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Augmentation index as a predictor of right ventricular dysfunction in coronary artery disease: a cross-sectional study

Arash Rahimi[1], Babak Geraiely[1], Hamed Vahidi[1], Mohammadreza Eftekhari[1], Ramyar Rahimi Darehbagh[2,3], Somayeh Mohammadi[1], Golrokh Ghaffari[1] and Meysam Khoshavi[1*]

Abstract

Background Arterial stiffness is a well-established predictor of cardiovascular events and mortality. However, its relationship with right ventricular (RV) function in patients with coronary artery disease (CAD) remains unclear. We aimed to investigate the association between aortic augmentation index (AIx), a marker of arterial stiffness, and RV dysfunction in CAD patients.

Methods In this cross-sectional study, 121 patients with stable CAD or acute coronary syndrome who underwent coronary angiography were enrolled. AIx was measured using radial artery applanation tonometry. Comprehensive echocardiography was performed to assess RV function using conventional and speckle-tracking derived parameters. Multivariable linear and logistic regression analyses were used to evaluate the relationship between AIx and RV function, adjusting for potential confounders.

Results Patients with high AIx (> 80%, $n = 53$) had significantly worse RV systolic function compared to those with normal AIx (≤ 80%, $n = 68$), as evidenced by lower tricuspid annular plane systolic excursion (TAPSE), fractional area change (FAC), RV free wall longitudinal strain (RVLS), and RV systolic velocity (RV S') (all $P < 0.05$). AIx correlated negatively with TAPSE (r=-0.421), FAC (r=-0.376), RVLS (r=-0.428), and RV S' (r=-0.355) (all $P < 0.001$), and positively with pulmonary artery systolic pressure ($r = 0.467$, $P < 0.001$) and pulmonary vascular resistance ($r = 0.297$, $P = 0.001$). In multivariable analyses, AIx remained an independent predictor of RV dysfunction (adjusted odds ratio 3.42, 95% confidence interval 1.56–7.51, $P = 0.002$) after adjusting for age, sex, hypertension, diabetes, dyslipidemia, smoking, left ventricular ejection fraction, and Gensini score.

Conclusions Increased aortic stiffness assessed by AIx is independently associated with RV dysfunction in patients with CAD. This association is evident across multiple echocardiographic parameters of RV systolic function and is independent of traditional cardiovascular risk factors, left ventricular systolic function, and the extent of coronary artery disease. Our findings suggest that arterial stiffness may play a role in the development of RV dysfunction in CAD patients and highlight the potential importance of assessing and targeting arterial stiffness in this population.

Keywords Augmentation index, Predictor, Ventricular dysfunction, Coronary artery disease

*Correspondence:
Meysam Khoshavi
meysamkhoshavi@yahoo.com
[1]Department of Cardiology, School of Medicine, Imam Khomeini Hospital Complex, Tehran University of Medical Sciences, Tehran, Iran
[2]Student Research Committee, Kurdistan University of Medical Sciences, Sanandaj, Iran
[3]Universal Scientific Education and Research Network (USERN), Sanandaj, Kurdistan, Iran

Introduction

Coronary artery disease (CAD) is a significant cause of morbidity and mortality worldwide, accounting for approximately one-third of all deaths in individuals over the age of 35 years [1]. Despite advances in prevention, diagnosis, and treatment, CAD remains a significant public health burden with a substantial impact on healthcare systems and society as a whole [2]. While the left ventricle (LV) has been the focus of most research and clinical attention in CAD, there is growing recognition of the importance of the right ventricular (RV) function in determining outcomes in this population [3, 4].

The right ventricle is crucial in maintaining adequate pulmonary perfusion and low systemic venous pressure, which is essential for optimal cardiovascular performance [5]. In the setting of CAD, RV dysfunction can occur due to various mechanisms, including ischemia, infarction, ventricular interdependence, and increased afterload secondary to left-sided heart failure [6]. Importantly, RV dysfunction is a powerful predictor of mortality and adverse cardiovascular events in patients with CAD, independent of LV function [7, 8]. Therefore, identifying factors contributing to RV dysfunction in CAD is of great clinical relevance.

Arterial stiffness, a hallmark of vascular aging, is an essential cardiovascular risk marker and a potential therapeutic target [9]. The aortic augmentation index (AIx), derived from pulse wave analysis, is an indirect measure of arterial stiffness that reflects the contribution of wave reflection to central aortic pressure [10]. Increased AIx has been associated with various cardiovascular risk factors, including hypertension, diabetes, dyslipidemia, and smoking, and has been shown to predict adverse cardiovascular events and mortality in diverse populations [11–13].

The relationship between arterial stiffness and LV function has been extensively studied, with evidence suggesting that increased stiffness contributes to LV remodeling, diastolic dysfunction, and heart failure [14, 15]. However, less is known about the impact of arterial stiffness on RV function, particularly in the context of CAD. Given the prognostic significance of RV dysfunction in CAD, understanding the potential link between arterial stiffness and RV performance could provide valuable insights into risk stratification and management of these patients.

Therefore, the primary objective of this study was to investigate the association between aortic AIx as a measure of arterial stiffness and echocardiographic parameters of RV function in patients with established CAD. We hypothesized that increased AIx would be associated with worse RV function, independent of traditional cardiovascular risk factors and the extent of coronary artery disease. A secondary aim was to explore the relationship between AIx and pulmonary vascular resistance as a potential mediator of RV dysfunction in this population.

To our knowledge, this is the first study to examine the link between AIx and RV function in patients with CAD. The results of this study could have important implications for risk assessment and management of these patients. They may provide a basis for future interventional studies targeting arterial stiffness to improve RV function and clinical outcomes in CAD.

Methods

Study population

This cross-sectional study enrolled 121 patients with stable CAD or acute coronary syndrome (ACS) who underwent coronary angiography at Tehran Heart Center, a tertiary referral center affiliated with Tehran University of Medical Sciences, between January and December 2023. The inclusion criteria were age ≥ 18 years and the presence of at least one epicardial coronary artery with > 50% luminal stenosis on angiography. Patients with acute ST-elevation myocardial infarction, prior coronary artery bypass grafting, severe valvular heart disease, congenital heart disease, pulmonary hypertension, or inadequate echocardiographic image quality were excluded. The institutional ethics committee approved the study protocol, and all participants provided written informed consent.

Data collection

Demographic characteristics, cardiovascular risk factors, and medication history were obtained through patient interviews and medical records review. Hypertension was defined as systolic blood pressure ≥ 140 mmHg, diastolic blood pressure ≥ 90 mmHg, or antihypertensive medications. Diabetes mellitus was defined as fasting blood glucose ≥ 126 mg/dL, glycated hemoglobin (HbA1c) ≥ 6.5%, or the use of glucose-lowering agents. Dyslipidemia was defined as total cholesterol ≥ 200 mg/dL, low-density lipoprotein cholesterol ≥ 130 mg/dL, high-density lipoprotein cholesterol < 40 mg/dL in men or < 50 mg/dL in women, triglycerides ≥ 150 mg/dL, or the use of lipid-lowering medications. Smoking status was classified as current, former, or never smoker.

Arterial stiffness assessment

Aortic augmentation index (AIx) was measured non-invasively using the SphygmoCor system (AtCor Medical, Sydney, Australia), which employs high-fidelity applanation tonometry of the radial artery and pulse wave analysis [16]. A high-fidelity micromanometer-tipped probe was placed on the wrist's radial artery, and 10–15 sequential pressure waveforms were recorded. The central aortic pressure waveform was then derived using a validated generalized transfer function. AIx was calculated

as the difference between the second and first systolic peak pressures, expressed as a percentage of the central pulse pressure. AIx was normalized to a heart rate of 75 beats per minute (AIx@75) to account for the influence of heart rate on the reflection wave. Based on previously established cutoff values, AIx was categorized as usual (≤ 80%) or high (> 80%) [17]. A single trained operator performed all measurements in duplicate, and the mean value was used for analysis.

Echocardiography

Right ventricular (RV) function was assessed using several parameters [18]:

1. Tricuspid annular plane systolic excursion (TAPSE): measured using M-mode echocardiography as the systolic excursion of the lateral tricuspid annulus, with values < 17 mm indicating RV systolic dysfunction.
2. RV fractional area change (FAC): calculated as the percentage change in RV area between end-diastole and end-systole in the apical four-chamber view, with values < 35% indicating RV systolic dysfunction.
3. RV free wall longitudinal strain (RVLS): measured using speckle-tracking echocardiography in the apical four-chamber view. RV systolic dysfunction was defined as RVLS with absolute value < 20% (i.e., values > -20%). All RVLS analyses were performed using absolute values to ensure consistent interpretation of the relationships between variables.
4. Tricuspid lateral annular systolic velocity (RV S'): measured using tissue Doppler imaging, with values < 9.5 cm/s indicating RV systolic dysfunction.

Left ventricular ejection fraction (LVEF) was calculated using the biplane Simpson's method. Left ventricular mass index was calculated and expressed in grams per meter squared (g/m^2) according to standard formulae [19]. Pulmonary artery systolic pressure (PASP) was estimated from the peak tricuspid regurgitation velocity using the simplified Bernoulli equation and adding the estimated proper atrial pressure based on inferior vena cava diameter and collapsibility [20].

Coronary angiography

Coronary angiography was performed using standard techniques via the radial or femoral approach. The severity of CAD was assessed by experienced interventional cardiologists blinded to the echocardiographic and arterial stiffness data. Significant CAD was defined as > 50% luminal narrowing in at least one major epicardial coronary artery. The extent of CAD was categorized as single-vessel, two-vessel, or three-vessel disease based on the number of affected vessels. Left central stenosis > 50% was considered equivalent to two-vessel disease. The Gensini score, a measure of the anatomic severity of CAD, was calculated based on the location and degree of luminal narrowing of coronary lesions [21].

Statistical analysis

Continuous variables are expressed as mean ± standard deviation or median (interquartile range), and categorical variables are presented as frequencies and percentages. Normality was assessed using the Shapiro-Wilk test. Comparisons between groups were made using Student's *t*-test or Mann-Whitney *U* test for continuous variables and chi-square test or Fisher's exact test for categorical variables, as appropriate. The correlation between AIx and other variables was assessed using Pearson's or Spearman's correlation coefficients (r). Multiple linear regression analysis was performed to identify independent predictors of RV function parameters, with AIx as the primary predictor variable and adjusting for age, sex, hypertension, diabetes, dyslipidemia, smoking, LVEF, and Gensini score. *Logistic* regression analysis evaluated the association between high AIx and RV dysfunction. A two-tailed P-value < 0.05 was considered statistically significant. All analyses were performed using SPSS version 26 (IBM Corp., Armonk, NY, USA).

Sample size calculation was based on a previous study [22] that reported a correlation coefficient (r) of 0.33 between AIx and RV function. With a type I error (α) of 0.05 and a power of 80%, a minimum sample size of 71 patients was required. We included 121 patients to account for potential missing data and increase the study's power.

Results

The study population comprised 121 patients with CAD (mean age 64.3 ± 10.8 years, 63% male). Patients were categorized based on their AIx values into normal (≤ 80%, $n = 68$) and high (> 80%, $n = 53$) groups(Table 1).

Patients with high AIx demonstrated significantly lower LVEF (45.6 ± 11.2% vs. 51.3 ± 9.8%, $P = 0.003$) and higher LV mass index (101.4 ± 24.6 g/m^2 vs. 91.3 ± 22.1 g/m^2, $P = 0.021$) compared to those with normal Aix (Table 2). RV function parameters showed significant impairment in the high AIx group, evidenced by:

- Lower TAPSE (18.2 ± 4.1 mm vs. 21.6 ± 3.5 mm, $P < 0.001$).
- Reduced FAC (37.5 ± 7.2% vs. 42.8 ± 6.4%, $P < 0.001$).
- Lower absolute RVLS (19.8 ± 4.9% vs. 23.6 ± 4.2%, $P < 0.001$).
- Decreased RV S' (10.7 ± 2.9 cm/s vs. 12.4 ± 2.6 cm/s, $P = 0.001$).

Table 1 Baseline characteristics of the study population

Variable	Normal AIx (≤ 80%) (*n* = 68)	High AIx (> 80%) (*n* = 53)	*P*-value
Age, years	61.7 ± 10.5	67.6 ± 10.2	0.002
Male, n (%)	44 (64.7)	32 (60.4)	0.619
Body mass index, kg/m^2	27.3 ± 4.2	26.8 ± 3.9	0.503
Systolic blood pressure, mmHg	128 ± 16	138 ± 18	0.002
Diastolic blood pressure, mmHg	76 ± 10	81 ± 11	0.011
Hypertension, n (%)	40 (58.8)	41 (77.4)	0.029
Diabetes, n (%)	22 (32.4)	20 (37.7)	0.536
Dyslipidemia, n (%)	36 (52.9)	38 (71.7)	0.035
Smoking status, n (%)			0.502
- Current smoker	12 (17.6)	8 (15.1)	
- Former smoker	7 (10.3)	4 (7.5)	
- Never smoker	49 (72.1)	41 (77.4)	
Medications			
- ACE inhibitors/ARBs, n (%)	41 (60.3)	37 (69.8)	0.273
- Beta-blockers, n (%)	48 (70.6)	40 (75.5)	0.545
- Calcium channel blockers, n (%)	18 (26.5)	19 (35.8)	0.263
- Statins, n (%)	55 (80.9)	47 (88.7)	0.239

Table 2 Echocardiographic parameters according to AIx

Variable	Normal AIx (≤ 80%) (*n* = 68)	High AIx (> 80%) (*n* = 53)	*P*-value
LVEF, %	51.3 ± 9.8	45.6 ± 11.2	0.003
LV mass index, g/m^2	91.3 ± 22.1	101.4 ± 24.6	0.021
TAPSE, mm	21.6 ± 3.5	18.2 ± 4.1	< 0.001
FAC, %	42.8 ± 6.4	37.5 ± 7.2	< 0.001
RVLS, %	-23.6 ± 4.2	-19.8 ± 4.9	< 0.001
RV S', cm/s	12.4 ± 2.6	10.7 ± 2.9	0.001
PASP, mmHg	28.6 ± 6.8	35.2 ± 8.4	< 0.001
PVR, Wood units	1.72 ± 0.38	1.94 ± 0.49	0.002

Table 3 Coronary angiographic findings according to AIx

Variable	Normal AIx (≤ 80%) (*n* = 68)	High AIx (> 80%) (*n* = 53)	*P*-value
Three-vessel/left main disease, n (%)	27 (39.7)	32 (60.4)	0.023
Gensini score, median (IQR)	36 (24–52)	48 (32–78)	0.001
Acute coronary syndrome, n (%)	22 (32.4)	23 (43.4)	0.208

Coronary Angiographic Findings The prevalence of three-vessel or left central disease was significantly higher in patients with high AIx than those with normal AIx (60.4% vs. 39.7%, $P = 0.023$). The Gensini score, reflecting the anatomic severity of CAD, was also higher in the high AIx group (median 48 [IQR 32–78] vs. 36 [IQR 24–52], $P = 0.001$). There was no significant difference in the prevalence of acute coronary syndrome between the two groups (Table 3).

AIx showed significant correlations with RV function parameters. Using absolute values for RVLS, the following correlations were observed:

Table 4 Multivariable linear regression analysis for predictors of RV function parameters

Variable	TAPSE	FAC	RVLS	RV S'
	β (*P*-value)	β (*P*-value)	β (*P*-value)	β (*P*-value)
AIx	-0.214 (0.006)	-0.186 (0.017)	-0.237 (0.002)	-0.175 (0.028)
Age	-0.152 (0.047)	-0.138 (0.078)	-0.167 (0.029)	-0.129 (0.102)
Male sex	0.092 (0.237)	0.078 (0.316)	0.113 (0.142)	0.086 (0.272)
Hypertension	-0.127 (0.096)	-0.114 (0.137)	-0.141 (0.062)	-0.105 (0.179)
Diabetes	-0.053 (0.501)	-0.046 (0.563)	-0.061 (0.435)	-0.042 (0.594)
Dyslipidemia	-0.163 (0.036)	-0.147 (0.057)	-0.178 (0.020)	-0.139 (0.072)
Smoking	-0.071 (0.363)	-0.063 (0.426)	-0.084 (0.271)	-0.059 (0.459)
LVEF	0.226 (0.003)	0.197 (0.010)	0.249 (0.001)	0.183 (0.017)
Gensini score	-0.184 (0.019)	-0.165 (0.035)	-0.202 (0.009)	-0.151 (0.054)

- TAPSE: r=-0.421, $P < 0.001$.
- FAC: r=-0.376, $P < 0.001$.
- Absolute RVLS: r=-0.428, $P < 0.001$.
- RV S': r=-0.355, $P < 0.001$.

Predictors of RV Function In univariable linear regression analysis, AIx, age, hypertension, dyslipidemia, LVEF, and Gensini score were significantly associated with TAPSE, FAC, RVLS, and RV S' (all $P < 0.05$). In multivariable linear regression analysis, AIx remained an independent predictor of TAPSE (β=-0.214, $P = 0.006$), FAC (β=-0.186, $P = 0.017$), RVLS (β=-0.237, $P = 0.002$), and RV S' (β=-0.175, $P = 0.028$) after adjusting for age, sex, hypertension, diabetes, dyslipidemia, smoking, LVEF, and Gensini score (Table 4).

In logistic regression analysis, AIx > 80% were independently associated with RV dysfunction (defined as TAPSE < 17 mm, FAC < 35%, RVLS less damaging than − 20%, or RV S' <9.5 cm/s) with an odds ratio of 3.42 (95% CI 1.56–7.51, $P = 0.002$) after adjusting for potential confounders (Table 5).

Subgroup Analysis In a subgroup analysis of patients with preserved LVEF (≥ 50%, $n = 74$), AIx remained significantly associated with RV dysfunction (OR 2.84, 95% CI 1.13–7.16, $P = 0.027$), suggesting that the relationship between arterial stiffness and RV function is independent of LV systolic function.

Reproducibility Intraobserver and interobserver variability for AIx measurement were assessed in 20 randomly selected patients. The intraclass correlation coefficients for intraobserver and interobserver variability were 0.96 (95% CI 0.90–0.98) and 0.93 (95% CI 0.83–0.97), respectively, indicating excellent reproducibility.

Table 5 Logistic regression analysis for the association between high AIx and RV dysfunction(RV dysfunction defined as the presence of any of the following: TAPSE < 17 mm, FAC < 35%, absolute RVLS < 20%, or RV S' <9.5 cm/s)

Variable	Odds Ratio	95% CI	*P*-value
AIx > 80%	3.42	1.56–7.51	0.002
Age	1.05	1.01–1.09	0.024
Male sex	1.27	0.56–2.89	0.573
Hypertension	1.93	0.83–4.52	0.129
Diabetes	1.15	0.49–2.68	0.752
Dyslipidemia	2.17	0.94–5.03	0.070
Smoking	0.78	0.32–1.92	0.590
LVEF	0.93	0.89–0.97	0.001
Gensini score	1.02	1.00-1.03	0.044

Discussion

In this cross-sectional study of patients with CAD, we found that increased aortic stiffness, as measured by AIx, was independently associated with RV dysfunction. This association was observed across multiple echocardiographic parameters of RV systolic function, including TAPSE, FAC, RVLS, and RV S'. Moreover, AIx correlated positively with PASP and PVR, suggesting a potential link between arterial stiffness and pulmonary vascular remodeling. These findings highlight the complex interplay between systemic and pulmonary circulations and the potential role of arterial stiffness in the development of RV dysfunction in patients with CAD.

Previous studies have demonstrated that arterial stiffness is a strong predictor of cardiovascular events and mortality in various populations, including those with hypertension, diabetes, and established cardiovascular disease [13, 15, 23]. However, the relationship between arterial stiffness and RV function has been less well characterized, particularly in the context of CAD. Our study extends the existing literature by showing that increased AIx is associated with worse RV systolic function, independent of traditional cardiovascular risk factors, LV systolic function, and the extent of coronary artery disease.

The mechanisms underlying the association between arterial stiffness and RV dysfunction are likely multifactorial. Increased aortic stiffness leads to higher pulsatile afterload on the left ventricle, impairing LV diastolic function and increasing left atrial pressure [24]. This, in turn, may lead to pulmonary venous congestion and increased RV afterload, contributing to RV dysfunction. Additionally, stiff central arteries are associated with more excellent transmission of pulsatile energy into the microvasculature, which can cause endothelial dysfunction and microvascular damage [14]. This may adversely impact pulmonary and coronary circulations, further exacerbating RV dysfunction.

Another potential mechanism linking arterial stiffness to RV dysfunction is ventricular-arterial coupling. In systemic circulation, increased arterial stiffness can lead to ventricular-arterial stiffening and impaired coupling, reducing cardiac efficiency and increasing myocardial oxygen demand [25]. A similar phenomenon may occur in the pulmonary circulation, where increased PVR and pulmonary arterial stiffness can impair RV-pulmonary artery coupling and contribute to RV dysfunction [26]. Our study found significant correlations between AIx PASP and PVR, supporting that systemic arterial stiffness may influence pulmonary vascular hemodynamics.

The finding that AIx was associated with RV dysfunction independent of LV systolic function suggests that the relationship between arterial stiffness and RV performance is not merely a reflection of global cardiac dysfunction. This is consistent with previous studies showing that RV dysfunction can occur without overt LV systolic dysfunction and that RV function is an independent predictor of outcomes in various cardiovascular diseases [27–29]. Our subgroup analysis of patients with preserved LVEF further supports that arterial stiffness may directly impact RV function, independent of LV performance.

The clinical implications of our findings are severalfold. First, given the prognostic significance of RV dysfunction in CAD, our results suggest that assessment of arterial stiffness may help identify patients at higher risk of adverse outcomes. AIx is a simple, noninvasive marker that can be easily measured in the outpatient setting using commercially available devices. Second, interventions targeting arterial stiffness, such as exercise training, dietary modifications, and pharmacological therapies (e.g., renin-angiotensin-aldosterone system inhibitors, statins), may improve RV function in patients with CAD [30–32]. Finally, our findings highlight the importance of considering the right ventricle and pulmonary circulation in managing CAD patients, as they may represent important therapeutic targets.

Several limitations of our study should be acknowledged. First, the cross-sectional design precludes conclusions about causality. Although we hypothesize that increased arterial stiffness contributes to RV dysfunction, the possibility of reverse causation cannot be excluded. Second, while AIx is a well-established marker of arterial stiffness, it is an indirect measure and may be influenced by factors other than intrinsic arterial wall properties, such as heart rate and ventricular ejection dynamics [33]. Third, we did not have data on pulmonary function tests or sleep studies, which could have provided insights into the contribution of lung disease or sleep-disordered breathing to RV dysfunction. Finally, our study was conducted at a single center and included predominantly male patients, which may limit the generalizability of our findings.

Conclusion

This study demonstrates that increased aortic stiffness, measured by AIx, independently associates with RV dysfunction in patients with CAD. This association remains evident across multiple echocardiographic parameters of RV systolic function and persists independently of traditional cardiovascular risk factors, LV systolic function, and coronary artery disease extent. The relationship between AIx and RV dysfunction appears to be mediated through multiple pathways, including:

1. Impaired LV diastolic function leading to elevated left atrial pressure.
2. Subsequent pulmonary venous congestion.
3. Increased RV afterload.
4. Altered ventricular-arterial coupling.

These findings suggest that arterial stiffness plays a significant role in the development of RV dysfunction in CAD patients and highlight the potential importance of assessing and targeting arterial stiffness in this population. Future longitudinal studies should investigate whether interventions to reduce arterial stiffness can improve RV function and clinical outcomes in patients with CAD.

Abbreviations

ACE	Angiotensin-Converting Enzyme
ACS	Acute Coronary Syndrome
AIx	Augmentation Index
ARBs	Angiotensin Receptor Blockers
CAD	Coronary Artery Disease
CI	Confidence Interval
FAC	Fractional Area Change
HbA1c	Glycated Hemoglobin
IQR	Interquartile Range
LV	Left Ventricular
LVEF	Left Ventricular Ejection Fraction
PASP	Pulmonary Artery Systolic Pressure
PVR	Pulmonary Vascular Resistance
RV S'	Right Ventricular Systolic Velocity
RV	Right Ventricular
RVLS	Right Ventricular Free Wall Longitudinal Strain
TAPSE	Tricuspid Annular Plane Systolic Excursion

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Author contributions

A.R. and M.K. conceptualized and designed the study. B.G. and M.K. developed the methodology. A.R., H.V., M.E., and S.M. conducted the investigations and collected the data. R.R.D. performed the statistical analysis and created the visualizations. A.R. wrote the original draft of the manuscript. B.G., H.V., M.E., S.M., and M.K. reviewed and edited the manuscript. G.G. assisted with data curation and investigation. M.K. supervised the project and was responsible for the overall administration. All authors contributed to interpreting the results, critically revising the manuscript for important intellectual content, and approving the final version.

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Data availability

The data supporting this study's findings are available from the corresponding author upon reasonable request.

Declarations

Ethical approval

This study was approved by the institutional ethics committee of Tehran Heart Center, Tehran University of Medical Sciences (Ethical code: IR.TUMS.IKHC.REC.1402.032). All participants provided written informed consent.

Consent for publication

Not Applicable.

Competing interests

The authors declare no competing interests.

Generative AI in scientific writing

While preparing this work, the authors used "Claude 3" AI to check the grammar issues and make the text more narrative. After using this tool, the authors reviewed and edited the content as needed and took full responsibility for the publication's content.

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