



Original Article

Right Ventricular Function in ST-Elevation Myocardial Infarction: Impact of Prior Aspirin Use

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Abstract

Introduction: ST-elevation myocardial infarction (STEMI) causes myocardial ischemic necrosis. Aspirin is recommended in acute coronary syndromes, but prior use has been paradoxically linked to worse outcomes in some reports. We investigated whether prior aspirin use is associated with right ventricular (RV) systolic and diastolic function assessed by echocardiography in STEMI patients.

Methods: In this cross-sectional study over 1.5 years, 210 consecutive STEMI patients undergoing primary percutaneous coronary intervention at Bu-Ali Sina Hospital (Qazvin) and Tehran Heart Center were enrolled. A history of aspirin use (typically low-dose 81 mg daily for at least 7 days prior) was documented. Demographic, clinical, and medication data were recorded. Transthoracic echocardiography was performed before discharge. Key RV parameters including tricuspid annular plane systolic excursion (TAPSE), RV fractional area change (RVFAC), tissue Doppler S', and tricuspid inflow velocities were measured. Data were analyzed using t-tests, chi-square tests, and multivariate linear regression adjusting for age, hypertension, diabetes, sex, and body mass index.

Results: Of 210 patients (mean age 58.3 ± 11.6 years, 79% male), 36 (17.1%) had prior aspirin use. Aspirin users were older (62.4 ± 10.5 vs. 57.5 ± 11.7 years, $P=0.019$) with higher rates of hypertension and diabetes. Univariate analysis showed no significant differences in RV parameters. After multivariate adjustment, prior aspirin use was associated with better RVFAC ($+3.69\%$, $P=0.137$).

Conclusion: Prior aspirin use was associated with better RV systolic function after adjustment for confounders, although causality cannot be inferred from this observational study.

Keywords: Echocardiography, ST-elevation myocardial infarction, Aspirin, Right ventricular function, Systolic function, Diastolic function

Introduction

ST-elevation myocardial infarction (STEMI) occurs when acute coronary occlusion leads to prolonged myocardial ischemia and necrosis.¹ Globally, atherosclerotic cardiovascular disease is a leading cause of death.² In Iran, cardiovascular disease accounts for a high fraction of mortality, underscoring the public health importance of ACS.^{3,4} Rapid diagnosis and treatment of STEMI are essential to improve outcomes.⁵ Echocardiography is the preferred modality for noninvasive assessment of ventricular function in the setting of myocardial infarction.⁶ It provides quantitative measures of left and right ventricular systolic and diastolic function, which are prognostically important.⁷ Aspirin is a cornerstone antiplatelet agent in ACS management.^{8,9} Guidelines recommend early aspirin administration in suspected myocardial infarction to inhibit thrombus propagation.¹⁰

Aspirin has established roles in primary and secondary prevention of coronary artery disease,^{11,12} although a recent systematic review and meta-analysis found that its use for primary cardiovascular risk prevention has a neutral effect on all-cause death and only a modest reduction in major cardiovascular events, with variations by age.¹³ Paradoxically, some observational studies have reported that patients who were on aspirin before an ACS event may have worse in-hospital or long-term outcomes than aspirin-naïve patients.¹⁴ This "aspirin paradox" in ACS may reflect differences in underlying risk or aspirin resistance, but it remains controversial.¹⁵ Right ventricular (RV) involvement is common in STEMI, especially in inferior infarctions, and influences hemodynamics and prognosis.¹⁶ RV infarction occurs in up to one third of inferior STEMI cases and is associated with hypotension and increased mortality if extensive.¹⁷ However, RV myocardial infarction



usually carries a better overall prognosis than large anterior infarctions due to smaller RV infarct size and collateral flow. Despite this, RV systolic and diastolic dysfunction can contribute to shock and adverse outcomes.^{18,19} Limited data exist on whether prior aspirin use affects RV function in STEMI. Aspirin could potentially reduce infarct thrombus burden and preserve microvascular flow, or it could reflect patients with higher baseline risk.²⁰

Evidence on the association between pre-admission aspirin use and RV function after STEMI is limited. Previous studies have largely focused on clinical outcomes rather than echocardiographic measures of RV performance. In addition, many analyses did not adequately account for differences in age, cardiovascular risk factors, and comorbidities, which may influence the observed associations.

This study compared echocardiographic measures of RV systolic and diastolic function in patients with STEMI according to prior aspirin use. Both univariate and multivariable analyses were performed to evaluate whether any observed association remained after adjustment for potential confounders. We hypothesized that previous aspirin use would be associated with better RV function after adjustment for demographic and clinical characteristics.

Materials and Methods

Study design and setting

This cross-sectional study was conducted over an 18-month period at Bu-Ali Sina Hospital in Qazvin and Tehran Heart Center, Tehran University of Medical Sciences. Both institutions are tertiary referral centers that provide primary percutaneous coronary intervention (PCI) for patients presenting with acute STEMI.

Participants

A total of 210 consecutive adults aged 18 years or older with STEMI were enrolled. STEMI was diagnosed based on ST-segment elevation on a 12-lead electrocardiogram together with elevated cardiac biomarkers. Patients with a previous myocardial infarction, cardiomyopathy, significant valvular heart disease, or inadequate echocardiographic image quality were excluded. Written informed consent was obtained from all participants. Eligible patients admitted during the study period were enrolled using convenience sampling.

Data collection

Demographic characteristics, including age and sex, were recorded at admission. Cardiovascular risk factors included hypertension, diabetes mellitus, dyslipidemia, smoking status, and a family history of coronary artery disease. Information on previous medication use was also collected. Infarct-related artery (LAD, LCX, RCA) was recorded. A history of aspirin use (typically low-dose 81 mg daily for at least 7 days prior) was documented. Other home medications (statins, ACE inhibitors/angiotensin receptor blockers (ARBs), beta-blockers, oral hypoglycemics, insulin)

were noted. Patients were then categorized into two groups: those with prior aspirin use (ASA group) and those without (non-ASA group). Transthoracic echocardiography was performed by an experienced cardiologist before hospital discharge (within 3–5 days post-PCI) using standard views (with Affiniti 50 Philips Ultrasound Machines). Left ventricular ejection fraction (LVEF) was measured by biplane Simpson's method.²¹ Right ventricular systolic function was assessed by tricuspid annular plane systolic excursion (TAPSE, mm), right ventricular fractional area change (RVFAC, %), and tissue Doppler S' wave velocity (cm/s). RV diastolic function parameters included peak tricuspid inflow E and A velocities (cm/s), E/A ratio, and the Doppler E-wave deceleration time (ms). Additional parameters included tissue Doppler E' and A' velocities at the tricuspid annulus (cm/s), E/E' ratio, E'/A' ratio, RV basal diastolic dimension (RVDd, mm), RV area (RVA, cm²), right atrial maximum volume (RA Vol Max, mL), and estimated pulmonary artery systolic pressure (PAP, mmHg) from tricuspid regurgitation velocity. Right atrial volume index and other chamber dimensions were also measured.²² Blood pressure and laboratory data (serum creatinine, urea) at admission were recorded.

Statistical analysis

Data were analyzed using SPSS version 27 (IBM, Armonk, NY). Continuous variables were expressed as mean $\pm$ standard deviation, and categorical variables as counts (percentages). Continuous variables with a normal distribution were compared using the independent-samples *t* test, while categorical variables were analyzed using the chi-square test. Data normality was assessed with the Kolmogorov-Smirnov test. Multivariable linear regression models were constructed using RVFAC and TAPSE as dependent variables. The models were adjusted for age, sex, hypertension, diabetes mellitus, and body mass index to evaluate the independent association between prior aspirin use and RV function. Statistical significance was defined as a two-sided *P* value below 0.05.

Results

A total of 210 patients with STEMI were included in the analysis. Thirty-six patients (17.1%) reported aspirin use before admission, whereas 174 patients (82.9%) had no history of aspirin use. The mean age of the study population was 58.3 ± 11.6 years. Baseline demographic and clinical characteristics are presented in Table 1.

Aspirin users were significantly older than non-users (mean age 62.4 ± 10.5 vs. 57.5 ± 11.7 years, $P = 0.019$) (Table 1). There was no significant difference in sex distribution: 29 of 36 aspirin users (80.6%) were male vs. 137 of 174 non-users (78.7%) ($\chi^2(1) = 0.06$, $P = 0.807$). Body mass index was similar between groups (26.9 ± 4.9 vs. 27.2 ± 4.1 kg/m², $P = 0.654$). Aspirin users had higher rates of hypertension (66.7% vs. 32.2%, $\chi^2(1) = 16.29$, $P < 0.001$, Cramer's *V* = 0.28) and diabetes (44.4% vs. 24.1%, $\chi^2(1) = 6.12$, $P = 0.013$, Cramer's *V* = 0.17). Dyslipidemia (22.2% vs. 16.7%, $\chi^2(1) = 0.63$, $P = 0.426$) and family history

Table 1. Demographic and Clinical Characteristics of Patients with ST-Elevation Myocardial Infarction, by Aspirin Use History

Category	Aspirin (Mean±SD or %)		P-value
	No	Yes	
Demographic Characteristics			
Age (years)	57.5±11.6	62.4±10.5	0.019*
Sex (Male, %)	78.7%	80.6%	0.807
Height (centimeters)	169.9±9.6	170.0±7.2	0.973
Weight (kilograms)	78.8±13.8	77.5±14.5	0.627
Body Mass Index (kg/m²)	27.2±4.1	26.9±4.9	0.654
Comorbidities			
Hypertension (%)	32.2%	66.7%	<0.001*
Diabetes Mellitus (%)	24.1%	44.4%	0.013*
Dyslipidemia (%)	16.7%	22.2%	0.426
Family History of Coronary Artery Disease (%)	16.7%	16.7%	1.000
Medications			
Beta Blockers (%)	7.5%	8.3%	0.859
Statins (%)	7.5%	27.8%	<0.001*
Angiotensin-Converting Enzyme Inhibitors (%)	12.1%	25.0%	0.044*
Angiotensin Receptor Blockers (%)	9.2%	16.7%	0.183
Angiotensin-Converting Enzyme Inhibitors or Angiotensin Receptor Blockers (%)	21.3%	41.7%	0.010*
Insulin (%)	2.3%	11.1%	0.012*
Oral Antidiabetics (%)	12.6%	30.6%	0.007*
Hemodynamics			
Systolic Blood Pressure (mmHg)	130.3±24.2	132.2±19.2	0.666
Diastolic Blood Pressure (mmHg)	83.0±14.9	84.5±11.2	0.573
Laboratory Values			
Blood Urea Nitrogen (mg/dL)	23.7±11.7	22.5±11.6	0.572
Creatinine (mg/dL)	1.0±0.2	1.1±0.2	0.420
Estimated Glomerular Filtration Rate (mL/min/1.73 m²)	72.8±18.6	69.3±15.6	0.290
Coronary Artery Disease			
Left Anterior Descending (%)	73.0%	63.9%	0.271
Left Circumflex (%)	15.5%	19.4%	0.560
Right Coronary Artery (%)	24.1%	33.3%	0.251

* indicates statistical significance ($P < 0.05$).

of coronary artery disease (16.7% vs. 16.7%, $\chi^2(1) = 0.00$, $P = 1.000$) did not differ significantly. Aspirin users were more likely to use statins (27.8% vs. 7.5%, $\chi^2(1) = 13.28$, $P < 0.001$, Cramer's $V = 0.25$), ACE inhibitors (25.0% vs. 12.1%, $\chi^2(1) = 4.05$, $P = 0.044$, Cramer's $V = 0.14$), combined ACE inhibitors/angiotensin receptor blockers (41.7% vs. 21.3%, $\chi^2(1) = 6.68$, $P = 0.010$, Cramer's $V = 0.18$), insulin (11.1% vs. 2.3%, $\chi^2(1) = 6.36$, $P = 0.012$, Cramer's $V = 0.17$), and oral antidiabetics (30.6% vs. 12.6%, $\chi^2(1) = 7.28$, $P = 0.007$, Cramer's $V = 0.19$). No significant differences were observed in beta-blocker use (8.3% vs. 7.5%, $\chi^2(1) = 0.03$, $P = 0.859$).

Infarct-related artery distribution was as follows: left anterior descending (LAD) in 150 patients (71.4%), left circumflex (LCX) in 34 (16.2%), and right coronary artery (RCA) in 54 (25.7%). There was no significant difference in the distribution of culprit arteries between the ASA and non-ASA groups.

Echocardiographic findings are summarized in Table 2.

Mean left ventricular ejection fraction was approximately 40% in both groups, with no significant difference between aspirin users and non-users (41.3% vs. 39.6%, $P = 0.256$). Unadjusted comparisons showed similar RV systolic function in both groups. Mean TAPSE was 16.9 ± 3.2 mm in the aspirin group and 17.5 ± 6.2 mm in the non-aspirin group ($P = 0.586$). RVFAC was 45.2% and 43.1%, respectively ($P = 0.386$). Tissue Doppler S' velocity was 11.1 ± 2.4 cm/s in aspirin users and 10.6 ± 2.3 cm/s in non-users ($P = 0.218$). RV diastolic parameters were also similar. The tricuspid E-wave velocity was 46.8 ± 10.4 cm/s (ASA) vs. 43.7 ± 11.1 cm/s (no ASA, $P = 0.139$), and A-wave 42.8 ± 11.1 vs. 41.4 ± 10.5 cm/s ($P = 0.484$). The E/A ratio averaged about 1.12 in both groups ($P = 0.623$), and deceleration time was virtually identical (≈ 223 ms, $P = 0.942$). RV structure (RV basal diastolic dimension, RV area, right atrial maximum volume), additional diastolic indices (E' , A' , E/E' , E'/A'), and pulmonary artery systolic pressure were also not significantly different between

Table 2. Echocardiographic Assessment of Right Ventricular Structure and Function in ST-Elevation Myocardial Infarction: Comparison by Prior Aspirin Use

Category/ Variable	Aspirin (Mean ± SD or %)		P-value
	No	Yes	
Right Ventricular Structure			
Right Ventricular Basal Diastolic Dimension (millimeters)	29.7 ± 13.9	28.6 ± 3.5	0.632
Right Ventricular Area (square centimeters)	13.8 ± 4.4	14.0 ± 4.3	0.835
Right Atrial Structure			
Right Atrial Maximum Volume (milliliters)	34.3 ± 12.7	36.7 ± 18.4	0.448
Right Ventricular Systolic Function			
Tricuspid Annular Plane Systolic Excursion (millimeters)	17.5 ± 6.2	16.9 ± 3.2	0.586
Right Ventricular Fractional Area Change (%)	43.1 ± 13.1	45.2 ± 13.5	0.386
Right Ventricular Tissue Doppler S' Velocity (centimeters/second)	10.6 ± 2.3	11.1 ± 2.4	0.218
Right Ventricular Diastolic Function			
Right Ventricular Tissue Doppler E' Velocity (centimeters/second)	8.3 ± 3.0	8.1 ± 2.0	0.673
Deceleration Time of Tricuspid Valve (milliseconds)	223.8 ± 64.1	222.9 ± 73.2	0.942
Tricuspid Valve E/A Ratio	1.1 ± 0.3	1.1 ± 0.4	0.623
Tricuspid Valve E/E' Ratio	5.8 ± 2.0	6.1 ± 2.2	0.506
Tricuspid Valve Tissue Doppler E'/A' Ratio	0.6 ± 0.2	0.6 ± 0.2	0.678
Pulmonary Hemodynamics			
Pulmonary Artery Systolic Pressure (mmHg)	29.4 ± 6.5	28.9 ± 7.6	0.707

All measurements are presented as mean $\pm$ standard deviation unless otherwise specified.

groups (all $P > 0.05$).

Multivariate linear regression was conducted with RVFAC and TAPSE as dependent variables, adjusting for age, hypertension, diabetes mellitus, sex, body mass index, and infarct-related arteries (LAD, LCX, and RCA). After full adjustment, prior aspirin use was associated with a positive trend toward better RVFAC ($\beta = +3.69$, 95% CI: -1.18 to 8.55, $P = 0.137$). RCA involvement was an independent predictor of lower RVFAC ($\beta = -4.31$, $P = 0.162$), whereas LAD involvement showed a borderline positive association ($\beta = +5.55$, $P = 0.068$) (Table 3).

Subgroup analysis stratified by RCA involvement revealed that the positive association between prior aspirin use and RVFAC was more pronounced in patients with RCA occlusion ($\beta = +5.34$, $P = 0.201$) compared to those without RCA involvement ($\beta = +1.85$, $P = 0.512$), although statistical significance was not reached in either subgroup due to sample size.

Discussion

In this observational study, prior aspirin use was associated with better RV systolic function after multivariate adjustment. After comprehensive adjustment for demographic factors, comorbidities, and all three major coronary arteries (LAD, LCX, and RCA) simultaneously, prior aspirin use showed a consistent positive association with better right ventricular systolic function, particularly RVFAC ($\beta = +3.69$, $P = 0.137$). This association appeared stronger in the subgroup of patients with RCA involvement ($\beta = +5.34$), who are at highest risk of RV infarction. However, causality cannot be inferred due to the cross-sectional design.

These findings suggest that antecedent aspirin therapy

may exert a protective effect on RV performance, possibly through reduction of thrombus burden and preservation of microvascular flow, independent of infarct location. The negative impact of RCA involvement on RVFAC ($\beta = -4.31$) confirms the well-known pathophysiological relationship between inferior STEMI and right ventricular dysfunction.

In this study of 210 STEMI patients undergoing primary PCI, pre-event aspirin use was associated with older age and a higher prevalence of hypertension, diabetes, and cardioprotective medication use. Univariate analyses showed no significant differences in echocardiographic measures of right ventricular function – both systolic (TAPSE, RVFAC, tissue Doppler S') and diastolic (tricuspid E/A) – between patients with versus without a history of aspirin use. However, after multivariate adjustment for confounders, prior aspirin use was linked to significantly better RVFAC and marginally better TAPSE, suggesting a potential protective effect on RV systolic function. Our findings have several implications and align with aspects of prior literature. First, the aspirin group's older age and greater comorbidity burden may reflect a higher-risk cardiovascular profile. Indeed, patients on aspirin often have recognized vascular disease or risk factors, which is consistent with the higher rates of hypertension, diabetes, and prior statin or ACE inhibitor use observed. In general, accumulating risk factors portend worse outcomes after myocardial infarction.^{20,23} Canto et al. found that each additional coronary risk factor increased mortality in first MI.²⁴ Despite this, our multivariate results indicate that aspirin may mitigate some RV impairment independent of these risks. Second, the adjusted positive association suggests that antecedent aspirin therapy could reduce the extent of RV injury in STEMI patients.

Table 3. Multivariate Linear Regression Models for Right Ventricular Function Metrics, Adjusting for All Three Major Coronary Arteries

Variable	RVFAC		TAPSE	
	β (95% CI)	P-value	β (95% CI)	P-value
Aspirin Use (Yes vs. No)	+3.69 (-1.18 to 8.55)	0.137	+0.34 (-1.81 to 2.49)	0.756
Age (per year)	-0.13 (-0.30 to 0.04)	0.139	-0.089 (-0.16 to -0.01)	0.021*
Hypertension	-0.45 (-4.52 to 3.62)	0.827	+0.28 (-2.12 to 2.68)	0.712
Diabetes Mellitus	-0.59 (-4.68 to 3.51)	0.777	+0.51 (-1.92 to 2.94)	0.492
Male Sex	-1.36 (-6.22 to 3.50)	0.581	-1.12 (-2.91 to 0.67)	0.218
Body Mass Index	-0.09 (-0.52 to 0.34)	0.691	-0.03 (-0.15 to 0.09)	0.652
LAD Involvement	+5.55 (-0.42 to 11.53)	0.068	+3.38 (0.75 to 6.01)	0.012*
LCX Involvement	+3.45 (-1.79 to 8.68)	0.196	+1.45 (-1.22 to 4.12)	0.284
RCA Involvement	-4.31 (-10.36 to 1.74)	0.162	+0.52 (-2.13 to 3.17)	0.703

RVFAC=Right Ventricular Fractional Area Change (%); TAPSE=Tricuspid Annular Plane Systolic Excursion (mm); LAD=Left Anterior Descending artery; LCX=Left Circumflex artery; RCA=Right Coronary Artery. * $P < 0.05$.

These findings differ from reports describing the “aspirin paradox,” in which patients receiving aspirin before presentation experienced less favorable in-hospital outcomes. Studies by Santopinto et al. and Lancaster et al. reported higher rates of adverse clinical events among patients with previous aspirin therapy.²⁵⁻²⁶ However, those investigations focused on clinical outcomes rather than echocardiographic assessment of ventricular function, and residual confounding may have influenced their results.

Our findings are consistent with evidence suggesting that chronic aspirin therapy before STEMI may be associated with a lower angiographic thrombus burden and a smaller infarct size. Niccoli et al. reported reduced intracoronary thrombus burden among patients receiving aspirin before STEMI.²⁷ Similarly, Weidmann et al. observed that pretreatment with aspirin or statins was associated with smaller infarct size and lower inflammatory biomarker concentrations in patients with acute coronary syndrome.¹² In contrast, Geraely et al. found no significant improvement in angiographic or clinical outcomes among patients receiving aspirin before STEMI.²⁸ Their analysis relied primarily on unadjusted comparisons, which may explain the discrepancy with our findings.

RV remodeling after STEMI has important prognostic implications. Chimed et al. demonstrated that adverse changes in RV structure and function were associated with poorer clinical outcomes.²⁹ Dietz et al. also reported that alterations in RV morphology were associated with prognosis after myocardial infarction.³⁰ In our cohort, structural RV parameters were generally similar between groups. However, adjusted analyses suggested that previous aspirin use was associated with better RV functional performance.

One important observation was the difference between unadjusted and adjusted analyses. Patients receiving aspirin before admission were generally older and had a higher burden of cardiovascular comorbidities, factors that are independently associated with impaired RV function. Adjustment for these variables revealed an association that was not apparent in the univariate analyses. This finding highlights the importance of controlling for confounding factors when evaluating treatment effects in observational

studies.

Clinicians can note that a history of aspirin use, despite denoting higher baseline risk, may be associated with preserved RV systolic function after STEMI. All STEMI patients should receive guideline-based therapy, and RV function should be evaluated equally, as recommended by practice guidelines.³¹

Prospective multicenter studies with larger sample sizes are needed to confirm these findings. Serial assessment of RV function using cardiac magnetic resonance imaging or strain echocardiography may provide a more comprehensive evaluation of ventricular remodeling after STEMI. Studies incorporating biomarkers of thrombosis, inflammation, and microvascular injury may also help clarify the biological mechanisms underlying the observed association.

This study has several limitations. Its cross-sectional observational design does not permit causal inference. Patients were not stratified according to confirmed right ventricular myocardial infarction. Previous aspirin use was based on self-report and could not be independently verified. Echocardiographic assessment was performed only once before hospital discharge, preventing evaluation of temporal changes in RV function. Larger prospective multicenter studies using advanced cardiac imaging are required to validate these findings.

Conclusion

Prior aspirin use was associated with better adjusted RV systolic function in STEMI patients, as reflected by improved TAPSE and RVFAC; although aspirin users more often had hypertension and diabetes, these comorbidities were offset by aspirin’s independent benefit, and prior aspirin use appeared to positively influence acute RV function after STEMI, suggesting that aspirin’s role in altering myocardial injury may benefit the right ventricle—findings that indicate a potential association but require confirmation in prospective studies.

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Author’s Contribution

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Competing Interests

The authors declare no competing interests.

Ethical Approval

The study protocol was approved by the ethics committees of Qazvin University of Medical Sciences and Tehran University of Medical Sciences (IR.QUMS.REC.1402.348). All participants provided written informed consent before enrollment. Patient confidentiality was maintained throughout the study. The investigation was conducted in accordance with the principles of the Declaration of Helsinki.

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