 in-hospital deaths occurred, restricting the complexity and precision of the multivariable models. Although Firth regression reduced small-sample estimation bias, it could not eliminate residual confounding.

Lipid sampling was not standardized according to fasting status or exact timing relative to decompensation and treatment. Important determinants of HF prognosis, including natriuretic peptides, detailed renal function, serum sodium, albumin,

inflammatory markers, congestion severity, ischemic etiology, nutritional status, and comprehensive HF therapy, were not uniformly available [2,3,5]. These unmeasured factors may influence both lipid parameters and mortality and should be considered when interpreting the observed associations.

Within these limitations, higher AIP remained associated with in-hospital mortality in both conventional and Firth-adjusted analyses, whereas TC/HDL-C did not. Because AIP can be calculated easily from routinely available triglyceride and HDL-C measurements, it may warrant further evaluation as an adjunctive prognostic marker rather than a stand-alone risk tool. Larger multicenter studies with standardized lipid sampling, greater event numbers, assessment of nonlinear associations, and more complete adjustment for HF severity, nutritional status, inflammation, and treatment are required before its clinical prognostic utility can be established.

4. CONCLUSION

Higher AIP was significantly associated with greater odds of in-hospital mortality among patients hospitalized with heart failure and LVEF <50%, and this association remained significant after multivariable adjustment and Firth penalized sensitivity analysis. In contrast, TC/HDL-C was not significantly associated with in-hospital mortality. These findings suggest that AIP may capture clinically relevant lipid-related risk information in this population, although its role should be regarded as associative rather than predictive. Larger prospective multicenter studies with standardized lipid measurements and more comprehensive adjustment for heart failure severity, nutritional status, inflammation, and treatment are needed to confirm these findings and establish their clinical relevance.

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AUTHOR CONTRIBUTIONS

SU: Conceptualization, Methodology, Investigation, Data Curation, Formal Analysis, and Writing – Original Draft.

SA: Conceptualization, Methodology, Supervision, Validation, and Writing – Review & Editing.

NAD: Conceptualization, Methodology, Supervision, Validation, and Writing – Review & Editing.

AB: Methodology, Supervision, Validation, and Writing – Review & Editing.

AM: Methodology, Formal Analysis, Validation, and Writing – Review & Editing.

AYS: Methodology, Supervision, Validation, and Writing – Review & Editing.

All authors critically reviewed the manuscript, approved the final version, and agreed to be accountable for the integrity of the work.

CONFLICT OF INTEREST

The authors declare that there is no conflict of interest.

FUNDING

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ETHICS STATEMENT

The study protocol was approved by the Health Research Ethics Committee of Hasanuddin University (approval No. 557/UN4.6.4.5.31/PP36/2026; protocol No. UH26040470) on 23 April 2026. Patient information was handled in de-identified form to maintain confidentiality.

DATA AVAILABILITY STATEMENT

The data supporting the findings of this study are available from the corresponding author upon reasonable request, subject to applicable ethical and institutional restrictions.

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