Original Article

Assessing Right Atrial Function With 2D Speckle Tracking Echocardiography in Inferior ST-Elevation Myocardial Infarction Patients Undergoing Primary Percutaneous Coronary Intervention

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ABSTRACT

Background: Right atrium (RA) plays a crucial role in cardiac function, yet evaluation of RA function in myocardial infarction (MI) has been underexplored. Inferior ST segment myocardial infarction (STEMI) may affect RA function. Two-dimensional speckle tracking echocardiography (2D-STE) provides a novel, non-invasive means of assessing RA mechanics.

Aim: To evaluate and compare RA function in patients with inferior STEMI, with and without RV infarction, against healthy controls using 2D-STE.

Patients and Methods: This prospective, observational, case-control study included 150 participants: 100 patients with inferior STEMI undergoing primary percutaneous coronary intervention (PCI) and 50 age-matched healthy controls. STEMI group was further divided into those with RV infarction (n = 26) and those without (n = 74). RA function was assessed using 2D-STE, with RA strain parameters measured in reservoir (RASr), conduit (RAScd), and contraction (RASct) phases.

Results: Compared to controls, patients with inferior STEMI exhibited significantly reduced RASr (33.54 ± 5.98% vs. 42.42 ± 5.85%, $p < 0.001$) and RAScd (22.48 ± 5.16% vs. 25.92 ± 4.21%, $p < 0.001$), while RASct remained comparable ($p = 0.062$). Within the STEMI group, RA strain parameters did not significantly differ between those with and without RV infarction ($p > 0.05$). However, RA emptying fraction (RAEF) was significantly lower in RV infarction patients (56.12 ± 10.58% vs. 60.26 ± 7.18%, $p = 0.029$).

Conclusion: RA reservoir and conduit functions are significantly impaired in inferior STEMI patients, irrespective of RV infarction. However, RAEF is reduced in patients with RV infarction, correlating with impaired RV function.

Key Words: Inferior STEMI, myocardial infarction, right atrium, right ventricular infarction, speckle tracking echocardiography.

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INTRODUCTION

While RA plays a crucial role in cardiac function, its significance has often been overlooked in research. However, emerging evidence suggests that RA function can be impaired in early stages of various heart diseases[1]. RA performs three essential functions: it serves as a reservoir for blood during RV contraction, facilitates direct passage of blood from veins to RV, and actively contributes to ventricular filling by contracting during final phase of cardiac cycle[2]. Consequently, RA is vital for maintaining adequate RV output.

Although the impact of MI on RA function remains incompletely understood, studies have established a link between left ventricular MI and impaired left atrial function[3, 4]. This suggests a potential parallel between right ventricular myocardial infarction (RVMI) and RA dysfunction. Fluctuations in RA pressure in the setting of inferior wall MI have been recognized for many years[5].

In inferior STEMI complicated by RV infarction, both systolic and diastolic RV functions are compromised, leading to elevated filling pressures. This altered RV function and increased filling pressure inevitably affect RA dynamics, suggesting potential changes in RA function[6]. 2D-STE provides a practical and non-invasive approach for assessing RA function[7].

This study aimed to evaluate and compare RA function between healthy individuals with no history of cardiac events or cardiovascular risk factors and patients with inferior STEMI, with and without RV involvement.

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PATIENTS AND METHODS

This prospective, observational, case-control study investigated a cohort of 150 participants. The study involved a case group comprising 100 patients experiencing inferior STEMI and seeking emergency care, and a control group comprising 50 healthy individuals matched for age. All STEMI patients underwent primary PCI to treat single-vessel disease. Furthermore, the STEMI group was further categorized into two subgroups: those with evidence of RV infarction ($n = 26$) and those without ($n = 74$). In the setting of inferior STEMI, RV infarction is suspected when there is hypotension associated with clear lungs and elevated jugular venous pressure. Also, the presence of ST segments elevation in right chest leads (V3R to V6R) along with STE in lead II > III is considered highly suggestive of RV involvement during inferior MI. Lastly, RV infarction was confirmed by the presence of RV dilatation and reduced RV systolic function as measured by tricuspid annular plane systolic excursion (TAPSE) and fractional area change (FAC) during echocardiography.

The control group had no prior history of acute coronary syndrome or cardiovascular risk factors. All participants underwent assessment, including demographic data collection, risk factor evaluation, physical examinations (general and local), laboratory tests, electrocardiograph (ECG), and comprehensive echocardiography, incorporating 2DSTE of RA.

ETHICS COMMITTEE

Ain Shams University ethics committee granted ethical approval before commencing the research [FMASU MS 160/2023], and all participants provided informed written consent, ensuring privacy and confidentiality. Data collection spanned from October 2022 to July 2023.

Eligibility criteria

The study included adult patients aged 18 or older who experienced inferior STEMI and subsequently underwent primary PCI. Exclusion criteria included patients not managed with primary PCI if their symptoms began more than 24 hours before presentation, or if they had a history of heart failure, a previous acute coronary syndrome, prior PCI or coronary artery bypass grafting (CABG) surgery, chronic lung disease, pre-existing right-sided heart disease, more than mild left-sided valvular stenosis or regurgitation, or non-sinus rhythm.

This clinical investigation adhered to the established guidelines, regulations, and policies of Ain Shams University. Before commencement of any study-related procedures and before study enrollment, all participants or their legal guardians provided written informed consent. A comprehensive evaluation was conducted on each participant at study's initiation. This assessment included a thorough medical history, demographic data (age and gender), and a review of cardiovascular risk factors such as smoking, hypertension, and diabetes.

Conventional 2D Echocardiography

All participants underwent a two-dimensional transthoracic echocardiogram within 48 hours of the infarction event. The examination was conducted by a skilled cardiologist, with the patient lying supine. A GE Vivid E95 ultrasound system (General Electric) provided with an M5Sc-D (1.4–4.6 MHz) transducer was utilized, with ECG gating employed for image acquisition.

Left ventricle (LV) was evaluated by measuring internal dimensions and volumes at both end-systole and end-diastole. Simpson's biplane rule was then applied to calculate left ventricular ejection fraction (LVEF)[8].

RV diameter and function assessment

Mid RV Diameter

In the apical four-chamber (A4C) echocardiographic view, measurement of RV mid-cavity diameter is taken at midpoint of RV cavity at level of LV papillary muscles[9].

Right Ventricular Systolic Pressure (RVSP)

RVSP was calculated by measuring peak velocity of tricuspid regurgitation, which reflects pressure gradient between RV and RA. The simplified Bernoulli equation (Pressure Gradient = 4 × [peak tricuspid regurgitation velocity]^2) determined pressure gradient. Subsequently, RVSP was derived by adding estimated right atrial pressure to calculated pressure gradient. Right atria pressure (RAP) was estimated based on respiratory variation and diameter of inferior vena cava observed in subcostal view[9].

Tricuspid Annular Plane Systolic Excursion (TAPSE)

RV systolic function was assessed utilizing M-mode Echocardiography in A4C view. The M-mode cursor was positioned across lateral tricuspid annulus, and total tricuspid annular displacement was measured from end-systole to end-diastole. A measurement below 17 millimeters was considered indicative of impaired RV systolic function[9].

2D STE

2DSTE was performed using same transducer employed for standard 2D imaging. Image acquisition occurred during periods of breath-holding with consistent ECG synchronization. The acquired data was saved and processed using ECHOPAC Dimension offline workstation software (version 12.0, General Electric Medical Systems GmbH, Germany). For each participant, three consecutive

cardiac cycles at end-expiration were captured from an A4C view optimized for RV visualization for 2DSTE analysis.

The analysis of the RA with 2DSTE software began with the identification of three anatomical landmarks: the medial and lateral tricuspid annuli and the middle of the RA roof. A three-click technique was used during end-diastole to delineate these markers. The program autonomously delineated the endocardial and epicardial boundaries of the right atrium, partitioning it into six equal portions. Manual modifications were implemented on traced lines to guarantee precise demarcation of RA's borders. Upon clicking the "Compute" button, global strain and strain rate curves were produced. The peak of the ECG R-wave functioned as the zero reference point for these curves. The resultant strain curve often displays a systolic apex, an early diastolic plateau, and a late diastolic trough. RASr was determined by subtracting the late diastolic nadir from the systolic peak. RAScd was calculated as the difference between the systolic peak and the early diastolic plateau. RASct was ultimately defined as the difference between the early diastolic peak and the late diastolic nadir[8].

RA emptying volume (EV) was estimated as difference between RA maximum volume and RA minimum volume. RA emptying fraction (EF) was then determined by expressing RA EV as a proportion of RA maximum volume[10].

Primary PCI

The data collected on infarct-related artery encompassed identification of culprit's vessel, degree of vessel occlusion (complete or partial), revascularization technique used (percutaneous transluminal coronary angioplasty [PTCA] or direct stenting), type of stent employed (bare metal or drug-eluting), number of stents implanted, administration of intracoronary glycoprotein IIb/IIIa inhibitors, thrombolysis in myocardial infarction (TIMI) flow grade achieved following procedure, and myocardial blush grade (MBG)[6].

Statistical analysis

Data management and statistical analysis were performed using SPSS version 27 (IBM, Armonk, New York, United States). Quantitative data were assessed for normality using the Shapiro-Wilk test and direct visualization methods. Consistent with conventional practice, quantitative data were characterized using means and standard deviations or medians and interquartile ranges (IQR). Categorical data were expressed as integer values and percentages. Quantitative data were evaluated by comparing the groups using the Independent t-Test for parametric variables and the Mann–Whitney U Test for non-parametric variables. Categorical data were examined with the Chi-square test or Fisher's exact test. Correlations were performed using Pearson's and Spearman's methodologies. All statistical tests were two-tailed. *P-values* less than 0.05 were considered significant.

RESULTS

Patients with inferior STEMI did not significantly differ from controls in terms of age ($P = 0.309$) or gender ($P = 0.618$). However, patients with inferior STEMI had significantly higher rates of dyslipidemia (61.0% vs. 0.0%, $P < 0.001$), smoking (60.0% vs. 42.0%, $P = 0.037$), DM (42.0% vs. 0.0%, $P < 0.001$), and HTN (25.0% vs. 2.0%, $P < 0.001$) compared to controls. Moreover, SBP (112.30 ± 18.52 vs. 126.60 ± 10.95 mmHg, $P < 0.001$) and DBP (70.60 ± 10.23 vs. 77.20 ± 9.04 mmHg, $P < 0.001$) were significantly lower in STEMI patients than in controls. Heart rate (HR) did not significantly differ between the groups ($P = 0.628$). (Table 1)

Table 1: Baseline characteristics of patients with inferior STEMI and controls.

		Patients (*n* =100)	Controls (*n* =50)	*P-value*
Age	Mean ± SD	51.82 ± 9.09	50.36 ± 6.22	0.309
Gender				
Female	*n* (%)	15 (15.0%)	6 (12.0%)	0.618
Male	*n* (%)	85 (85.0%)	44 (88.0%)	
Dyslipidemia	*n* (%)	61 (61.0%)	0 (0.0%)	<0.001*
Smoking	*n* (%)	60 (60.0%)	21 (42.0%)	0.037*
DM	*n* (%)	42 (42.0%)	0 (0.0%)	<0.001*
HTN	*n* (%)	25 (25.0%)	1 (2.0%)	<0.001*
SBP (mmHg)	Mean ± SD	112.30 ± 18.52	126.60 ± 10.95	<0.001*
DBP (mmHg)	Mean ± SD	70.60 ± 10.23	77.20 ± 9.04	<0.001*
HR (bpm)	Mean ± SD	74.63 ± 13.51	75.64 ± 8.16	0.628

n: number; SD: standard deviation; DM: diabetes mellitus; HTN: hypertension; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; bpm: beats per minute; *: significant *P-value*.

Procedural and angiographic characteristics of patients with inferior STEMI were shown in (Table 2).

Table 2: Procedural and angiographic characteristics of patients with inferior STEMI.

		n = 100
Pain to Door (Hours)	Median (IQR)	10 (4 – 15)
Number of DES	Median (IQR)	1 (1 – 2)
RV Infarction	*n* (%)	26 (26.0%)
Culprit		
RCA	*n* (%)	62 (62.0%)
LCX	*n* (%)	38 (38.0%)
Extent of occlusion		
Subtotal	*n* (%)	30 (30.0%)
Total	*n* (%)	70 (70.0%)
PTCA	*n* (%)	77 (77.0%)
IC Tirofiban	*n* (%)	35 (35.0%)
TIMI Flow		
TIMI I	*n* (%)	0(0.0%)
TIMI II	*n* (%)	18 (18.0%)
TIMI III	*n* (%)	82 (82.0%)
MBG		
MBG I	*n* (%)	0(0.0%)
MBG II	*n* (%)	35 (35.0%)
MBG III	*n* (%)	65 (65.0%)

n: number; IQR: interquartile range; DES: drug-eluting stent; RV: right ventricle; RCA: right coronary artery; LCX: left circumflex artery; PTCA: percutaneous transluminal coronary angioplasty; IC: intracoronary; TIMI: thrombolysis in myocardial infarction; MBG: myocardial blush grade.

Patients with inferior STEMI exhibited significantly larger LVESD compared to controls (31.67 ± 2.91 vs. 29.54 ± 4.41 mm, $P = 0.001$). Additionally, EF was significantly lower in STEMI patients (48.72 ± 4.36% vs. 58.14 ± 4.18%, $P < 0.001$). Right ventricular function was also affected, with significantly reduced TAPSE in STEMI patients (19.91 ± 2.76 vs. 23.72 ± 4.03 mm, $P < 0.001$) and elevated right ventricular systolic pressure (RVSP) (27.89 ± 4.77 vs. 19.64 ± 1.83 mmHg, $P < 0.001$).

Moreover, mid-right ventricular diameter was significantly larger in STEMI patients than in controls (29.09 ± 1.98 vs. 27.98 ± 1.86 mm, $P < 0.001$). Right atrial strain parameters, including reservoir strain (RASr: 33.54 ± 5.98 vs. 42.42 ± 5.85%, $P < 0.001$) and conduit strain (RAScd: 22.48 ± 5.16 vs. 25.92 ± 4.21%, $P < 0.001$), were significantly lower in STEMI patients, whereas contractile strain (RASct) did not significantly differ between the groups ($P = 0.062$).

No significant differences were observed in left ventricular end diastolic diameter (LVEDD) ($P = 0.833$), RV FAC ($P = 0.187$), RA EF ($P = 0.095$), or RA EV ($P = 0.062$). (Table 3).

Table 3: Echocardiographic parameters in patients with inferior STEMI and controls.

	Patients (*n* =100)	Controls (*n* =50)	*P-value*
LVEDD (mm)	49.70 ± 3.39	49.56 ± 4.61	0.833
LVESD (mm)	31.67 ± 2.91	29.54 ± 4.41	0.001*
EF By Simpson (%)	48.72 ± 4.36	58.14 ± 4.18	<0.001*
RV FAC (%)	38.74 ± 2.85	39.62 ± 5.28	0.187
TAPSE (mm)	19.91 ± 2.76	23.72 ± 4.03	<0.001*
RVSP (mmHg)	27.89 ± 4.77	19.64 ± 1.83	<0.001*
Mid RV Diameter (mm)	29.09 ± 1.98	27.98 ± 1.86	<0.001*
RASr (%)	33.54 ± 5.98	42.42 ± 5.85	<0.001*
RAScd (%)	22.48 ± 5.16	25.92 ± 4.21	<0.001*
RASct (%)	13.12 ± 2.48	13.90 ± 2.22	0.062
RA EF (%)	59.18 ± 8.34	61.52 ± 7.42	0.095
RA EV (ml)	21.83 ± 4.48	20.26 ± 4.86	0.062

n: number; SD: standard deviation; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; EF: ejection fraction; RV FAC: right ventricular fractional area change; TAPSE: tricuspid annular plane systolic excursion; RVSP: right ventricular systolic pressure; RASr: right atrial reservoir strain; RAScd: right atrial conduit strain; RASct: right atrial contractile strain; RA EF: right atrial emptying fraction; RA EV: right atrial emptying volume; *: significant *P-value*.

The patients with inferior STEMI (n = 100) were divided into two groups based on the presence or absence of RV infarction: those with RV infarction (n = 26) and those without RV infarction (n = 74).

Patients with inferior STEMI and RV infarction did not significantly differ from those without RV infarction in terms of age (P = 0.051) or gender distribution (P = 0.949). However, dyslipidemia was significantly more prevalent in patients without RV infarction compared to those with RV infarction (80.8% vs. 54.1%, P = 0.016), whereas hypertension was more frequent in the RV infarction group (31.1% vs. 7.7%, P = 0.018). No significant differences were found in smoking (P = 0.691) or diabetes mellitus (P= 0.971).

Patients with RV infarction exhibited significantly worse RV function, as indicated by lower RV fractional area change (RV FAC: 36.38 ± 2.90% vs. 39.57 ± 2.34%, P < 0.001) and tricuspid annular plane systolic excursion (TAPSE: 17.85 ± 2.39 vs. 20.64 ± 2.51 mm, P < 0.001). Additionally, these patients had significantly higher RV systolic pressure (RVSP: 30.15 ± 4.70 vs. 27.09 ± 4.56 mmHg, P = 0.004) and a larger mid-RV diameter (30.38 ±1.63 vs. 28.64 ± 1.89 mm, P < 0.001).

Right atrial strain parameters (RASr, RAScd, RASct) did not significantly differ between groups (P > 0.05). However, patients with RV infarction had significantly lower right atrial emptying fraction (RA EF: 56.12 ± 10.58% vs. 60.26 ± 7.18%, P = 0.029) and right atrial emptying volume (RA EV: 20.27 ± 3.50 vs. 22.37 ± 4.67 ml, P = 0.038). (Table 4)

Table 4: Clinical and echocardiographic parameters between inferior STEMI patients with and without RV infarction.

		RV Infarction		
		Yes (*n* = 74)	No (*n* = 26)	*P-value*
Age	Mean ±SD	50.77 ± 9.36	54.81 ± 7.68	0.051
Gender				
Female	*n* (%)	11 (14.9%)	4 (15.4%)	0.949
Male	*n* (%)	63 (85.1%)	22 (84.6%)	
Dyslipidemia	*n* (%)	40 (54.1%)	21 (80.8%)	0.016*
Smoking	*n* (%)	51 (68.9%)	19 (73.1%)	0.691
DM	*n* (%)	31 (41.9%)	11 (42.3%)	0.971
HTN	*n* (%)	23 (31.1%)	2 (7.7%)	0.018*
RV FAC (%)	Mean ±SD	39.57 ± 2.34	36.38 ± 2.90	<0.001*
TAPSE (mm)	Mean ±SD	20.64 ± 2.51	17.85 ± 2.39	<0.001*
RVSP (mmHg)	Mean ±SD	27.09 ± 4.56	30.15 ± 4.70	0.004*
Mid RV Diameter (mm)	Mean ±SD	28.64 ± 1.89	30.38 ± 1.63	<0.001*
RASr (%)	Mean ±SD	34.00 ± 5.67	32.23 ± 6.74	0.196
RAScd (%)	Mean ±SD	22.96 ± 5.05	21.12 ± 5.33	0.118
RASct (%)	Mean ±SD	13.28 ± 2.60	12.65 ± 2.04	0.266
RA EF (%)	Mean ±SD	60.26 ± 7.18	56.12 ± 10.58	0.029*
RA EV (ml)	Mean ±SD	22.37 ± 4.67	20.27 ± 3.50	0.038*

n: number, SD: standard deviation, DM: diabetes mellitus, HTN: hypertension, RV FAC: right ventricular fractional area change, TAPSE: tricuspid annular plane systolic excursion, RVSP: right ventricular systolic pressure, RASr: right atrial reservoir strain, RAScd: right atrial conduit strain, RASct: right atrial contractile strain, RA EF: right atrial emptying fraction, RA EV: right atrial emptying volume, *: significant *P-value*.

RA EF showed significant positive correlations with RV FAC (r = 0.425, $P < 0.001$), TAPSE (r = 0.377, $P<0.001$), and LVEF (EF by Simpson) (r = 0.305, $P=0.002$). In contrast, RA EF exhibited a significant negative correlation with LVEDD (r = -0.231, $P = 0.021$) and mid-right ventricular diameter (r = -0.289, $P = 0.004$). RA EV demonstrated significant positive correlations with RV FAC (r = 0.378, $P< 0.001$) and TAPSE (r = 0.239, $P=0.017$), while it showed a significant negative correlation with mid-RV diameter (r= -0.219, $P = 0.028$). Additionally, RA EV was inversely correlated with heart rate (r = -0.256, $P = 0.010$). RAScd was significantly negatively correlated with RVSP (r =-0.301, $P = 0.002$). RASct showed a significant negative correlation with LVEDD (r = -0.341, $P = 0.001$).

No significant correlations were observed between RASr and any of the analyzed clinical or echocardiographic parameters ($P > 0.05$). Similarly, RAScd and RASct did not show significant correlations with age, blood pressure, or most echocardiographic measures. (Table 5)

Table 5: Correlation between right atrial function parameters and clinical and echocardiographic variables in inferior STEMI patients.

	RA Sr		RA Scd		RA Sct		RA EF		RA EV	
	R	*P-value*	r	*P-value*	r	*P-value*	R	*P-value*	r	*P-value*
Age (Years)	-0.048	0.639	0.069	0.495	-0.111	0.269	0.092	0.363	0.121	0.23
SBP (mmHg)	0.111	0.273	0.03	0.771	0.087	0.388	0.039	0.702	0.014	0.893
DBP (mmHg)	-0.011	0.915	0.06	0.556	-0.025	0.807	-0.045	0.656	-0.186	0.064
HR (bpm)	0.072	0.476	0.102	0.312	-0.089	0.379	-0.134	0.185	-0.256	0.01*
Pain to Door (Hours)	-0.127	0.494	-0.242	0.015*	0.108	0.284	-0.097	0.339	0.025	0.806
RV FAC (%)	0.077	0.444	0.069	0.497	0.051	0.616	0.425	<0.001*	0.378	<0.001*
TAPSE (mm)	0.311	0.002*	0.36	<0.001*	0.104	0.304	0.377	<0.001*	0.239	0.017*
RVSP (mmHg)	0.129	0.2	-0.301	0.002*	0.536	<0.001*	0.047	0.642	0.167	0.097
LVEDD (mm)	-0.212	0.034*	0.024	0.815	-0.341	0.001*	-0.231	0.021*	-0.087	0.39
LVESD (mm)	-0.122	0.227	-0.137	0.173	-0.161	0.109	-0.184	0.067	-0.041	0.687
EF By Simpson (%)	0.051	0.613	-0.066	0.512	0.261	0.009*	0.305	0.002*	0.17	0.09
Mid RV Diameter (mm)	-0.14	0.165	0.126	0.211	-0.338	0.001*	-0.289	0.004*	-0.219	0.028*

RA Sr: right atrial reservoir strain; RA Scd: right atrial conduit strain; RA Sct: right atrial contractile strain; RA EF: right atrial emptying fraction; RA EV: right atrial emptying volume; SBP: systolic blood pressure; DBP: diastolic blood pressure; HR: heart rate; bpm: beats per minute; RV FAC: right ventricular fractional area change; TAPSE: tricuspid annular plane systolic excursion; RVSP: right ventricular systolic pressure; LVEDD: left ventricular end-diastolic diameter; LVESD: left ventricular end-systolic diameter; EF: ejection fraction; *: significant *P-value*.

DISCUSSION

RA plays a crucial role in RV performance through reservoir, conduit, and contraction functions, ensuring adequate cardiac output. However, its alterations in MI remain unclear[10]. This study, conducted at Ain Shams University Hospitals, assessed RA function using 2D-STE in 100 patients with inferior MI (26 with RV infarction and 74 without) and compared findings with healthy controls free of cardiovascular risk factors.

Analysis of RA strain parameters revealed significant differences between the groups. The reservoir phase strain was notably reduced in patients with inferior STEMI compared to healthy controls. Similarly, the conduit phase strain showed a notable decline in the patient group. However, the contractile phase strain remained relatively preserved, with no notable variation between patients and controls.

The selective impairment of RA reservoir and conduit functions is physiologically consistent with the pathophysiologic cascade following inferior MI, particularly in the presence of RV involvement. The RA reservoir phase, which occurs during RV systole, and the conduit phase, occurring during early RV diastole, are both highly dependent on RV compliance and pressure dynamics. RV ischemia, a common complication of inferior MI due to shared perfusion territories via the right coronary artery, can lead to elevated RV filling pressures and impaired relaxation. These changes result in increased RA afterload and diminished RA compliance, compromising both reservoir and conduit performance. In contrast, RA contractile function, which represents active atrial systole during late diastole, is primarily governed by intrinsic atrial contractility and the integrity of atrial electrical conduction. In the absence of direct atrial ischemia or atrial arrhythmias, this component of RA function may remain intact despite hemodynamic disturbances.

Our results corroborate the findings of Nilda and colleagues, who utilized 2DSTE to examine the effect of LV inferior wall MI on RA function. Their study, which included 30 patients with LV inferior MI and 30 controls, revealed a marked decrease in RASr and RAScd in the MI group compared to the controls. Specifically, RASr averaged 31.5% in the inferior STEMI group versus 56.2% in the control group ($P < 0.01$), and RAScd averaged 12.5% versus 35% ($P = 0.01$), respectively. Conversely, RASct remained similar between the inferior STEMI and control groups (20.17% vs. 24.4%, $P = 0.07$)[10].

Our research found no significant difference in RA EF between patients with inferior STEMI and controls. Similarly, RA EV remained comparable between the two groups. These findings were contrasted with those of ***Mokhtar et al.***, who also observed preserved RA EF in patients with anterior STEMI compared to controls (55 ± 7 vs. 54 ± 9, $P = 0.494$). However, ***Mokhtar et al.*** reported a decrease in RA EV in the anterior STEMI group (17 [12-23] vs. 21 [20-29], $P = 0.001$), a key distinction being their focus on anterior rather than inferior STEMI[11].

A prior investigation by ***Yonghuai et al.***[12] assessed RA phasic function in individuals with coronary slow flow phenomenon (CSFP). Their study, which compared 82 persons with CSFP to a control group, revealed no significant decrease in either RA EV or RA EF in the CSFP cohort.

A comparison of right atrial function using 2D-STE between patients with and without RVMI showed no significant difference in RASr, RAScd, or RASct. These findings contrast with those of ***Nourian et al.***, who reported impaired RA reservoir and conduit function in patients with both inferior MI and RVMI but found preserved RA contractile function, aligning with our results. However, our study demonstrated a significant reduction in both RA EF and RA EV in patients with RVMI compared to those without, partially consistent with ***Nourian et al.***, who also observed a significant decrease in RA EF but reported a non-significant reduction in RA EV[13].

Two-dimensional echocardiographic assessment revealed notable variations in RV function between patients with and without RV infarction. The RV infarction group exhibited a notable increase in mid-RV diameter, while both FAC and TAPSE were significantly reduced. These findings contrast with those of ***Kanar et al.***, who reported no marked variation in RV diameter but found a statistically significant reduction in TAPSE in patients with RV involvement. Although ***Kanar et al.*** also observed a decrease in FAC in the RV infarction group, this difference did not reach statistical significance[14].

A significant elevation in RVSP was observed in patients with RV infarction compared to those without. This finding contradicts the results of ***Nourian et al.***, who did not report a significant difference in RVSP between these two groups[13].

LIMITATIONS

This research was conducted at a single institution. While cardiac magnetic resonance remains the benchmark for RA assessment, its use was precluded in this acute post-MI setting due to patient instability. Further extensive validation is needed for strain measurements in the left ventricular inferior wall and RV infarction. Several factors limit the clinical application of RA strain, including suboptimal temporal resolution, reliance on high-quality images and a consistent frame rate, potential inaccuracies in delineating myocardial borders in suboptimal images, and the necessity of expertise in using the analysis software.

CONCLUSION

Individuals with inferior wall MI exhibited a significant decrease in RASr and RAScd compared to controls. However, in the two groups RASct, RA EF, and RA EV remained similar. These findings underscore the importance of assessing atrial mechanics as part of the comprehensive evaluation of right heart performance in the setting of inferior MI. Clinically, RA dysfunction may serve as an early and sensitive indicator of RV impairment, with potential utility in guiding fluid management, anticipating hemodynamic instability, and informing prognosis. The integration of RA functional assessment - particularly through speckle-tracking echocardiography or strain imaging - may enhance risk stratification and refine therapeutic strategies in this high-risk population.

DECLARATION OF CONFLICTING INTERESTS

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CONTRIBUTION

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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تقييم وظيفة الأذين الأيمن باستخدام تخطيط صدى القلب بتتبع البقع ثنائي الأبعاد في مرضى احتشاء عضلة القلب السفلي مع إجراء التداخل التاجي الأولي

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المقدمة: يلعب الأذين الأيمن دورًا حاسمًا في وظيفة القلب، إلا أن أهميته في احتشاء عضلة القلب لم تحظَ بالاهتمام الكافي. قد يؤثر احتشاء عضلة القلب السفلي، خاصة عند تعقيده باحتشاء البطين الأيمن، على وظيفة الأذين الأيمن. يوفر تخطيط صدى القلب بتتبع البقع ثنائي الأبعاد وسيلة مبتكرة وغير جراحية لتقييم ميكانيكا الأذين الأيمن في هذا السياق.

هدف الدراسة: تقييم وظيفة الأذين الأيمن لدى مرضى احتشاء عضلة القلب السفلي، مع أو بدون احتشاء البطين الأيمن، ومقارنتها بالأصحاء باستخدام تخطيط صدى القلب بتتبع البقع ثنائي الأبعاد.

المرضى والطرق: شملت هذه الدراسة المستقبلية الرصدية للحالات والشواهد مئة وخمسين مشاركًا: مئة مريض مصاب باحتشاء عضلة القلب السفلي خضعوا لتداخل تاجي أولي، وخمسين فردًا أصحاء متوافقين في العمر. تم تقسيم مجموعة المرضى إلى مجموعتين فرعيتين: مرضى يعانون من احتشاء البطين الأيمن، ومرضى بدون احتشاء البطين الأيمن. تم تقييم وظيفة الأذين الأيمن باستخدام تخطيط صدى القلب بتتبع البقع ثنائي الأبعاد، حيث تم قياس مؤشرات الإجهاد الأذيني في مراحل التخزين والنقل والانقباض.

النتائج: أظهر مرضى احتشاء عضلة القلب السفلي انخفاضًا ملحوظًا في كل من إجهاد التخزين وإجهاد النقل مقارنةً بالأفراد الأصحاء، بينما ظل إجهاد الانقباض مشابهًا. لم تظهر فروق ذات دلالة إحصائية في مؤشرات الإجهاد الأذيني بين المرضى الذين يعانون من احتشاء البطين الأيمن وأولئك الذين لا يعانون منه. ومع ذلك، كان معدل القذف الأذيني أقل بشكل ملحوظ في المرضى الذين لديهم احتشاء في البطين الأيمن، مما يعكس تأثر وظيفة البطين الأيمن.

الاستنتاج: تتأثر وظائف التخزين والنقل في الأذين الأيمن بشكل ملحوظ لدى مرضى احتشاء عضلة القلب السفلي، بغض النظر عن وجود احتشاء البطين الأيمن. ومع ذلك، فإن معدل القذف الأذيني يكون منخفضًا لدى المرضى الذين يعانون من احتشاء البطين الأيمن، مما يرتبط بضعف وظيفة البطين الأيمن. يعد تخطيط صدى القلب بتتبع البقع ثنائي الأبعاد أداة قيّمة لفهم ميكانيكا الأذين الأيمن في هذه الفئة من المرضى.