International Journal of Scientific Advances
ISSN: 2708-7972
Volume: 6 | Issue: 6 | Nov – Dec 2025 Available Online: www.ijscia.com
DOI: 10.51542/ijscia.v6i6.41

De Ritis Ratio as Major Adverse Cardiovascular Events Predictor Up to 30 Days After Hospital Discharge in Acute Myocardial Infarction Patients Undergoing Percutaneous Coronary Intervention

Primanto Tantiono*, I Kadek Susila Surya Darma, Hendy Wirawan

1Department of Cardiology and Vascular Medicine, Faculty of Medicine, Udayana University, Prof. Dr. I.G.N.G. Ngoerah Central General Hospital, Denpasar, Bali, Indonesia, 80114

E-mail: primanto.tantiono1@student.unud.ac.id; kadeksusila138@gmail.com; hendy.md76@gmail.com

*Corresponding author: Primanto Tantiono; primanto.tantiono1@student.unud.ac.id

ABSTRACT

Background: Aspartate aminotransferase (AST) and alanine transaminase (ALT) are commonly used to assess liver function, but they also increase in acute myocardial infarction (AMI) as a result of myocardial injury and hemodynamic compromise. ALT is relatively specific for hepatocellular injury, whereas AST is also present in cardiac tissue and rises during cellular necrosis. Therefore, the AST/ALT (De Ritis) ratio has been proposed as a potential marker for end-organ ischemic injury. This study aimed to evaluate the prognostic value of the De Ritis ratio in AMI patients undergoing percutaneous coronary intervention (PCI). ***Methods:*** A retrospective cohort study was conducted on patients with STEMI and NSTEMI undergoing PCI. Blood samples were taken upon emergency department admission to measure AST and ALT, and the De Ritis ratio was calculated. Major adverse cardiovascular events (MACE) were assessed during hospitalization and up to 30 days after discharge. Receiver operating characteristic analysis determined the optimal cutoff, while Kaplan–Meier and Cox regression analyses were used for outcome evaluation. ***Results:*** Among 278 patients, in-hospital MACE occurred significantly more often in those with a De Ritis ratio ≥2.03 ($p = 0.014$). A high De Ritis ratio was an independent predictor of in-hospital MACE (HR = 1.842, $p = 0.001$), along with pre-procedural TIMI flow, hs-troponin, and NLR. However, the De Ritis ratio was not an independent predictor of 30-day MACE (HR = 2.208, $p = 0.557$). ***Conclusion:*** The De Ritis ratio independently predicts in-hospital MACE in AMI patients undergoing PCI.

Keywords: acute myocardial infarction; De Ritis ratio; major adverse cardiovascular events

INTRODUCTION

Cardiovascular disease (CVD) is the leading cause of mortality and morbidity worldwide [1]. In 2021, it was estimated that 19.41 million deaths globally were caused by CVD [2]. According to the Global Burden of Disease (GBD) data, in Indonesia, approximately 750,000 deaths occurred due to CVD in 2021 [3]. In 2019, it was estimated that 5.8 million new cases of ischemic heart disease occurred in 57 countries that are members of the European Society of Cardiology (ESC). In the United States, one case of acute myocardial infarction (AMI) occurs every 40 seconds. AMI is the most severe form of coronary artery disease (CAD), with a risk of sudden death or severe hemodynamic disturbances and high mortality rates: 38% in women and 44% in men [2].

In Indonesia, the prevalence of CVD in 2021 was 13.8 million [3]. Data from the One ACS multicenter registry reported that out of 7,634 AMI patients, 48.8% had ST-elevation myocardial infarction (STEMI), and the rest had non-ST-elevation myocardial infarction (NSTEMI). Among STEMI patients, 65.2% received reperfusion therapy, but only 45.7% underwent primary percutaneous coronary intervention (PCI) [4]. In NSTEMI patients, an invasive strategy was applied to 17.6%, with only 6.7% undergoing early PCI (<24 hours). The mortality rate among AMI patients was 8.9%, with higher mortality in STEMI compared to NSTEMI (11.7% vs 6.2%). In recent years, there has been a significant decline in the incidence of AMI in European countries and the United States [5]. Despite a global decline in AMI mortality due to advances in management over the past two decades, AMI remains the most frequent cause of heart failure, with high morbidity and mortality rates [6]. Heart failure due to AMI has a high morbidity and mortality rate [7]. Heart failure related to AMI increases the risk of death from all causes by three

times and cardiovascular death by four times [8]. Heart failure has a significant impact on the healthcare system in the United States, affecting 6 million people, causing 300,000 deaths annually, and costing approximately 40 billion dollars [9]. Additionally, the economic impact of AMI is substantial. In the United States, in 2010, there were more than 1.1 million hospitalizations due to AMI, with direct costs estimated at 450 billion dollars [10].

Therefore, assessing the individual risk of major cardiovascular events (MACE), which could be fatal, is a crucial diagnostic approach to ensure a reduction in readmission rates and overall mortality in high-risk patient populations. In the era of personalized medicine, the role of biomarkers in diagnosis and prognosis is becoming increasingly important. This rapidly developing field requires the availability of easily assessable prognostic markers to identify at-risk patients with ease. Several independent cardiovascular risk factors associated with insulin, which have been shown to impact clinical outcomes in AMI patients, include obesity, sedentary lifestyle, hypertriglyceridemia, and inflammatory biomarkers (such as high-sensitivity C-reactive protein [hs-CRP]) [11].

The liver, as the largest visceral organ, receives about 25% of cardiac output 20-25% via the hepatic artery and 75-80% through the hepatic portal vein [12]. This unique dual blood supply supports the liver's high metabolic activity, which requires a high oxygen demand, approximately 20% of the body's total needs [12]. Additionally, the liver extracts 95% of the blood oxygen to maintain adequate oxygen absorption [13]. This mechanism makes the liver relatively resistant to hepatocyte necrosis caused by perfusion disturbances alone. However, when visceral blood flow significantly decreases or severe hypoxemia occurs, the protective mechanisms of the liver against hypoxic damage are overwhelmed [13]. Hemodynamic changes due to systemic congestion and/or increased cardiac filling pressures are critical for liver dysfunction to occur [14]. The severity and pattern of liver injury depend on the relative contributions of passive congestion and reduced perfusion [15]. Acute heart failure caused by STEMI can lead to acute cardiac liver injury due to a combination of decreased cardiac output, sudden tissue perfusion impairment, and venous congestion [16]. Evidence of liver dysfunction in myocardial injury and heart failure is associated with the occurrence of MACE and mortality from all causes, as supported by studies on STEMI patients who underwent primary PCI and subsequently had liver magnetic resonance imaging (MRI) [17].

The acute and chronic interactions between the heart and liver are known as cardiohepatic syndrome (CHS), which can be initiated by either heart or liver dysfunction [18]. CHS is divided into five types: types 1 and 2 show the impact of acute and chronic heart disease on the liver, while types 3 and 4 describe the effects of acute and chronic liver disease on the heart. Type 5 is characterized by simultaneous heart and liver dysfunction due to systemic acute or chronic disturbances, such as sepsis [18].

In this context, transaminases like aspartate aminotransferase (AST) and alanine aminotransferase (ALT) are easy to perform and assess in clinical practice and, importantly, are widely available [19]. ALT is predominantly found in hepatocytes and is a widely used serum marker specific to liver disease. AST primarily originates from the liver, but it is also present in other tissues such as the heart, red blood cells, and muscles, making it a less specific marker for liver function [19]. AST has high activity in the heart and will be released into circulation during heart disturbances such as ischemia and myocardial infarction. Existing studies show increasing interest in investigating the role of liver transaminases in independently predicting heart-related morbidity and mortality [20].

Several prospective epidemiological studies have shown that liver dysfunction is common in heart disease [21]. In the absence of other identified causes of liver injury, elevated liver aminotransferase levels are associated with a higher incidence of heart-related death [22]. However, the results of these studies have not been consistent, and geographic variations have been found in the relationship between ALT and all-cause mortality [23]. Some studies emphasize that liver dysfunction is common in CVD patients, even examining the predictive value of transaminases for morbidity and mortality in cardiovascular populations [19]. Initially, most of these studies focused on patients with chronic heart failure, with only a few conducted on individuals with CAD or AMI. However, recent studies have shown an association between liver dysfunction and mortality in patients with AMI undergoing PCI [20]. A study by Gao et al. on 2,417 STEMI patients undergoing primary PCI found that elevated serum transaminase levels were associated with both short-term and long-term mortality [19]. Similarly, a retrospective study by Li et al. on 712 AMI patients found that elevated ALT levels were an independent predictor of increased in-hospital mortality [20].

Based on recent evidence, transaminases such as AST and ALT can serve as predictive markers for patient outcomes following acute cardiac events [24]. Given that ALT primarily reflects liver-specific dysfunction and AST is known to increase following ischemic cell death in several other tissues, including the kidneys, skeletal muscles, and even the brain, the AST/ALT ratio (De Ritis) can be a surrogate marker for ischemic organ damage during the acute phase of AMI. This has been demonstrated in studies such as that by Steininger et al., who found a strong correlation between the De Ritis ratio and variables such as age, female sex, STEMI, maximum troponin T levels, creatine kinase, and inversely with glomerular filtration rate (GFR). The strongest correlations were found with NT-proBNP and CRP levels. The study concluded that the De Ritis ratio is a strong independent predictor of long-term mortality [25].

A recent study on 120 AMI patients found that a De Ritis ratio ≥2.0 was strongly associated with total coronary artery occlusion [26]. Since the De Ritis ratio enhances the discriminative power of risk assessment in AMI patients, and existing studies show it to be an independent predictor of long-term mortality in AMI, we aim to investigate the prognostic potential of the De Ritis ratio in patient outcomes after AMI from both the in-hospital and 30-day post-discharge perspectives, addressing the limitation of modern biomarker availability, such as high-sensitivity troponin I (hs-TnI), particularly in remote areas.

METHODS

Study Design

This study was an analytical observational study with a retrospective cohort design. The investigation began with the assessment of serum aspartate aminotransferase (AST) and alanine aminotransferase (ALT) levels obtained at the time of first presentation to the emergency department (ED). The De Ritis ratio (AST/ALT) was subsequently calculated and evaluated as the exposure variable. Patients were then followed for the occurrence of major adverse cardiovascular events (MACE) during hospitalization and within 30 days after discharge.

Study Setting and Period

The study was conducted at RSUP Prof. I. G. N. G. Ngoerah, Denpasar, Bali, Indonesia. Eligible patients were identified at the emergency department and subsequently admitted to the cardiac intensive care unit or cardiology ward. Blood samples were analyzed at the Clinical Pathology Laboratory of RSUP Prof. I. G. N. G. Ngoerah. Outcome assessment was based on data retrieved from the hospital's electronic medical records. Data collection was performed between June and October 2025, while patient records were retrospectively reviewed for the period from January 2023 to December 2024.

Study Population and Sample

The target population consisted of adult patients aged 18 years or older diagnosed with acute myocardial infarction, including ST-segment elevation myocardial infarction (STEMI) and non-ST-segment elevation myocardial infarction (NSTEMI), who underwent percutaneous coronary intervention and required hospitalization. The accessible population included all eligible patients fulfilling these criteria who were treated at RSUP Prof. I. G. N. G. Ngoerah during the specified study period. Sample selection was performed using a consecutive sampling method, whereby all patients meeting the inclusion criteria and none of the exclusion criteria were included sequentially until the required sample size was achieved.

Eligibility Criteria

Eligible participants were patients aged 18 years or older with a diagnosis of STEMI or NSTEMI who underwent percutaneous coronary intervention and had no prior history of liver disease before the procedure. Patients were excluded if they had incomplete medical records, severe infection or sepsis, active malignancy, autoimmune diseases such as systemic lupus erythematosus, Sjögren's syndrome, or rheumatoid arthritis, severe renal dysfunction defined as chronic kidney disease stage 4 or 5, drug-induced liver injury, chronic hepatitis or liver cirrhosis, acute stroke or a history of stroke, prior acute coronary syndrome within one month before the index event, recent trauma, or recent strenuous physical exercise.

Sample Size Calculation

The minimum sample size was calculated using the cohort study formula proposed by Lwanga and Lemeshow. With a type I error of 5% and a statistical power of 80%, the assumed proportion of major adverse cardiovascular events was 24.9% in the high De Ritis ratio group and 4.9% in the low De Ritis ratio group, based on previous literature. The calculated minimum sample size was 49 participants per group. To account for potential dropouts, an additional 10% was added, resulting in 54 participants per group and a total sample size of 108 patients.

Study Variables

The primary exposure variable was the De Ritis ratio, calculated from AST and ALT values obtained from the first blood sample collected before percutaneous coronary intervention. The ratio was analyzed as both a continuous variable and a categorical variable. Categorization into high and low De Ritis ratio groups was performed using an optimal cut-off value derived from receiver operating characteristic curve analysis. The primary outcome was major adverse cardiovascular events occurring during hospitalization or within 30 days after discharge. Major adverse cardiovascular events were defined as a composite outcome consisting of cardiovascular death, acute heart failure, malignant arrhythmia, cardiogenic shock, mechanical complications such as left ventricular free wall rupture, interventricular septal rupture, or acute mitral regurgitation, and cardiovascular-related rehospitalization. The outcome variable was treated as nominal.

Potential confounding variables included demographic factors such as age and sex, clinical characteristics including infarct type and location, symptom-to-balloon time particularly in STEMI cases, left ventricular ejection fraction, tricuspid annular plane systolic excursion, comorbidities such as diabetes mellitus, hypertension and smoking status, as well as coronary angiography findings including the number of diseased vessels, pre- and post-procedural TIMI flow, residual thrombus, lesion characteristics and revascularization strategy. These variables were considered in multivariable analyses.

Data Collection Procedures

Venous blood samples for AST and ALT measurement were obtained at the emergency department prior to percutaneous coronary intervention. Laboratory analyses were performed

using the Alinity C Analyzer with an enzymatic method, and the results were expressed in units per liter. The De Ritis ratio was calculated by dividing the AST value by the ALT value. Patients were followed throughout hospitalization and for 30 days after discharge. The time from percutaneous coronary intervention to the occurrence of major adverse cardiovascular events was recorded in hours as time-to-event data.

Outcome Assessment

Major adverse cardiovascular events were identified through a comprehensive review of electronic medical records. Malignant arrhythmias were confirmed by electrocardiographic findings, cardiogenic shock was assessed based on hemodynamic parameters and clinical judgment, acute heart failure and mechanical complications were evaluated using echocardiography and chest radiography, and rehospitalization events were verified through hospital records or structured telephone interviews conducted after discharge. Cardiovascular death occurring outside the hospital was confirmed through family interviews and official death certificates.

Statistical Analysis

All statistical analyses were performed using SPSS software version 29.0. Descriptive statistics were used to summarize baseline characteristics of the study population. Continuous variables with normal distribution were presented as mean and standard deviation, while non-normally distributed variables were expressed as median and interquartile range. Categorical variables were summarized as frequencies and percentages. Receiver operating characteristic curve analysis was conducted to determine the optimal cut-off value of the De Ritis ratio for predicting major adverse cardiovascular events, including calculation of the area under the curve, sensitivity, and specificity. Comparisons between high and low De Ritis ratio groups were performed using the Chi-square test for categorical variables. Cox proportional hazards regression analysis was used to evaluate the independent association between the De Ritis ratio and the occurrence of major adverse cardiovascular events after adjustment for potential confounders. Adjusted hazard ratios with 95% confidence intervals were reported, and a two-sided p-value of less than 0.05 was considered statistically significant.

RESULT

This study involved patients with acute myocardial infarction (both STEMI and NSTEMI) who underwent percutaneous coronary intervention. A total of 278 patients were included as study participants, and observations were conducted to assess the occurrence of major adverse cardiovascular events and their association with the De Ritis ratio. The baseline characteristics of the study subjects are presented in Table 1.

TABLE 1: Baseline Characteristics of Study Participants According to De Ritis Ratio.

Variable	Total (n = 278)	De Ritis ≥ 2.03 (n = 142)	De Ritis < 2.03 (n = 136)	p-value
Age (years), mean ± SD	56.74 ± 10.36	58.88 ± 10.51	45.52 ± 9.77	<0.001
Sex, n (%)				**0.484**
Male	247 (88.8%)	128 (90.1%)	119 (87.5%)	
Female	31 (11.2%)	14 (9.9%)	17 (12.5%)	
Type of Myocardial Infarction, n (%)				**0.732**
STEMI	261 (93.9%)	134 (94.4%)	127 (93.4%)	
NSTEMI	17 (6.1%)	8 (5.6%)	9 (6.6%)	
Killip Class, n (%)				**0.271**
I	194 (69.8%)	93 (68.9%)	101 (79.5%)	
II	40 (14.4%)	26 (19.3%)	14 (11.0%)	
III	8 (2.9%)	4 (3.0%)	4 (3.1%)	
IV	19 (6.8%)	11 (8.1%)	8 (6.3%)	
Infarct Location				**0.154**
LAD	162 (58.3%)	86 (60.6%)	76 (55.9%)	
LCx	20 (7.2%)	14 (9.9%)	6 (4.4%)	
LM	4 (1.4%)	2 (1.4%)	2 (1.5%)	
RCA	92 (33.1%)	40 (28.2%)	52 (38.2%)	
Hypertension, n (%)	146 (52.5%)	75 (52.8%)	71 (52.2%)	0.919
Diabetes Mellitus, n (%)	65 (23.4%)	33 (23.2%)	32 (23.5%)	0.954
Smoking, n (%)	166 (59.7%)	88 (62.0%)	78 (57.4%)	0.433
Atrial Fibrillation, n (%)	12 (4.3%)	7 (4.9%)	5 (3.7%)	0.607
Thrombus Grade				**0.003**
0	13 (4.7%)	9 (6.3%)	4 (2.9%)	
1	2 (0.7%)	2 (1.4%)	0 (0%)	
2	7 (2.5%)	4 (2.8%)	3 (2.2%)	
3	23 (8.3%)	14 (9.9%)	9 (6.6%)	
4	96 (34.5%)	60 (42.3%)	36 (26.5%)	
5	137 (49.3%)	53 (37.3%)	84 (61.8%)	

Variable	Total (n = 278)	De Ritis ≥ 2.03 (n = 142)	De Ritis < 2.03 (n = 136)	p-value
LVEF (%), median (min–max)	47 (18–66)	45 (18–66)	49 (28–65)	0.005
TAPSE (mm), median (min–max)	21 (2–37)	21 (2–37)	21 (11–30)	0.793
Creatinine (mg/dL), median (min–max)	1.00 (0.59–2.22)	1.01 (0.59–2.13)	1.02 (0.59–2.22)	0.313
BUN (mg/dL), median (min–max)	12.4 (5.80–37.20)	12.5 (5.80–28.80)	12.5 (6.50–37.20)	0.499
eGFR (mL/min/1.73 m^2), median (min–max)	80.90 (21.92–124.87)	81.41 (21.92–112.97)	80.66 (31.29–124.87)	0.903
Symptom onset (hours), median (min–max)	6 (1–12)	7 (2–12)	5 (0.5–12)	<0.001
Pre-procedural TIMI Flow				**<0.001**
0	136 (48.8%)	52 (36.6%)	84 (61.8%)	
1	4 (1.4%)	4 (2.8%)	0 (0%)	
2	44 (15.8%)	24 (16.9%)	20 (14.7%)	
3	94 (33.8%)	62 (43.7%)	32 (23.5%)	
Coronary Angiography Findings, n (%)				**0.006**
Single-vessel disease	96 (34.5%)	50 (35.2%)	46 (33.8%)	
Two-vessel disease	98 (35.3%)	59 (41.5%)	39 (28.7%)	
Three-vessel disease	56 (20.1%)	17 (12.0%)	39 (28.7%)	
Left main disease	6 (2.2%)	2 (1.4%)	4 (2.9%)	
Chronic total occlusion	19 (6.8%)	13 (9.2%)	6 (4.4%)	
Thrombus-prone morphology	3 (1.1%)	1 (0.7%)	2 (1.5%)	
In-hospital MACE, n (%)	193 (69.4%)	108 (76.1%)	85 (62.5%)	0.014
Mortality	14 (5.0%)	8 (5.6%)	6 (4.4%)	0.641
Acute Heart Failure	188 (67.6%)	105 (73.9%)	83 (61.0%)	0.021
Malignant Arrhythmia	21 (7.6%)	8 (5.6%)	13 (9.6%)	0.216
Cardiogenic Shock	33 (11.9%)	19 (13.4%)	14 (10.3%)	0.426
Mechanical Complications	0 (0%)	0	0	–
Rehospitalization	8 (2.9%)	6 (4.2%)	2 (1.5%)	0.170
AST (U/L), median (min–max)	65.5 (14–1241)	130.3 (26–1241)	39.5 (15–738)	<0.001
ALT (U/L), median (min–max)	33 (7–2304)	33 (6–568)	33 (11–702)	<0.001
Troponin, median (min–max)	2,763 (4.3–270,374)	13,995 (214–270,374)	868 (4.3–104,162)	<0.001
Hemoglobin (g/dL), median (min–max)	14.10 (7.80–19.20)	14.3 (7.9–19.2)	14.1 (7.8–18.3)	0.812
WBC ($\times 10^9$/L), median (min–max)	12.36 (4.45–27.97)	12.69 (4.45–27.97)	12.08 (4.98–26.68)	0.017
Neutrophils ($\times 10^9$/L), median (min–max)	9.76 (2.49–25.60)	10.63 (2.49–25.60)	9.13 (2.71–20.40)	<0.001
Lymphocytes ($\times 10^9$/L), median (min–max)	1.62 (0.43–5.64)	1.52 (0.43–3.55)	1.75 (0.64–5.64)	<0.001
Neutrophil-to-Lymphocyte Ratio	6.10 (1.06–28.07)	7.15 (1.92–28.07)	5.54 (1.06–26.94)	<0.001
Time to MACE (hours), median (min–max)	4.5 (0–107.5)	9 (2.10–168.25)	7.75 (1–168)	0.485

Based on the ROC curve analysis, the area under the curve (AUC) for the De Ritis ratio in predicting in-hospital and 30-day MACE among patients with acute myocardial infarction undergoing percutaneous coronary intervention was presented in Figure 1 and Table 2.

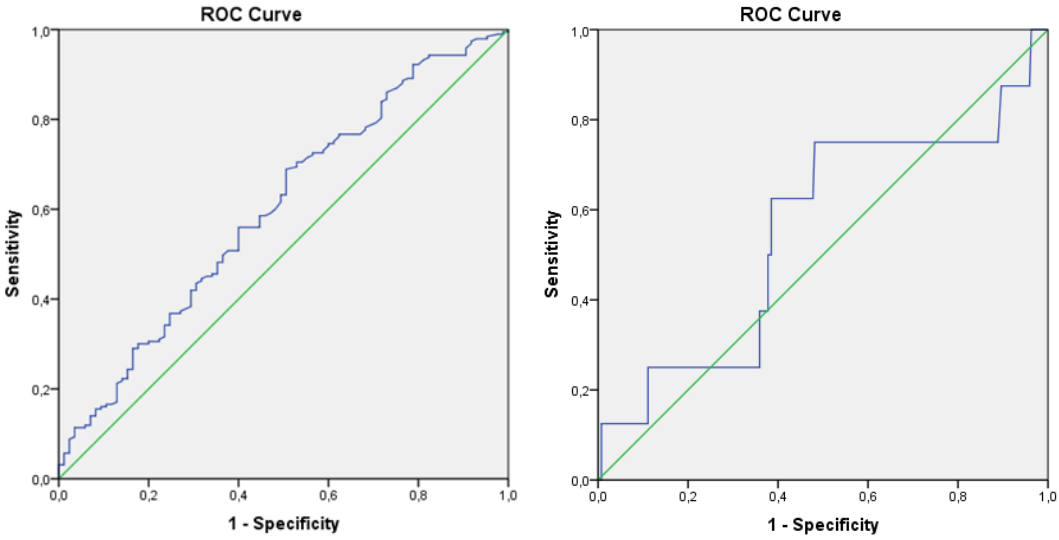

FIGURE 1: ROC Curves for De Ritis Ratio in Predicting In-Hospital and 30-Day MACE in Acute Myocardial Infarction Patients Undergoing Percutaneous Coronary Intervention.

TABLE 2: Cut-off Value of the De Ritis Ratio as a Predictor of In-Hospital and 30-Day Major Adverse Cardiovascular Events in Patients with Acute Myocardial Infarction Undergoing Percutaneous Coronary Intervention.

Variable	AUC	P value	95%CI	*Cut-off*	Sensitivity	Specificity
In-hospital MACE	0,600	0,008	0,527-0,673	2,03	56%	60%
30-Day MACE	0,553	0,608	0,333-0,773	2,55	62,5%	61,5%

Kaplan–Meier survival analysis and Cox proportional hazards regression were performed to evaluate the association between the De Ritis ratio and in-hospital MACE while controlling for various clinical and laboratory variables (Figure 2). The analysis demonstrated that a De Ritis ratio ≥2.03 was a significant predictor of MACE, with an adjusted hazard ratio of 1.842 (95% CI: 1.297–2.616; p = 0.001). This indicates that patients with a De Ritis ratio ≥2.03 had a 1.8-fold higher risk of experiencing MACE during hospitalization compared with those with a ratio <2.03, after adjustment for other variables. In addition to the De Ritis ratio, troponin levels, and the neutrophil-to-lymphocyte ratio were also identified as significant predictors. Furthermore, pre-procedural TIMI flow showed a significant association with in-hospital MACE, with a hazard ratio of 1.392 (95% CI: 1.168–1.659; p < 0.001), indicating that poorer TIMI flow before intervention was associated with a higher risk of MACE during hospitalization. In contrast, other variables, including age, sex, ejection fraction, TAPSE, hypertension, diabetes mellitus, smoking status, thrombus grade, estimated glomerular filtration rate, and blood urea nitrogen, were not significantly associated with MACE (p > 0.05) (Table 3).

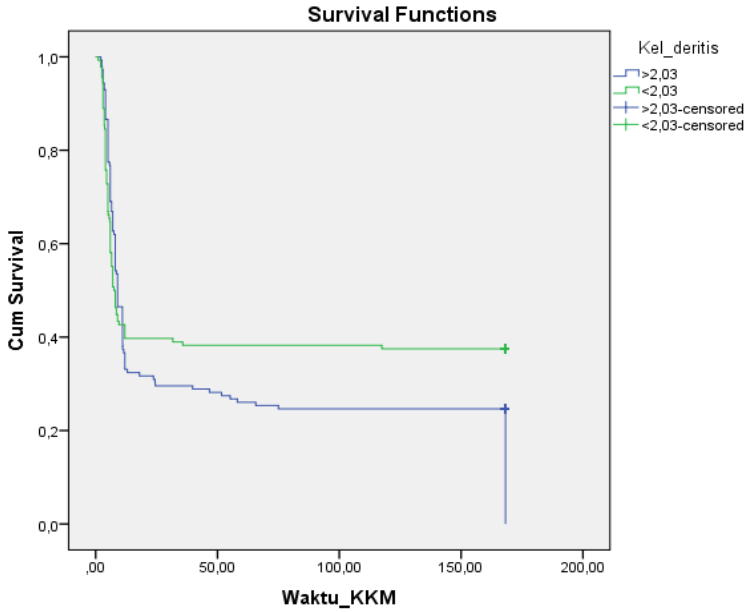

FIGURE 2: Kaplan-Meier Curve of De-Ritis Ratio as a Predictor of In-Hospital MACE in Acute Myocardial Infarction Patients Undergoing Percutaneous Coronary Intervention.

TABLE 3: Cox Regression Analysis of De-Ritis Ratio as a Predictor of In-Hospital MACE in Acute Myocardial Infarction Patients Undergoing Percutaneous Coronary Intervention.

Variable	Adjusted Hazard Ratio	95% CI Lower	95% CI Upper	p-value
De-Ritis ≥2.03	1.842	1.297	2.616	0.001
Age	0.981	0.958	1.006	0.129
Gender	0.708	0.375	1.337	0.288
EF	0.998	0.974	1.023	0.881
TAPSE	0.984	0.940	1.030	0.491
Hypertension	1.249	0.911	1.714	0.168
Diabetes Mellitus	1.182	0.831	1.682	0.353
Smoking	0.874	0.613	1.246	0.456
TIMI	1.392	1.168	1.659	0.000
Thrombus Grade	1.166	0.967	1.406	0.109
Location	1.072	0.942	1.220	0.289
CAG Result	0.959	0.844	1.089	0.519
Troponin ≥1411.6	1.695	1.208	2.380	0.002
LFG	0.983	0.957	1.011	0.227
WBC ≥11.98	1.303	0.974	1.745	0.075
NLR ≥4.91	1.680	1.225	2.302	0.001
SC	0.371	0.059	2.316	0.289
BUN	0.988	0.950	1.027	0.551

Cox regression analysis was performed to evaluate whether the De-Ritis ratio plays a role as a predictor of 30-day MACE after hospitalization in acute myocardial infarction patients undergoing percutaneous coronary intervention, adjusted for other clinical and laboratory factors. The analysis results showed that a De-Ritis ratio ≥ 2.55 was not significantly associated with 30-day MACE occurrence (Adjusted Hazard Ratio [HR] 2.208; 95% CI: 0.157–31.140; p = 0.557). This indicates that, although there is a tendency for an increased risk of MACE in patients with a high De-Ritis ratio, the relationship is not statistically significant after adjusting for other variables (Table 4).

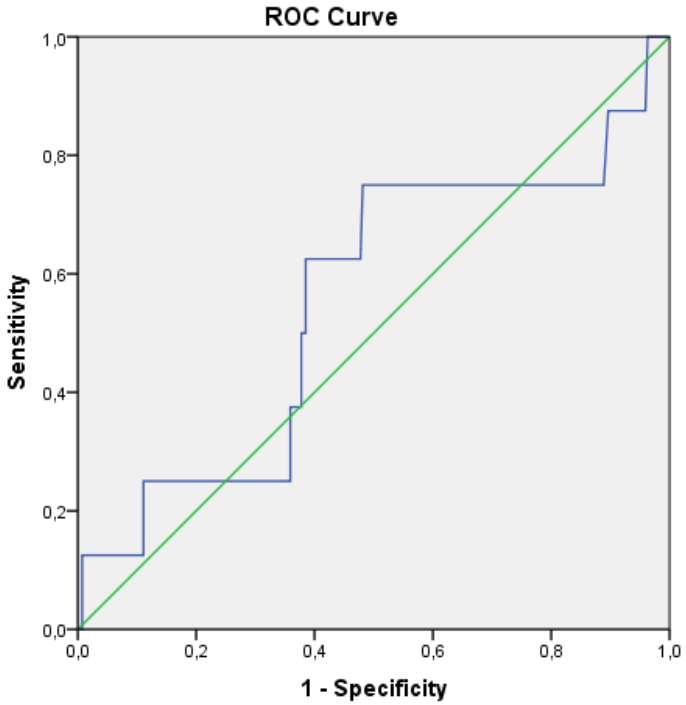

FIGURE 3: Kaplan-Meier Curve of De-Ritis Ratio as a Predictor of In-Hospital MACE in Acute Myocardial Infarction Patients Undergoing Percutaneous Coronary Intervention.

TABLE 4: Cox Regression Analysis of De-Ritis Ratio as a Predictor of In-Hospital MACE in Acute Myocardial Infarction Patients Undergoing Percutaneous Coronary Intervention.

Variable	Adjusted Hazard Ratio	95% CI Lower	95% CI Upper	p-value
De-Ritis ≥2.55	2.208	0.157	31.140	0.557
Age	1.231	1.015	1.493	0.035
Gender	0.792	0.014	45.80	0.910
EF (Ejection Fraction)	0.913	0.849	0.981	0.013
Troponin	1.000	1.000	1.000	0.970
WBC	1.487	0.949	2.329	0.083
NLR	0.902	0.675	1.206	0.486
LFG (Left-ventricular Fraction)	1.064	0.968	1.169	0.197
TAPSE	0.993	0.660	1.496	0.975
Hypertension (HT)	3.038	0.245	37.602	0.387
Diabetes Mellitus (DM)	0.143	0.013	1.564	0.111
Smoking	0.899	0.057	14.100	0.939
TIMI	1.072	0.451	2.549	0.876

DISCUSSION

The average age of patients with AMI undergoing PCI in this study was 56.74 ± 10.36 years, indicating that AMI most commonly occurs in the middle-aged to elderly population. In the study by Steininger et al., AMI patients in the lower De-Ritis quartile were 64 (40–72) years old, while those in the upper quartile were 75 (45–83) years old [25]. In a study conducted in the United States from 2005 to 2019, the average age of AMI patients undergoing PCI was 78.3 ± 6.7 years [27], while in Bhutan, it was 63.5 ± 16.8 years [28] and in Japan, 68.5 ± 12.1 years [29]. A meta-analysis of 29 studies showed that the risk of AMI occurrence progressively increases with age due to the chronic process of atherosclerosis [30]. The aging process causes a decrease in blood vessel elasticity, increased arterial stiffness, and changes in endothelial function, leading to vascular dysfunction and an increased risk of thrombosis. Prognostically, elderly patients typically have poorer responses to reperfusion therapy, increased risks of post-PCI complications, and slower myocardial recovery compared to younger patients [31].

In this study, the majority of AMI patients undergoing PCI were male (88.8%). This is consistent with other studies that report a higher incidence of AMI in males compared to females, with variations of 75.6% [29], 53% [27], and 81.44% [31]. Biologically, this difference is largely due to the protective effect of estrogen on the cardiovascular system in premenopausal women, which improves insulin sensitivity, endothelial function, and increases HDL cholesterol levels. After menopause, when estrogen levels decrease, the risk of coronary artery disease in women increases, approaching or even surpassing that of men in the elderly. Men are also more likely to have higher exposure to classical risk factors such as smoking, work-related stress, and alcohol consumption, which contribute to early-onset atherosclerosis. Conversely, women often present with atypical symptoms (such as dyspnea, fatigue, or epigastric pain), which can lead to delayed diagnosis and management of AMI [32].

Based on infarction type, the majority of patients experienced ST-elevation myocardial infarction (STEMI) at 93.9%, while non-ST-elevation myocardial infarction (NSTEMI) was found in 17 patients (6.1%). This number is similar to a study in China, where 86.6% of PCI patients had STEMI [31]. In this study, there were more STEMI patients because PCI is the preferred reperfusion therapy for STEMI. STEMI is caused by total occlusion of the coronary artery due to acute thrombosis, requiring immediate action to restore coronary blood flow. In contrast, NSTEMI patients typically experience partial occlusion or temporary coronary spasm, often managed first with medication or performed electively (non-emergency PCI) [33].

According to the Killip classification, most patients in this study were in Killip class I (69.8%), followed by Killip II (14.4%), Killip IV (6.8%), and Killip III (2.9%), indicating that the majority of patients presented with hemodynamically stable conditions without signs of heart failure. The Killip classification is used to assess the degree of left ventricular dysfunction and the severity of acute heart failure in myocardial infarction patients, where Killip I indicates no heart failure signs, and Killip IV represents cardiogenic shock as the most severe manifestation [34].

The most common infarction lesions were found in the Left Anterior Descending (LAD) artery at 58.3%, followed by the Right Coronary Artery (RCA) at 33.1%, the Left Circumflex (LCx) at 7.2%, and the LM at 1.4%. In the study by Cheng et al. (2022), 63.91% of infarction lesions were in the LAD, 19.58% in LCx, and 21.65% in RCA. Other studies reported LAD involvement at 49%, RCA at 35.3%, and LCx at 15.7% (Geraiely et al., 2018). The dominance of lesions in the LAD artery aligns with findings from several studies showing that LAD is the most frequent site of occlusion in AMI [35]. This is due to the anatomical role of LAD, which supplies blood to the majority of the anterior left ventricular wall and most of the heart's conduction system, making occlusion in this

artery leads to large myocardial necrosis and a significant impact on cardiac pump function. RCA involvement in 33.1% of cases represents lesions in the inferior wall of the heart, which, although relatively smaller, can still cause complications like atrioventricular conduction issues and hemodynamic disturbances when accompanied by right ventricular involvement. The smaller proportion of LCx involvement (7.2%) corresponds to the more limited coronary distribution, while LM involvement (1.4%), though rare, is the most fatal as it can cause massive ischemia in nearly the entire left ventricle [36].

The main cardiovascular risk factors identified were hypertension in 146 patients (52.5%), a history of smoking in 166 patients (59.7%), and diabetes mellitus in 65 patients (23.4%). Atrial fibrillation (AF) was found in 12 patients (4.3%). In the study by Steininger et al., 66.4% of AMI patients undergoing PCI had hypertension, 21.2% had diabetes mellitus, 71.2% had hypercholesterolemia, and 63.1% were smokers.[25] In the study by Cheng et al., 40.2% of AMI patients undergoing PCI had hypertension, 25.77% had diabetes mellitus, 62.88% were smokers, and 3.1% had AF.[31] In the study by Chempay et al., 42.6% of AMI patients undergoing PCI had hypertension, 17% had diabetes mellitus, 13.8% had dyslipidemia, 32.8% were smokers, and 3.2% had AF [28].

The thrombus grade assessment in this study showed that the majority of patients had high-grade thrombus, with grade 5 at 49.3% and grade 4 at 34.5%, while grades 0–3 were found in less than 15% of patients. This finding indicates that most patients presented with heavy thrombus burden, an important indicator in determining the severity of coronary artery occlusion and the potential for thrombotic complications during PCI. The high proportion of grade 4–5 thrombus aligns with the study population's characteristics, which predominantly consisted of STEMI patients, where total artery occlusion due to a ruptured atherosclerotic plaque and massive thrombus formation is the main mechanism. The presence of large thrombus not only complicates mechanical reperfusion during PCI but also increases the risk of no-reflow phenomena, distal embolization, and major cardiovascular events post-procedure [31].

The TIMI flow distribution before intervention showed that nearly half of the patients (48.8%) had TIMI flow 0, which indicates no coronary blood flow due to total thrombotic occlusion, while only 33.8% of patients had TIMI flow 3, indicating normal blood flow before the intervention. These results are similar to the study by Cheng et al., which reported that 69.07% of AMI patients undergoing PCI had TIMI flow 0 before PCI, and 16.49% had TIMI flow 3 [31]. This finding illustrates that most patients presented with significant coronary artery occlusion, typically associated with the dominance of STEMI cases in this study population. Physiologically, TIMI flow is an important parameter for assessing myocardial perfusion and is often linked to post-intervention prognosis. Patients with TIMI flow 0–1 before the procedure typically have a larger area of myocardial necrosis, worse left ventricular function, and higher risks of heart complications such as heart failure and ventricular arrhythmias. In contrast, patients with TIMI flow 3 show partial spontaneous reperfusion, which is usually correlated with better clinical outcomes post-procedure [37].

The TAPSE value had a median of 21 mm (range 2–37), indicating that, in general, the right ventricular function of the patients remained within normal limits, considering that TAPSE ≥17 mm is typically regarded as the lower limit of normal right ventricular systolic function according to ASE/EACVI guidelines [38]. This suggests that the majority of patients in this study did not experience significant right ventricular dysfunction, despite having AMI and undergoing PCI. The right ventricle plays an essential role in maintaining total cardiac output and pulmonary perfusion, particularly in the post-infarction setting. Right ventricular dysfunction often occurs in inferior or inferoposterior infarctions due to RCA involvement, which supplies most of the right ventricular wall. Therefore, the normal TAPSE in the majority of patients suggests that right myocardial damage was relatively minimal, or successful reperfusion was achieved, preventing severe right ventricular contractility disturbances. However, the wide range of TAPSE values (2–37 mm) indicates clinical variability between patients, where a small subset may have experienced severe right ventricular failure due to extensive RCA involvement or delayed reperfusion. Low TAPSE in this group may correlate with worse prognosis, including increased risks of cardiogenic shock, ventricular arrhythmias, and short-term mortality [39].

Renal function parameters in this study showed a median creatinine level of 1.0 mg/dL (0.59–2.22), BUN 12.4 mg/dL (5.80–37.20), and e-LFG 80.90 mL/min/1.73 m^2 (21.92–124.87), which generally indicated that the renal function of the majority of patients was relatively good. In the study by Wang et al., the average e-LFG in AMI patients post-PCI was 61.32 (24.61) [40]. In the study by Steininger et al. (2018), the median e-LFG in AMI patients without liver dysfunction was 101.9 (67.6–122.0), whereas with liver dysfunction, it was 66.7 (45.2–103.2). An e-LFG value above 60 mL/min/1.73 m^2 indicates no significant renal filtration dysfunction in the majority of subjects. This finding is important because AMI patients often experience acute renal dysfunction due to systemic hypoperfusion, decreased cardiac output, or the nephrotoxic effects of contrast agents used during PCI. However, the wide range of e-LFG values suggests that a small number of patients may have experienced mild to moderate renal function decline, which requires attention, as decreased e-LFG is known to increase the risk of cardiovascular complications and mortality post-PCI.

Liver and heart enzyme parameters showed a median SGOT of 65.5 U/L (14–1241), SGPT of 33 U/L (7–2304), with a De-Ritis ratio of 2.12 (0.53–14.65),

indicating a dominance of SGOT elevation in the acute phase of myocardial infarction. In the study by Wang et al., the average AST in AMI patients post-PCI was 24.66±10.93, while ALT was 20.90±12.19 [40]. In the study by Steininger et al., the average AST in AMI patients post-PCI was 29 (17–42), while ALT was 36 (22–60) [25]. Troponin levels varied widely, with a median of 2763 ng/L (4.3–270,374), reflecting the degree of myocardial damage in some patients. The median hemoglobin was 14.10 g/dL (7.80–19.20), leukocytes 12.36 $\times 10^9$/L (4.45–27.97), neutrophils 9.76 $\times 10^9$/L (2.49–25.60), lymphocytes 1.62 $\times 10^9$/L (0.43–5.64), and a median NLR ratio of 6.10 (1.06–28.07), indicating systemic inflammatory response in most patients.

During treatment, 193 patients (69.4%) experienced in-hospital MACE, while only 8 patients (2.9%) experienced 30-day MACE after hospitalization. Clinical outcomes showed a mortality rate of 5.0%, heart failure in 67.6% of patients, malignant arrhythmias in 7.6%, and cardiogenic shock in 11.9% of patients. No complications were found post-PCI. In the study by Wang et al., 28 out of 204 patients (13.7%) experienced MACE, and 8.8% died within 24 months post-PCI [40]. In the study by Steininger et al., 40.9% of 11,046 AMI patients died within 8.7 years post-PCI [25]. In the study by Ndrepepa et al., 384 out of 5020 patients (7.6%) with AMI died within 3 years post-PCI [41].

This study found that a De-Ritis ratio ≥ 2.03 was a significant predictor of MACE occurrence with an Adjusted Hazard Ratio (HR) of 1.842 (95% CI: 1.297–2.616; p = 0.001) after adjusting for variables such as troponin, NLR, renal function, and comorbidities. These findings align with several international studies that have highlighted the De-Ritis ratio as an important predictor of clinical outcomes in AMI patients. The study by Steininger et al. showed that in 1,355 AMI patients, a high De-Ritis ratio was significantly associated with increased long-term mortality, even after adjusting for key clinical factors such as NT-proBNP, troponin T, and creatine kinase. The median De-Ritis ratio in that population was 1.5, and for every 1-SD increase in the De-Ritis ratio, the long-term mortality risk increased by 23% (HR = 1.23; 95% CI: 1.07–1.42; p = 0.004) [25].

Similarly, Wang et al. found that a high De-Ritis ratio was an independent risk factor for major adverse cardiac and cerebrovascular events (MACCE) and mortality in patients with stable coronary artery disease undergoing elective PCI. In multivariate Cox analysis, patients with a high De-Ritis ratio had nearly three times the risk of MACCE (HR = 2.96; 95% CI: 1.29–6.78; p = 0.01) and more than three times the risk of all-cause mortality (HR = 3.61; 95% CI: 1.31–9.86; p = 0.012) [40].

Ndrepepa et al. reported a similar relationship in 5,020 patients with chronic coronary syndrome (CCS) undergoing PCI. Patients with a De-Ritis ratio in the highest tertile (>1.08) had a higher 3-year mortality rate (14.5%) compared to those in the first tertile (5.0%). Multivariate analysis showed a 9% increased risk of death for each unit increase in the De-Ritis ratio (HR = 1.09; 95% CI: 1.06–1.12; p < 0.001). Interestingly, adding the De-Ritis ratio to the prediction model increased the C-statistic from 0.815 to 0.818 (p = 0.005), indicating that this ratio contributes to the accuracy of predicting mortality risk [41].

Djakpo et al. studied 120 AMI patients without a history of liver disease to assess the relationship between the De-Ritis ratio and infarct type and severity. The results showed that the mean De-Ritis ratio was significantly higher in STEMI patients (3.23 ± 2.41) compared to NSTEMI patients (2.21 ± 1.63), with a statistically significant difference (p = 0.002). The researchers also found that a De-Ritis ratio of AST/ALT ≥ 2.0 strongly correlated with total coronary artery occlusion [26].

The higher cut-off for the De-Ritis ratio in this study (≥ 2.03) compared to previous studies may be due to the high proportion of STEMI patients in the study population. STEMI typically causes larger areas of myocardial necrosis, significantly increasing AST release, while ALT remains relatively stable. This condition shifts the De-Ritis ratio distribution to higher values, so the optimal cut-off for predicting MACE is at a higher level compared to populations with a larger proportion of NSTEMI.

The sensitivity of the De-Ritis ratio as a predictor of MACE appears low (56%), which could be attributed to the dominance of Killip class I patients in the study population. In this low-risk group, MACE events are less frequent, and the degree of systemic hypoperfusion affecting AST/ALT elevation is not as pronounced, making the difference in De-Ritis ratios between patients with and without MACE less distinct. This increases the proportion of false negatives and lowers the test's sensitivity.

The De-Ritis ratio may affect MACE because physiologically, AST is found not only in the liver but also in the heart, kidneys, and skeletal muscles, whereas ALT is more specific to the liver. During a myocardial infarction, necrotic heart cells release AST into the circulation, significantly increasing the AST/ALT ratio. Additionally, systemic hypoperfusion due to reduced cardiac output can cause hepatic hypoxia, worsening liver dysfunction, and further increasing AST release. This phenomenon explains the positive relationship between a high De-Ritis ratio and the increased risk of in-hospital MACE. Patients with high De-Ritis ratios generally have unstable hemodynamic conditions and more severe systemic ischemia [25].

The analysis results showed that the De-Ritis ratio ≥ 2.55 was not significantly associated with 30-day MACE occurrence after hospitalization (Adjusted Hazard Ratio [HR] 2.208; 95% CI: 0.157–31.140; p = 0.557). This finding differs from several previous studies that reported the De-Ritis ratio as a predictor of long-term cardiovascular outcomes [25], [41].

This difference can be explained by several factors. First, the follow-up duration in this study was relatively short (30 days), while studies like Steininger et al. evaluated risks over several years, with a follow-up period of 8.6 years [25]. The De-Ritis ratio tends to reflect tissue damage and chronic systemic stress, effects that become more apparent in the subacute or chronic phase. Second, the study population consisted of AMI patients undergoing PCI with high reperfusion success, so acute factors such as left ventricular function and TIMI flow success were more determinative of outcomes than enzymatic biomarkers. Third, the low number of MACE events in the 30-day post-hospitalization period reduced the statistical power of the analysis, resulting in a wide confidence interval. Thus, although the direction of the relationship indicates a trend toward increased risk in the high De-Ritis ratio group, the impact on 30-day MACE after hospitalization was not statistically significant enough.

This study has several limitations that should be considered in interpreting the results. First, the low incidence of 30-day MACE may reduce the statistical power of the analysis, particularly in multivariate models, so some potential relationships may not achieve statistical significance. Second, this study only observed 30-day MACE, which is a short period compared to other studies with follow-up times spanning several years. Third, this study did not evaluate longitudinal changes in AST and ALT levels during treatment or after discharge, while fluctuations in liver enzymes could affect the interpretation of the De-Ritis ratio as a dynamic biomarker. Fourth, the distribution of STEMI and NSTEMI patients was uneven, with a dominance of STEMI cases, so the sample may not accurately represent the NSTEMI population. Fifth, the majority of STEMI patients were in Killip class I, reducing sensitivity in predicting MACE. Sixth, the study was conducted in a single tertiary healthcare center, so the generalization of the results to broader populations or facilities with different patient characteristics, such as primary healthcare centers, should be done with caution. Nonetheless, this study provides important contributions to understanding the potential of the De-Ritis ratio as a prognostic marker in AMI patients undergoing PCI.

CONCLUSION

The De Ritis ratio is a predictor of in-hospital major adverse cardiovascular events (MACE) in patients with acute myocardial infarction (AMI) undergoing percutaneous coronary intervention (PCI), but it is not a predictor of 30-day MACE after hospitalization. This study emphasizes the value of the De Ritis ratio as a simple and cost-effective additional parameter for predicting and stratifying the risk of complications during hospitalization in AMI patients. Patients with a high De Ritis ratio may benefit from more potent antiplatelet therapy and high-intensity statins, and AMI patients, particularly those with NSTEMI and a high De Ritis ratio, may be considered for early PCI. Future research should focus on achieving a balanced sample of STEMI and NSTEMI patients, particularly STEMI patients with Killip class ≥2, to improve sensitivity. Additionally, studies conducted over a longer follow-up period and across multiple centers would enhance the strength and generalizability of the findings.

Acknowledgments

All authors, patients, and supporters in the paper.

Declarations

Funding: No funding sources.
Conflict of interest: None declared.
Ethical approval: The study was approved by Udayana University with the number 1996/UN14.2.2.VII.14/LT/2025.

REFERENCES

[1] Moraes-Silva, I. C., Rodrigues, B., Coelho-Junior, H. J., Feriani, D. J., & Irigoyen, M.-C. (2017). Myocardial Infarction and Exercise Training: Evidence from Basic Science. *Advances in Experimental Medicine and Biology*, 999, 139–153. https://doi.org/10.1007/978-981-10-4307-9_9

[2] Diseasestroke, S., Update, S., Hypertensive, S., LDL, & High, S. (2024). Global cardiovascular disease statistics. *No.* July 2023, 2023–2024.

[3] Lindstrom, M., et al. (2022). Global Burden of Cardiovascular Diseases and Risks Collaboration, 1990-2021. *Journal of the American College of Cardiology*, 80(25), 2372–2425. https://doi.org/10.1016/j.jacc.2022.11.001

[4] Juzar, D., et al. (2022). Management of Acute Coronary Syndrome in Indonesia: Insight from One ACS Multicenter Registry. *Indonesian Journal of Cardiology*, 43(2 SE-Clinical Research). https://doi.org/10.30701/ijc.1406

[5] Dégano, I. R., et al. (2015). Twenty-five-year trends in myocardial infarction attack and mortality rates, and case-fatality, in six European populations. *Heart*, 101(17), 1413–1421. https://doi.org/10.1136/heartjnl-2014-307310

[6] Jenča, D., et al. (2021). Heart failure after myocardial infarction: incidence and predictors. *ESC Heart Failure*, 8(1), 222–237. https://doi.org/10.1002/ehf2.13144

[7] Juillière, Y., et al. (2012). Heart failure in acute myocardial infarction: a comparison between patients with or without heart failure criteria from the FAST-MI registry. *Revista Española de Cardiología (English Edition)*, 65(4), 326–333. https://doi.org/10.1016/j.recesp.2011.10.027

[8] Gerber, Y., et al. (2016). Mortality Associated With Heart Failure After Myocardial Infarction: A Contemporary Community Perspective. *Circulation: Heart Failure*, 9(1), e002460. https://doi.org/10.1161/CIRCHEARTFAILURE.115.002460

[9] Roger, V. L., et al. (2012). Heart disease and stroke statistics--2012 update: a report from the American Heart Association. *Circulation*, 125(1), e2–e220. https://doi.org/10.1161/CIR.0b013e31823ac046

[10] Weintraub, W. S., et al. (2011). Value of primordial and primary prevention for cardiovascular disease: a policy statement from the American Heart Association. *Circulation*, 124(8), 967–990. https://doi.org/10.1161/CIR.0b013e3182285a81

[11] Zarich, S., Luciano, C., Hulford, J., & Abdullah, A. (2006). Prevalence of metabolic syndrome in young patients with acute MI: does the Framingham Risk Score underestimate cardiovascular risk in this population? *Diabetes Vascular Disease Research*, 3(2), 103–107. https://doi.org/10.3132/dvdr.2006.012

[12] Vollmar, B., & Menger, M. D. (2009). The hepatic microcirculation: mechanistic contributions and therapeutic targets in liver injury and repair. *Physiological Reviews*, 89(4), 1269–1339. https://doi.org/10.1152/physrev.00027.2008

[13] Naschitz, J. E., Slobodin, G., Lewis, R. J., Zuckerman, E., & Yeshurun, D. (2000). Heart diseases affecting the liver and liver diseases affecting the heart. *American Heart Journal*, 140(1), 111–120. https://doi.org/10.1067/mhj.2000.107177

[14] Shirakabe, A., et al. (2023). Organ dysfunction, injury, and failure in cardiogenic shock. *Journal of Intensive Care*, 11(1), 26. https://doi.org/10.1186/s40560-023-00676-1

[15] Giallourakis, C. C., Rosenberg, P. M., & Friedman, L. S. (2002). The liver in heart failure. *Clinical Liver Disease*, 6(4), 947–67, viii–ix. https://doi.org/10.1016/s1089-3261(02)00056-9

[16] Hilscher, M., & Sanchez, W. (2016). Congestive hepatopathy. *Clinical Liver Disease*, 8(3), 68–71. https://doi.org/10.1002/cld.573

[17] Lechner, I., et al. (2024). Hepatic Tissue Alterations in ST-Elevation Myocardial Infarction: Determinants and Prognostic Implications. *Circulation Cardiovascular Imaging*, 17(12), e017041. https://doi.org/10.1161/CIRCIMAGING.124.017041

[18] Poelzl, G., & Auer, J. (2015). Cardiohepatic syndrome. *Current Heart Failure Reports*, 12(1), 68–78. https://doi.org/10.1007/s11897-014-0238-0

[19] Gao, M., et al. (2017). Association of serum transaminases with short- and long-term outcomes in patients with ST-elevation myocardial infarction undergoing primary percutaneous coronary intervention. *BMC Cardiovascular Disorders*, 17(1), 43. https://doi.org/10.1186/s12872-017-0485-6

[20] Li, J., et al. (2021). Predictive value of elevated alanine aminotransferase for in-hospital mortality in patients with acute myocardial infarction. *BMC Cardiovascular Disorders*, 21(1), 82. https://doi.org/10.1186/s12872-021-01903-z

[21] Fouad, Y. M., & Yehia, R. (2014). Hepato-cardiac disorders. *World Journal of Hepatology*, 6(1), 41–54. https://doi.org/10.4254/wjh.v6.i1.41

[22] Yun, K. E., et al. (2009). Elevated alanine aminotransferase levels predict mortality from cardiovascular disease and diabetes in Koreans. *Atherosclerosis*, 205(2), 533–537. https://doi.org/10.1016/j.atherosclerosis.2008.12.012

[23] Kunutsor, S. K., et al. (2014). Liver enzymes and risk of all-cause mortality in general populations: a systematic review and meta-analysis. *International Journal of Epidemiology*, 43(1), 187–201. https://doi.org/10.1093/ije/dyt192

[24] Sulzgruber, P., et al. (2015). Butyrylcholinesterase predicts cardiac mortality in young patients with acute coronary syndrome. *PLoS One*, 10(5), e0123948. https://doi.org/10.1371/journal.pone.0123948

[25] Steininger, M., et al. (2018). De-Ritis Ratio Improves Long-Term Risk Prediction after Acute Myocardial Infarction. *Journal of Clinical Medicine*, 7(12). https://doi.org/10.3390/jcm7120474

[26] Djakpo, D. K., Wang, Z. Q., & Shrestha, M. (2020). The significance of transaminase ratio (AST/ALT) in acute myocardial infarction. *Archives of Medical Sciences: Atherosclerotic Diseases*, 5, e279–e283. https://doi.org/10.5114/amsad.2020.103028

[27] DeBarmore, B. M., Zègre-Hemsey, J. K., Kucharska-Newton, A. M., Michos, E. D., & Rosamond, W. D. (2023). Patient characteristics and outcomes of acute myocardial infarction presenting without ischemic pain: Insights from the Atherosclerosis Risk in Communities Study. *American Heart Journal* plus *Cardiology Research and Practice*, 25. https://doi.org/10.1016/j.ahjo.2022.100239

[28] Chempay, Y., Dorjey, U., Tshering, M., & Watts, M. R. (2025). Clinical characteristics and outcomes of acute coronary syndrome patients in a PCI-Limited setting: a prospective study from Bhutan. *BMC Cardiovascular Disorders*, 25(1), 324. https://doi.org/10.1186/s12872-025-04782-w

[29] Sonoda, T., et al. (2022). Clinical features and predictors of outcome in patients with acute myocardial infarction complicated by out-of-hospital cardiac arrest. *BMC Cardiovascular Disorders*, 22(1), 185. https://doi.org/10.1186/s12872-022-02628-3

[30] Rajati, F., Rajati, M., Chegeni, M., & Kazeminia, M. (2024). The prevalence of myocardial infarction in the elderly: A systematic review and meta-analysis. *ARYA Atherosclerosis*, 20(2), 61–73. https://doi.org/10.48305/arya.2024.42327.2930

[31] Cheng, Z., Shi, Y., Peng, H., Zhao, D., Fan, Q., & Liu, J. (2022). Prognostic Significance of Percutaneous Coronary Intervention for First Acute Myocardial Infarction with Heart Failure: Five-Year Follow-Up Results. *Cardiology Research and Practice*, 2022, p. 5791295. https://doi.org/10.1155/2022/5791295

[32] Lee, M., et al. (2020). Gender differences in clinical outcomes of acute myocardial infarction undergoing percutaneous coronary intervention: insights from the KAMIR-NIH Registry. *Journal of Geriatric Cardiology*, 17(11), 680–693. https://doi.org/10.11909/j.issn.1671-5411.2020.11.006

[33] Fatima, S., Harinstein, M. E., Hussain, M., & Pacella, J. J. (2025). Percutaneous coronary intervention for stable late ST-elevation myocardial infarction with symptoms onset between 12 and 72 h - A systematic review. *Cardiovascular Revascularization Medicine*, 78, 1–9. https://doi.org/10.1016/j.carrev.2024.12.014

[34] Hashmi, K. A., et al. (2020). Risk Assessment of Patients After ST-Segment Elevation Myocardial Infarction by Killip Classification: An Institutional Experience. *Cureus*, 12(12), e12209. https://doi.org/10.7759/cureus.12209

[35] Rus, M., et al. (2025). Proximal vs. Distal LAD Lesions in ST-Elevation Myocardial Infarction: Insights from ECG and Coronary Angiography. *Journal of Clinical Medicine*, 14(16). https://doi.org/10.3390/jcm14165637

[36] Garcia, D. C., et al. (2019). Metaanalysis of Multivessel vs Culprit Artery Only Percutaneous Coronary Intervention in ST Elevation Myocardial Infarction. *Ochsner Journal*, 19(2), 107–115. https://doi.org/10.31486/toj.18.0033

[37] Shavadia, J. S., et al. (2020). Novel Biomarkers, ST-Elevation Resolution, and Clinical Outcomes Following Primary Percutaneous Coronary Intervention. *Journal of the American Heart Association*, 9(13), e016033. https://doi.org/10.1161/JAHA.120.016033

[38] Modin, D., Møgelvang, R., Andersen, D. M., & Biering-Sørensen, T. (2019). Right Ventricular Function Evaluated by Tricuspid Annular Plane Systolic Excursion Predicts Cardiovascular Death in the General Population. *Journal of the American Heart Association*, 8(10), e012197. https://doi.org/10.1161/JAHA.119.012197

[39] Tao, R., et al. (2024). Relationship of TAPSE Normalized by Right Ventricular Area With Pulmonary Compliance, Exercise Capacity, and Clinical Outcomes. *Circulation: Heart Failure*, 17(5), e010826. https://doi.org/10.1161/CIRCHEARTFAILURE.123.010826

[40] Wang, K., Chen, Z., Zeng, D., & Ran, M. (2022). Impact of the De Ritis Ratio on the Prognosis of Patients with Stable Coronary Artery Disease Undergoing Percutaneous Coronary Intervention. *Medical Science Monitor*, 28, e937737-1–e937737-8. Available at: https://api.semanticscholar.org/CorpusID:254331897.

[41] Ndrepepa, G. (2023). De Ritis ratio and cardiovascular disease: evidence and underlying mechanisms. *Journal of Laboratory and Precision Medicine*, 8. https://doi.org/10.21037/JLPM-22-68.