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Speckle Tracking Analysis of Mitral Annular Calcification and Left Ventricular Mechanics

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Abstract

BACKGROUND: Clinical echocardiography often reveals mitral annular calcification (MAC), and this condition has been connected to atherosclerosis and cardiovascular risk factors. To this day, our knowledge of the left ventricle's (LV) physiology is still somewhat restricted. Using 2D speckle tracking echocardiography, we analyzed the connection between MAC and LV mechanical functions (2DSTE).

AIM: The purpose of the current investigation is to assess how left ventricular mechanics are impacted by MAC.

MATERIALS AND METHODS: Between April 2020 and October 2021, this prospective and cross-sectional study was carried out in Islamic Center of Cardiology Al Azhar University and Cardiology Department of Al Hussien University Hospital. Eighty participants were utilized in the study. When necessary, patients were referred to the Cardiology Clinics at the Medical Schools of Al Azhar University for follow-up care. Sixty MAC patients were studied with 20 healthy controls of similar age and gender. All patients had a 2D echocardiogram. Sixty consecutive patients with a MAC diagnosis and 20 age- and sex-matched controls with normal cardiac evaluations made up the study population. The patients were all given a 2D echocardiogram.

RESULTS: The longitudinal strain significantly decreased as the MAC severity rose. The LV's rotating mechanics varied greatly among MAC organizations. The E/A ratio, the global longitudinal speed (GLS), and the systolic tissue velocity were all shown to have a negative correlation with MAC thickness. Additional variables were found to have statistically significant connections. There were significant correlations between MAC severity and age, LV mass index, and t-LV UR. Age, LV mass index, and linear speed were significantly related to MAC severity.

CONCLUSION: The LV ability to contract systolically and diastolically is compromised when MAC is both present and severe. The LV ability to contract systolically and diastolically is likewise impacted by an increase in MAC severity.

Introduction

The mitral valve annulus, a ring of fibrous tissue in the shape of a saddle, divides the left atrium from the left ventricle (LV). The medical name for calcium building along the annulus, called mitral annular calcification (MAC), often begins on the posterior side and progresses anteriorly or into the posterior leaflet [1].

A defective calcium metabolism, valvular and atherosclerotic degeneration, increased mitral valve stress, inflammation, and MAC are the main causes of chronic and growing calcium deposition at the fibrous annulus of the mitral valve [2].

Where the mitral valve links to the mitral annulus, atherosclerotic lipids form and act as a nidus for the progression of atherosclerosis, leading to the development of MAC [3].

The frequency and clinical significance of MAC are sometimes misinterpreted since it occurs more frequently in clinical practice with an older population and is usually seen incidentally on echocardiography and/or computed tomography. It is well established that a variety of traditional cardiovascular risk factors, including valvular heart disease and systemic atherosclerosis, are linked to MAC [4].

Clinical atherosclerosis and cardiovascular risk factors seem to be linked to MAC. But more than only systemic atherosclerosis is known to be indicated by the presence of MAC. It has also been linked to the chance of dying from all causes and cardiovascular disease, which includes myocardial infarction, non-hemorrhagic stroke, and coronary insufficiency [5]. The risk variables include valvular calcification as a general sign of atherosclerotic burden, common risk factors between MAC and CV outcomes, and mechanical elements of MAC [6].

The left atrium, LV, and mitral valve (LV) complex is significant, active, and interconnected. The mitral annulus is necessary for both effective valve closure and ongoing LV filling. The complex structure and mobility of the mitral annulus excursion have been related to LV function. In addition, even though the

chordae tendineae and mitral leaflets are often intact, calcification may gradually form beneath the posterior leaflet with encroachment on the leaflet and may affect LV systolic functioning [7].

Due to the advent of two-dimensional speckle tracking echocardiography, automated and statistical evaluations of heart mechanics are now within reach (2DSTE). According to this idea, ultrasonic vibrations may cause constructive and destructive interference in tissue, which results in the formation of speckles and, by extension, 2DSTE. Speckle analysis is conducted in real-time, frame by frame, during the whole cardiac cycle. Fewer errors in measurements, a stronger signal-to-noise ratio, and no translation or tethering issues are only a few of the advantages of this approach over conventional echocardiography [8].

The purpose of the current investigation is to assess how left ventricular mechanics are impacted by MAC.

Materials and Methods

This prospective and cross-sectional research was conducted in Islamic Center of Cardiology Al Azhar University and Cardiology Department of Al Hussien University Hospital between April 2020 and October 2021. A total of 80 people took part in the research. Follow-up treatment was provided by the Cardiology Clinics at Al Azhar University.

In this study, 60 patients with MAC were matched with 20 age- and gender-matched controls who had normal cardiac examination (Table 1). Heart problems were one of the list of disqualifiers (such as aortic stenosis, moderate-to-severe aortic or mitral regurgitation, rheumatic valvular disease, heart failure (HF), hypertrophic-dilated cardiomyopathy, arrhythmias, renal dysfunction, asthma, malignancy, and a poor echocardiographic window).

Those who were included in the final sample of participants were considered part of the study population. The local ethics committee green-lit the trial after patients gave their informed consent to participate. An individual's eGFR may be determined with the use of a formula developed for the Modification of Diet in Renal Disease Study (Modification of Diet in Renal Disease). Respondents' BMIs were calculated by dividing their kilograms of body weight by their square meter height (BMI). The following formula, which results in square meters, was used to determine the body's surface area: $0.007184 \times \text{weight (kg)}^{0.425} \times \text{height (cm)}^{0.725}$.

Conventional echocardiography

The standard frequency of echocardiography transducers was used to capture the echoes. The American Society of Echocardiography's guidelines were strictly followed during all operations.

Echo-dense bands were used to diagnose MAC, and they were shown to originate from the posterior mitral valve leaflet, be anterior to, and parallel to, the posterior LV wall during both systole and diastole on parasternal long-axis imaging.

Maximum thickness in millimeters over the whole length of the lesion (from anterior to posterior) was used to quantify the severity of MAC.

The severity of MAC was determined by the thickness of the calcifications: 1–3 mm for mild, 2–4 mm for moderate, and 4–5 mm for severe.

Table 1: Sociodemographic characteristics of mitral annular calcification group and control group

Variable	Normal controls (n = 20)	Patients with MAC (n = 60)	p-value
Age (years)	71.64 ± 15.68	72.86 ± 8.53	0.7408
Gender, n (%)			
Male	4 (20)	11 (18.33)	0.869
Female	16 (80)	49 (81.67)	
HT, n (%)	12 (60)	45 (75)	0.199
DM, n (%)	4 (20)	22 (36.67)	0.168
BSA (m^2)	1.59 ± 0.4	1.7 ± 0.5	0.3217
BMI (kg/m^2)	27.66 ± 2.2	28.67 ± 2.39	0.0859
eGFR>60 (mL/min per 1.73 m^2), n (%)	16 (80)	48 (80)	1
LDL-cholesterol (mg/dL)	125.62 ± 15.06	136.39 ± 18.98	0.0116*
HDL-cholesterol (mg/dL)	40.38 ± 6.21	39.16 ± 6.28	0.4503
Triglycerides (mg/dL)	150.65 ± 15.68	161.73 ± 21.16	0.0148*
Total cholesterol (mg/dL)	186.11 ± 18.29	196.37 ± 16.65	0.0293*
Fast plasma glucose (mg/dL)	97.31 ± 23.07	105.73 ± 25.45	0.1725
Calcium channel blockers (%)	4 (20)	28 (46.67)	0.168
B-blockers (%)	5 (25)	20 (33.33)	0.486
ACE inhibitors or ARB (%)	3 (15)	26 (43.33)	0.022*
Statins (%)	5 (25)	24 (40)	0.227
LV end-diastolic volume (rnL/m^2)	83.76 ± 13.84	86.13 ± 18.11	0.5429
LV end-systolic volume (mL/m^2)	33.4 ± 6.84	35.48 ± 8.35	0.2697
LV ejection fraction (%)	60.98 ± 4.68	60.24 ± 4.81	0.5449
E/A ratio	1.1 ± 0.4	0.88 ± 0.2	0.0206*
Sm (systolic tissue velocity, cm/s)	12.95 ± 1.91	11.46 ± 1.89	0.0033*
E/em ratio	7.52 ± 2.51	8.99 ± 1.91	0.0189*
MPI	0.33 ± 0.04	0.37 ± 0.04	0.0002*
LV mass index (g/m^2)	106.03 ± 6.49	112.35 ± 11.28	0.0029*
LAVI (mL/m^2)	25.58 ± 7.95	30.74 ± 6.48	0.0104*

MAC: Mitral annular calcification, HT: Hypertension; DM: Diabetes mellitus, BSA: Body surface area, BMI: Body mass index, eGFR: Estimated glomerular filtration fraction, LDL: Low-density lipoprotein, HDL: High-density lipoprotein, ACE: Angiotensin-converting enzyme, ARB: Angiotensin receptor blocker, LV: Left ventricle, E: Transmitral Doppler E-wave, A: Transmitral doppler A-wave, Sm: Mitral annular tissue Doppler S-wave, Em: Mitral annular tissue Doppler E-wave, MPI: Myocardial performance index, LAVI: Left atrial volume index.

In this research, we determined LV volumes and ejection fraction percentages using Simpson's method (LVEF). According to the Devereux method, we determined the LV's mass by indexing it to the subject's total body area. Using pulse-wave Doppler, we determined the peak early (E) and late (T) transmitral flow velocities during diastole (A). We measured both the mitral inflow velocity and the LV outflow velocity using Doppler echocardiography.

Pulse-wave using both Doppler and tissue Doppler imaging was utilized to evaluate the systolic and early diastolic velocities of the annular tissue at the lateral annular corner (TDI). Using the E/Em quotient, the LV's filling pressures may be determined. The area-length (L) method may be used to determine the volume of the left atrium during ventricular end-systole from either the apical two-chamber (A2) or apical four-chamber (A1) viewpoint (A2).

Using the calculation (A1 + A2/L for the atrial volume), we get a value of 0.85 for the left atrial volume index (relative to BSA) (LAVI).

Two-dimensional speckle tracking analysis

Several different 2DST perspectives were shown, each of which focused in on a unique aspect of the LV. The number of apical chambers might range from four (A4C) to three (A3C) to two (A2C). During end-systole, SAX was used to capture images of the mitral valve level and the area proximal to the LV luminal obstruction site.

Photos taken with a SAX camera and a circular aspect ratio were very popular. Using 2D strain analysis technologies that are already on the market, we were able to extend our research over the whole myocardium. Finding the endocardial border in the end-systolic frame required manual analysis of the three apical and SAX images. Using the apical and SAX angles, the LV may be divided into 6 equal halves.

By tallying the absolute differences between each pair of frames, we were able to follow the moving elements of the video. A bespoke program was developed to compute the average global longitudinal strain (GLS) over all 17 sections. Researchers were able to determine the vessel's circumferential strain, radial strain, and systolic strain rates by analyzing SAX images captured at the vessel's base and tip.

The average number of revolutions and the speed at which they occurred was calculated automatically for both the bottom and top perspectives. The counterclockwise rotation was recorded as a negative value since it was seen from above the LV.

To get the LV twist and twist velocity, you must know the absolute apex-to-base difference in rotational velocity (in degrees and degrees/s). The left ventricular torsion (LV-tor) was calculated by measuring the degree of twist in the LV in relation to its length in an apical four-chamber image.

Setting all time sequences to the same percentage of diastolic length may help decrease timing differences caused by patients' varied heart rates. Similar to how t-LV UR was calculated, peak LV twist, peak LV twist rate, LV-tor, and LV UR were all calculated as a percentage of end-diastolic time, which was held constant at 100%.

Data management analysis

After data cleaning, the Statistical Package for the social sciences-18 was used for data processing [9]. Kolmogorov–Smirnov test was done to ensure that the data were normally distributed. Descriptive statistics was done for data summarization in the form of frequency and percentage for qualitative variables and means ± SD for quantitative variables. Inferential statistics was done for comparing the participant groups using Chi-square test for qualitative data, ANOVA test, and Student's t test for parametric quantitative data, The Monte Carlo test may be used to adjust for any number of cells with a count < 5. Fischer Chi-square adjustment was applied to tables demonstrating non-continuous data. With $p < 0.05$ considered significant, $p < 0.01$ considered highly significant.

Ethical aspects

1. Written consent of each participant was accustoming to be obtained before collecting data
2. Confidentially of collected data was maintained
3. Ethical approval was obtained from Al-Azhar university's ethical committee. The World Medical Association's Declaration of Helsinki was adhered to as the ethical framework for this study.

Table 2: Quantitative analysis of two-dimensional spectral transform parameters for the control and mitral annular calcification groups

Variable	Normal controls (n = 20)	Patients with MAC (n = 60)	p-value
Strain parameters			
CircSbase (%)	−16.78 ± 4.32	−16.43 ± 3.82	0.7478
RadSbase (%)	47.6 ± 11.48	40.99 ± 11.96	0.0303*
CircSapex (%)	− 24.58 ± 7.58	− 24.38 ± 6.33	0.9156
RadSapex (%)	31.9 ± 11.57	31.39 ± 9.64	0.8595
CircSR-Sbase (s^{-1})	1.13 ± 0.28	1.07 ± 0.15	0.3627
CircSR-Sapex (s^{-1})	1.7 ± 0.35	1.68 ± 0.33	0.823
RadSR-Sbase (s^{-1})	2.31 ± 0.54	2 ± 0.44	0.0228*
RadSR-Sapex (s^{-1})	1.96 ± 0.63	1.85 ± 0.47	0.4754
GLS (%)	− 20.92 ± 1.39	− 19.26 ± 1.79	0.0001*
Rotational parameters			
Ar (°)	11.13 ± 2.29	13.87 ± 2.44	<0.0001*
Ar-rate systole (°/s)	75.22 ± 25.17	91.94 ± 21.09	0.0091*
Br (°)	− 7.75 ± 2.51	− 7.26 ± 2.4	0.447
Br-rate systole (°/s)	65.26 ± 29.22	60.08 ± 15.29	0.4502
Peak LV twist (°)	18.86 ± 3.39	20.82 ± 3.22	0.0262*
Peak LV twist rate (°/s)	103.38 ± 19.28	113.83 ± 19.24	0.0389*
LV-tor (°/cm)	2.59 ± 0.5	2.89 ± 0.5	0.0227*
LV UR (°/s)	106.29 ± 20.68	103.28 ± 24.78	0.594
t-LV UR (%, diastole)	13.35 ± 4.28	19.28 ± 6.87	<0.0001*

MAC: Mitral annular calcification, CircSbase: Basal circumferential strain, RadSbase: Basal radial strain, CircSapex: Apical circumferential strain, RadSapex: Apical radial strain, CircSR-Sbase: Basal systolic circumferential strain rate, CircSR-Sapex: Apical systolic circumferential strain rate, RadSR-Sbase: Basal systolic radial strain rate, RadSR-Sapex: Apical systolic radial strain rate, GLS: Global longitudinal strain, Ar: Peak systolic apical rotation, Ar-rate systole: Peak systolic apical rotation rate, Br: Peak systolic basal rotation, Br-rate systole: Peak systolic basal rotation rate, LV-tor: LV torsion, LV UR: LV untwisting rate, t-LV UR: Time to LV untwisting rate, LV: Left ventricle.

Table 3: The left ventricle basal 6-segment longitudinal strain (mitral annular calcification thickness)

Variable	Mild (n = 19)	Moderate (n = 23)	Severe (n = 18)	f	P1 (mild, moderate)	P2 (moderate, severe)	P3 (mild, severe)
Anteroseptum LS (%)	20.24 ± 1	19.16 ± 1.31	18.35 ± 1.2	< 0.0001	0.0043*	0.95784	0.04483*
Anterolateral LS (%)	20.34 ± 1.39	19.24 ± 1.31	18.66 ± 1.2	< 0.0001	0.01237*	0.98922	0.14587
Anterior LS (%)	20.38 ± 1.2	19.34 ± 1.31	18.61 ± 1.31	< 0.0001	0.01059*	0.9852	0.07981
Inferoseptum LS (%)	20.12 ± 1.2	19.08 ± 1.1	18.69 ± 1	< 0.0001	0.006*	0.98647	0.24053
Inferior LS (%)	20.32 ± 1.29	19.38 ± 1.21	18.79 ± 1.1	0.372923	0.0203*	0.9893	0.10917
Posterolateral LS (%)	20.34 ± 1.1	19.52 ± 1.11	18.66 ± 1.2	0.346923	0.02141*	0.99866	0.0207*

LS: Longitudinal strain, LV: Left ventricle.

Results

The MCA group was shown to have higher baseline values for LDL-cholesterol, triglyceride, total cholesterol, E/Em ratio, MPI, LV mass index, and LAVI than the control group. Systolic tissue velocities and E/A ratios were both considerably reduced in the MCA group as compared to the control group (Figures 1-3).

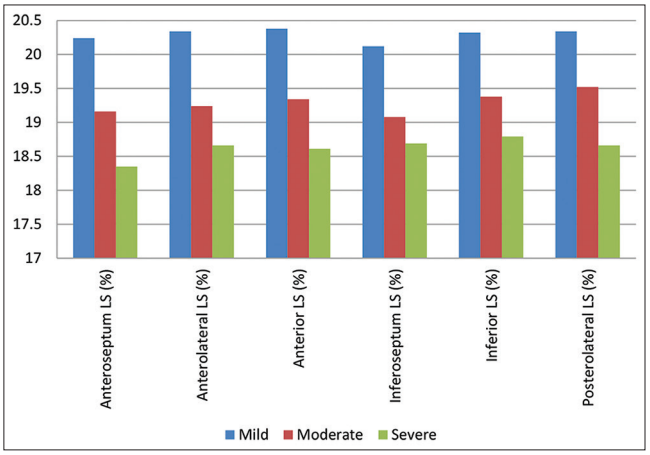


Figure 1: The LV basal 6-segment longitudinal strain (MAC thickness)

Regarding 2DSTE-derived Parameters, RadSbase and RadSR-Sbase were significantly decreased in MCA group compared with controls. GLS, Ar, Ar-rate systole, Peak LV twist, Peak LV twist rate, LV-tor, and t-LV UR were significantly increased in MCA group(Tables 2-4).

With increase MAC severity, longitudinal strain was significantly decreasing.

There was significant differences in LV rotational mechanics among MAC groups.

There was a significant negative correlation between MAC thickness and E/A ratio, systolic tissue velocity, and GLS. Other parameters showed a significant positive correlation(Table 5).

There was a significant association between age, LV mass index, and t-LV UR with MAC severity (Table 6).

There was a significant association between age, LV mass index, and GLS with MAC severity(Table 7).

Discussion

During an echocardiography, MAC is a common finding and its frequency increases with age [10] An earlier study showed that individuals with MAC had an increased risk of long-term CV events, strokes, HF, and overall mortality compared to those without MAC [11]. According to another study, diabetic patients with MAC showed poorer LV systolic performance than non-diabetic patients [12].

In addition, another research discovered that in those with chronic renal failure, the severity of MAC was connected to a drop in LVEF [13]. The development of HF may be delayed or prevented if preclinical LV systolic failure is promptly diagnosed medically [14].

Systolic failure may first manifest in this direction because to the longitudinal orientation of subendocardial fibers, which are more prone to myocardial ischemia and fibrosis [15]. Results from MRI and echocardiography show a favorable correlation between GLS and EF. They do examine different aspects of heart function even while GLS only assesses longitudinal performance and EF measures some radial and longitudinal performance. According to past study, EF is a worse predictor of outcomes than GLS, and

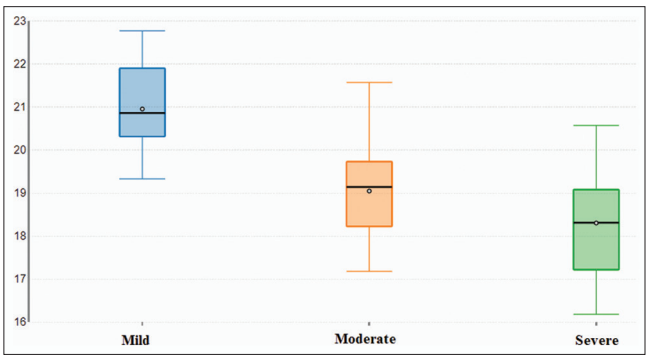


Figure 2: GLS distribution according to different MAC thickness

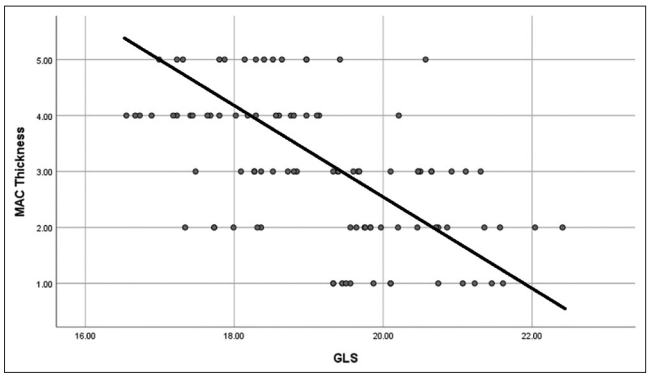


Figure 3: Correlation between GLS and MAC thickness

Table 4: Mechanics of left ventricle

Variable	Mild (n = 19)	Moderate (n = 23)	Severe (n = 18)	f	P1 (mild, moderate)	P2 (moderate, severe)	P3 (mild, severe)
RadSbase (%)	45.07 ± 13.86	42.91 ± 10.02	34.9 ± 10.04	0.001	0.5734	0.7554	0.01379*
RadSR-Sbase (s^{-1})	2.21 ± 0.7	2.01 ± 0.4	1.69 ± 0.3	0.001	0.27567	0.60806	0.00634*
Ar (°)	11.14 ± 2.39	13.27 ± 2.01	14.76 ± 1.4	< 0.0001	0.00367*	0.93049	0.00937*
Ar-rate systole (°/s)	77.15 ± 24.28	92.04 ± 20.4	107.15 ± 16.6	< 0.0001	0.03986*	0.9002	0.01314*
Br-rate systole (°/s)	63.98 ± 17.1	63.35 ± 15.16	52.29 ± 10.45	0.004	0.90111	0.9155	0.01025*
Peak LV twist (°)	18.88 ± 3.71	20.96 ± 2.59	21.39 ± 2.71	0.004	0.0456*	0.86527	0.60289
Peak LV twist rate (°/s)	105.11 ± 24.05	117.86 ± 17.15	119.14 ± 12.04	0.004	0.05956	0.85189	0.7853
LV-tor (°/cm)	2.61 ± 0.5	2.91 ± 0.4	3.02 ± 0.4	0.004	0.04068*	0.91378	0.38035
t-LV UR (%, diastole)	13.73 ± 3.82	19.64 ± 5.79	24.85 ± 6.17	< 0.0001	0.0003*	0.77567	0.00749*

LV: Left ventricle, RadSbase: Basal radial strain, RadSR-Sbase: Basal systolic radial strain rate, Ar: Peak systolic apical rotation, Ar-rate systole: Peak systolic apical rotation rate, Br-rate systole: Peak systolic basal rotation rate, LV-tor: LV torsion, t-LV UR: Time to LV untwisting rate.

assessing myocardial strain may indicate subclinical changes in LV function [16].

In the current study, we found that GLS is lower and linked with illness severity in MAC patients. There are several ways to explain this. MAC has been connected to alterations in coronary microvascular function in earlier studies. Similar to atherosclerosis, it has been proposed that endothelial damage occurs at regions of high mechanical stress, such as the intersection of the ventricular myocardium and the mitral valve annulus [17].

Calcification of the mitral valve and left ventricular (LV) systolic dysfunction has both been connected to abnormal levels of parathormone, Vitamin D, and/or calcium-phosphorous metabolism [18]. Left ventricular (LV) systolic failure may generate systemic inflammation, which in turn can cause myocardial infarction (MAC) [17]. Along the long axis of the ventricle (from base to apex), the mitral annular ring sphincters and bends downward [19].

These motions during systole and diastole must be in sync for the mitral annular ring to function properly. The system's other two components will be put at risk if one is compromised. Reduced global longitudinal strain (GLS) and abnormal long-axis ventricular motion were identified as primary contributors to LV subclinical dysfunction (from base to apex). While early diastolic annular motion has been linked to LV recoil, systolic annular motion has been linked to LV ejection fraction [20]. To determine whether MAC affected the LV on a regional or global scale, we compared individuals with varying degrees of MAC by analyzing the LV's six fundamental components. Each of the six subsegments saw a decrease in LS value as MAC severity increased. These findings illustrate the far-reaching, rather than localized, nature of MAC's effects on the LV.

Table 5: The correlation between mitral annular calcification thickness and clinical and echocardiographic variables

Variable	r	p-value
Age	0.436	< 0.001
LV mass index (g/m^2)	0.41	< 0.001
LAVI (mL/m^2)	0.271	< 0.001
E/A ratio	− 0.401	< 0.001
E/Em ratio	0.339	< 0.001
MPI	0.351	< 0.001
Sm (systolic tissue velocity, cm/s)	− 0.322	< 0.001
GLS	− 0.512	< 0.001
t-LV UR (%, diastole)	0.546	< 0.001

LV: Left ventricle, t-LV UR: Time to LV untwisting rate, LAVI: Left atrial volume index, GLS: Global longitudinal strain, Sm: Mitral annular tissue Doppler S-wave, E: Transmitral Doppler E-wave, A: Transmitral doppler A-wave, Em: Mitral annular tissue doppler E-wave, MPI: Myocardial performance index.

RadSbase and RadSR-Sbase levels in MAC patients were found to be lower than in controls. Individuals with severe MAC had lower levels of RadSbase and RadSR-Sbase than those with mild-to-moderate MAC. In MAC patients, calcium extension was often seen during surgical and postmortem investigations in the muscles of the LV base and beneath the ventricular surface of the posterior mitral leaflet.

Table 6: Multivariate linear regression analysis of the association between mitral annular calcification severity and left ventricle diastolic properties

Variable	B-coefficient	p-value
Age	0.251	< 0.05
LV mass index	0.232	< 0.05
LAVI	0.032	> 0.05
E/Em ratio	0.114	> 0.05
t-LV UR	0.358	< 0.001

LV: Left ventricle, LAVI: Left atrial volume index, E: Transmitral Doppler E-wave, Em: Mitral annular tissue Doppler E-wave, t-LV UR: Time to LV untwisting rate.

This technique could be able to explain why the LV's vulnerable region experiences less radial strain and strain rate (base). Furthermore, it is conceivable that individuals with severe MAC have radial fibers that have been more severely injured and that have calcified more widely near the base of the LV. Our findings concur with those of, who indicated that greater LV filling pressure and longer age may be associated with MAC in nonagenarians. In MAC patients, LV diastolic function was also impaired [21].

Table 7: Multivariate linear regression analysis of the association between myocardial infarction severity and left ventricular systolic parameters

Variable	B-coefficient	p-value
Age (years)	0.331	< 0.001
LV mass index (g/m^2)	0.197	< 0.05
MPI	0.298	< 0.001
Sm (systolic tissue velocity, cm/s)	− 0.098	> 0.05
GLS (%)	− 0.338	< 0.05

LV: Left ventricle, MPI: Myocardial performance index, Sm: Mitral annular tissue doppler S-wave, GLS: Global longitudinal strain.

Some aspects of left ventricular (LV) diastolic function were also shown to correlate with the severity of MAC. In contrast, multivariate analysis revealed that MAC severity was independently linked with t-LV UR. Patients with severe MAC had slower mitral velocities than those without the disease because of aberrant LV relaxation and restricted mitral annular motion.

Our findings corroborate those of earlier studies showing that the E/Em ratio is not a reliable predictor of filling pressure in individuals with severe MAC. To accurately measure the amount of diastolic relaxation experienced by the LV, 2D speckle tracking echocardiography is often employed (STE). Our

data, along with those of, who discovered MAC in 91 (65.5%) of patients, suggest that this calcification does not seem to disrupt the regular flow pattern across the mitral valve [22]. Changes were made to the E/Em ratio, LAVI, LV mass index, and t-LV UR, all of which are diastolic LV characteristics. At the start and progression of MAC, Ar, Ar-rate systole, peak LV twist, peak LV twist rate, LV-tor, and MPI all rose, whereas GLS and Sm decreased. There was a strong correlation between LV mass index, t-LV UR, LV mass index, MPI, and GLS in a multivariate linear regression analysis of MAC severity.

Conclusion

When MAC is present and severe, it compromises the LV's systolic and diastolic functioning. An increase in MAC severity also affects the systolic and diastolic functioning of the LV.

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