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ORIGINAL ARTICLE

Correlation between Mitral Annular Calcification and Coronary Artery Severity in Chronic Coronary Syndrome Patients

Mohamed Elsayed Saleh*, Doaa Mohamed Elsayed, Nada Hassan Metwally,

Cardiology Department, Faculty of Medicine, Zagazig University, Zagazig, Egypt

*Corresponding

author:

Mohamed Elsayed
Saleh

Email address:

Msssaleh123123123@gmail.com

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ABSTRACT

Background: Mitral annulus calcification (MAC) is a progressive, degenerative disease that deteriorates with time. It mostly impacts women and persons aged over 70 years. Our work is to evaluate the prognostic significance of MAC in identifying serious coronary artery disease (CAD).

Methods: This cross-sectional research was performed at the Cardiology Department, Zagazig University Hospital. This research was done on 100 cases with chronic anginal pain who were undergoing percutaneous coronary angiography, were less than 65 years and had MAC by echocardiography. Cases were segregated into two Groups: MAC Group: 50 cases and No MAC Group: 50 cases.

Results: The multivariate logistic regression analysis identified several significant independent predictors of MAC. Family history of coronary artery disease was associated with a slight but significant increase in the odds of MAC (OR = 1.08; 95% CI: 1.02–1.15; $p = 0.01$). Smoking significantly increased the odds of MAC (OR = 2.1; 95% CI: 1.05–4.18; $p = 0.03$). Left main (LM) stenosis showed a strong association with MAC, with patients having more than three times the odds of MAC compared to those without LM stenosis (OR = 3.25; 95% CI: 1.4–7.56; $p = 0.005$). Conversely, having no significant CAD was associated with lower odds of MAC (OR = 0.45; 95% CI: 0.2–0.98; $p = 0.04$). Age was not a significant predictor in this model (OR = 1.65; 95% CI: 0.72–3.8; $p = 0.23$).

Conclusions: These findings highlight the potential clinical value of assessing MAC as a non-invasive marker to aid in the early detection and risk stratification of patients at higher risk for severe CAD. Incorporating MAC evaluation into routine cardiovascular assessment may enhance early diagnosis and guide more targeted management strategies.

Keywords: Mitral Annular Calcification (MAC), Coronary Artery Severity, Chronic Coronary Syndrome

INTRODUCTION

Mitral Annular Calcification (MAC) is a progressive, long-term disease. It mostly impacts older adults and women [1]. Previous research has shown that atrial arrhythmias, heart blocks, mitral regurgitation, infective endocarditis, and mitral stenosis can all be caused by MAC. The severity of this risk factor, as defined by the thickness of the valve in M mode, is linearly linked with the risk of stroke [2]. It is recognized to be a separate risk factor for systemic embolism and stroke. One of the many elements that contribute to

MAC's development is a process similar to atherosclerosis, which causes some cells in the heart valves to undergo transformation [3]. Typically seen in the posterior cusp, the amount of calcification can range from a little, visible spicule to a massive, chip-like mass. Calcification of the mitral valve can develop either as a ring or along the leaflets. On rare occasions, it may even cause the anterior leaflet to grow in length. Transthoracic echocardiography (TTE) is a common way to accidentally find MAC, although the condition is typically asymptomatic [4]. Several cardiovascular

diseases (CVD), including enlarged atria and ventricles, myocardial infarction, atrial fibrillation, and cerebrovascular accidents, have been linked to MAC in previous research [5, 6]. Both the Framingham Heart Study and a research including individuals younger than 61 years old have established that there is an elevated risk for mortality related to MAC [7]. Although the relationship between mitral annular calcification and coronary artery disease has been established in elderly patients, this association remains insufficiently explored in younger patients with chronic coronary syndrome. Evaluating this relationship in the younger age group carries significant clinical importance, as calcification may represent an early marker of accelerated and severe atherosclerosis, allowing for more effective preventive interventions to improve long-term health outcomes. Therefore, we set out to determine if MAC is a reliable indicator of serious coronary artery disease (CAD) in this investigation.

METHODS

This cross-sectional research was done at the Cardiology Department, Zagazig University Hospital. This study was performed on 100 cases with chronic anginal pain who were undergoing percutaneous coronary angiography, were less than 65 years old, and had MAC by echo. Patients were segregated into two groups: the MAC group and the No MAC group (50 cases each). The sample size was calculated to be (100) cases by assuming that interval 95% by using the Open EPI database. Inclusion criteria: patients with chronic coronary syndrome candidates for coronary angiography due to recurrent anginal pain with echo finding of MAC. Exclusion criteria: Moderate or severe valve stenosis or regurge, left ventricular ejection fraction (LVEF) <50%, AF or other arrhythmias, renal or hepatic failure.

Every single patient underwent complete history taking, including age, sex, comorbidities, DM, HTN, and smoking. Comprehensive clinical examination, including a pulse, blood pressure, respiratory rate, and temperature. Laboratory examination: troponin, Ck-MB, lipid profile, and creatinine level. Radiological examination: electrocardiogram and conventional echocardiographic examination to assess the degree of MAC, wall motion abnormalities plus ejection

fraction, and coronary angiography for assessing severity of coronary affection by SYNTAX score.

Study Procedures

TTE revealed MAC as a highly echogenic structure at the parasternal long axis view junction of the posterior mitral leaflet and the atrioventricular groove. For mild to severe MAC, the distance between the leading and trailing edges must be between 1 and 4 mm, while severe MAC is defined as >4 millimeters, taking its thickness into account, angiographic pattern and severity of CAD. During the same hospital stay, CAG was performed after all regular investigations. Two cardiologists used visual estimate to interpret coronary angiograms and determine the degree of CAD. The SYNTAX score is used in coronary angiography to determine the severity of coronary disease.

Ethical Consideration

The institution research board (IRB) approved the investigation for the Faculty of Medicine at Zagazig University. Each participant who participated in the investigation provided valid informed assent. It was ensured that the data collected was not used for any other purpose, and confidentiality and personal privacy were respected at all stages of the study (1189/17/6/2025)

Statistical analysis

Data analysis was carried out using SPSS version 25, which stands for the Statistics Package for the Social Sciences. Quantitative measures used to depict qualitative data included percentages and frequencies. The mean $\pm$ standard deviation (mean $\pm$ SD) was used to represent continuous quantitative data. The categorical variables were compared utilizing the chi-square test, while the continuous variables were analyzed utilizing the independent sample t-test. Regression analysis (odd ratio) was used for studying the effects of one variable on the outcome of cases.

RESULTS

Table 1 showed that there was no statistically significant difference between the MAC and no-MAC groups regarding age ($p > 0.05$) or gender distribution ($p > 0.05$). Although the mean age was slightly higher in the MAC group (57.39 ± 6.87 years) compared to the no-MAC group (55.47 ± 7.56 years), this difference did not reach statistical significance. Similarly, gender distribution showed a higher percentage of males in the no-MAC group

(74%) compared to the MAC group (64%), but the difference was also not significant. Regarding cardiovascular risk factors, there was a highly significant difference in family history of CAD, which was more frequent in the MAC group (42%) compared to the no-MAC group (14%) ($p = 0.001$). Likewise, smoking was significantly more prevalent in the MAC group (54%) versus the no-MAC group (24%) ($p = 0.001$). On the other hand, there were no significant differences between the groups in terms of other comorbidities, including diabetes mellitus (34% vs. 24%, $p = 0.27$), hypertension (40% vs. 48%, $p = 0.42$), or hyperlipidemia (36% vs. 44%, $p = 0.41$). Table (2) showed that the left main (LM) stenosis was more frequent in the MAC group (18%) compared to the no-MAC group (4%) ($p = 0.02$). Similarly, three-vessel disease (TVD) was significantly more common in the MAC group (54%) versus 30% in the no-MAC group ($p = 0.01$). Double-vessel disease (DVD) and single-vessel disease (SVD) were also significantly higher in the MAC group (22% and 26%, respectively) compared to the no-MAC group (8% and 6%) with p -values of 0.04 and 0.001, respectively. Conversely, no significant CAD was more prevalent in the no-MAC group (38%) compared to the MAC group (12%) ($p = 0.001$), highlighting the strong correlation between MAC and more extensive CAD. Furthermore, the distribution of the studied group based on the severity classification showed that 22% had no detectable changes, 34% had minor involvement, 28%

had marked changes, and 16% had diffuse involvement. This spread indicates a varied range of severity within the cohort (Table 3). In table (4), the mortality rate was significantly higher in the MAC group (12%) compared to the no-MAC group (2%), with the difference reaching statistical significance ($p = 0.05$), suggesting that the presence of MAC may be associated with increased risk of mortality besides the highly significant elevation of the syntax score among the MAC group ($p = 0.001$). The multivariate logistic regression analysis identified several significant independent predictors of MAC. Family history of coronary artery disease was associated with a slight but significant increase in the odds of MAC (OR = 1.08; 95% CI: 1.02–1.15; $p = 0.01$). Smoking significantly increased the odds of MAC (OR = 2.1; 95% CI: 1.05–4.18; $p = 0.03$). Left main (LM) stenosis showed a strong association with MAC, with patients having more than three times the odds of MAC compared to those without LM stenosis (OR = 3.25; 95% CI: 1.4–7.56; $p = 0.005$). Conversely, having no significant CAD was associated with lower odds of MAC (OR = 0.45; 95% CI: 0.2–0.98; $p = 0.04$). Age was not a significant predictor in this model (OR = 1.65; 95% CI: 0.72–3.8; $p = 0.23$), as shown in Table (5). The distribution of echocardiography findings in patients based on the syntax score was revealed in Table (6).

Table (1): Distribution of clinical characteristics and symptoms between the studied groups.

		MAC N=50	No MAC N=50	p-value
Age (years)				
mean± SD		57.39±6.87	55.47±7.56	0.15
Gender	Male	32 (64%)	37 (74%)	0.27
	Female	18 (36%)	13 (26%)	
Family history		21 (42%)	7 (14%)	0.001**
Comorbidities				
Diabetes mellitus		17 (34%)	12 (24%)	0.27
Hypertension		20 (40%)	24 (48%)	0.42
Hyperlipidemia		18 (36%)	22 (44%)	0.41
Smoking		27 (54%)	12 (24%)	0.001**
Symptoms				
CRF		16 (32%)	6 (12%)	0.01*
CABG		20 (40%)	10 (20%)	0.02*
PCI		25 (50%)	9 (18%)	0.001**
Chest pain		21 (42%)	18 (36%)	0.53

MAC: Mitral annular calcification

Table 2. Distribution of Angiographic findings between the studied groups

	MAC N=50	No MAC N=50	p-value
LM stenosis	9 (18%)	2 (4%)	0.02*
TVD	27 (54%)	15 (30%)	0.01*
DVD	11 (22%)	4 (8%)	0.04*
SVD	13 (26%)	3 (6%)	0.001**
No significant CAD	19 (38%)	6 (12%)	0.001**

MAC: Mitral annular calcification

Table 3. Distribution of mitral valve calcification between the studied groups

Grading	Studied group N=50
No	11 (22%)
Minor	17 (34%)
Marked	14 (28%)
Diffuse	8 (16%)

Table 4. Distribution of outcome between the studied groups.

	MAC N=50	No MAC N=50	p-value
Mortality	6 (12%)	1 (2%)	0.05*
Syntax Score (Mean $\pm$ SD)	29.3 $\pm$ 5.39	21.25 $\pm$ 3.21	0.001**

MAC: Mitral annular calcification

Table (5): Regression analysis of the factors associated with the presence of significant coronary artery disease

	95% CI	OR	p-value
Hyperlipidemia	4.58	3.51	0.01
Gender	5.47	2.36	0.02
MAC	5.87	2.11	0.04
Smoking	6.56	2.21	0.05
Chest pain	3.98	2.09	0.001**
Diabetes mellitus	3.71	1.97	0.25
Age $\geq$ 60 years	2.14	1.99	0.04
CRF	1.24	0.30	0.31

OR: odd ratio, CI: confidence interval

Table (6): Distribution of echocardiography findings in patients based on the syntax score

	<23 N= 54	$\geq$ 23 N=46	p-value
LA diameter	39 $\pm$ 4.49	39.1 $\pm$ 4.21	0.90
LA area	20.6 $\pm$ 3.74	20.0 $\pm$ 3.0	0.38
LA volume	62.3 $\pm$ 15.74	61.3 $\pm$ 15.78	0.75
LA volume indexed	33.16 $\pm$ 7.12	31.3 $\pm$ 7.51	0.20
LVEDD	49.6 $\pm$ 3.74	49.6 $\pm$ 5.26	1
LVESD	35.2 $\pm$ 5.24	39.6 $\pm$ 6.01	0.001**
LVEF	48.3 $\pm$ 7.49	41.8 $\pm$ 12.7	0.001**

	<23 N= 54	≥23 N=46	p-value
E septal	6.9±1.49	5.1±2.02	0.001**
E lateral	8.3±2.24	7.3±2.25	0.02*
E velocity	60.8±16.8	63.1±18.7	0.51
TAPSE	19.6±2.9	19.3±3.02	0.61
RVSm	11.1±1.49	11.2±1.50	0.73
Aortic atherosclerosis	11 (20.3%)	11 (23.91%)	0.66
Aortic calcification	5 (9.2%)	8 (17.3%)	0.22
LVH	4 (7.4%)	5 (10.8%)	0.54

LVESD: Left ventricular end systolic diameter, LVEDD: Left ventricular end Diastolic diameter,

DISCUSSION

Our findings revealed that the groups that were examined did not differ significantly in terms of age, diabetes mellitus, sex, hypertension & hyperlipidemia. However, there was a significant difference in terms of family history and smoking. The results showed that the groups were significantly different when it came to CRF, CABG, and PCI, but when it came to chest pain, the groups were not significantly different. Our findings were consistent with Atar et al. [9] who established the frequency of serious CAD in younger individuals with MAC. They reported that there was no statistically significant variation amongst individuals with MAC and the control group (no MAC) concerning risk factors or clinical presentation, including age, sex, CABG, and chest pain. Contrarily, the index group had 24% of its participants receive PCI, whereas the control group had 40% ($p = 0.024$). Our results were also consistent with those of Moradi et al. [10] who used coronary CT angiography to examine the clinical and prognostic significance of MAC in CAD in a sample of Iranian patients. They mentioned that those with MAC are contrasted with those without MAC in terms of their basic traits. Cases with MAC had significantly larger mean ages (69.34 ± 8.20 vs. 60.64 ± 11.42 , $P < 0.001$) and GFRs (69.67 ± 20.92 vs. 78.00 ± 20.23 , $P = 0.005$) contrasted with those without MAC. Those with MAC probably had a greater significant CAD than those without MAC (53.8% vs. 46.2%, $P = 0.036$). Comparing the two groups, we find that hypertension is more common in one (47.9% vs. 52.1%, $P = 0.024$). Other fundamental factors did not show a statistically significant difference ($P > 0.05$) between the two

groups. Rahman et al. [11] also found that MAC was related to a more severe case of CAD in individuals younger than 65 years old, which is consistent with our findings. Both the MAC and non-MAC groups of patients were found to be of comparable age and sex, according to their findings. In contrast, the MAC group had substantially higher rates of smoking ($p = 0.001$) and a family history of IHD ($p=0.03$). In terms of angiographic results, we found a significant association between the presence of MAC and the severity of CAD. Left main (LM) stenosis, three-vessel disease (TVD, double-vessel disease (DVD), and single-vessel disease (SVD) were more frequent in the MAC group compared to the no-MAC group. Conversely, no significant CAD was more prevalent in the no-MAC group (38%) compared to the MAC group (12%) ($p = 0.001$), highlighting the strong correlation between MAC and more extensive CAD. Our results were also consistent with those of Atar et al. [9], who discovered a statistically significant increase in the prevalence of obstructive CAD in the index group in comparison with the control group (88% vs. 68%, $p = 0.0004$). Significant LM coronary artery stenosis was more common in the index group (14% v 4%, $p = 0.009$), as was triple vessel CAD (54% v 33%, $p = 0.002$). Both groups had similar rates of both double and single vessel disease. Also, our results were in harmony with Rahman et al. [11] who reported a significantly greater incidence of anterior MI ($p = 0.03$) in the MAC group. Compared to the non-MAC group, the MAC group had significantly greater levels of LM and TVD ($p = 0.001$, $p = 0.01$), and more normal vessels ($p = 0.001$). This study revealed that 22% of patients had no MVC, 34% of them had minor MVC, 28% had

marked MVC, and 16% had diffuse MVC. Our findings revealed that mortality was significantly higher in the MAC group (12%) compared to the no-MAC group (2%), suggesting that the presence of MAC may be associated with increased risk of mortality. Our results reported that the multivariate logistic regression analysis identified several significant independent predictors of MAC. Family history of coronary artery disease was associated with a slight but significant increase in the odds of MAC (OR = 1.08; 95% CI: 1.02–1.15; $p = 0.01$). Smoking significantly increased the odds of MAC (OR = 2.1; 95% CI: 1.05–4.18; $p = 0.03$). Left main (LM) stenosis showed a strong association with MAC, with patients having more than three times the odds of MAC compared to those without LM stenosis (OR = 3.25; 95% CI: 1.4–7.56; $p = 0.005$). Past research has shown a robust correlation between MAC and CVD. Case in point: Fox et al. [7] discovered that MAC increased the odds of incident CVD and death from any cause. Just as Potpara et al. [12] found that MAC was related to an elevated risk of cardiovascular mortality and morbidity in atrial fibrillation patients, so too did other researchers. Ischemic stroke and vascular mortality were among the cardiovascular outcomes that Kohsaka et al. [1] found to be significantly greater in a group of 1,955 patients who were treated with MAC. Most notably, the researchers found that MAC elevated the risk of MI (adjusted hazard ratio: 1.75). As a whole, these results point to MAC as a strong predictor of adverse cardiovascular outcomes. In agreement with our findings, Atar et al. [9] revealed that independent predictors of significant CAD included hyperlipidemia ($p = 0.002$), MAC ($p = 0.02$), chest pain ($p = 0.02$), smoking ($p = 0.05$), age ≥ 60 ($p = 0.04$), and male sex ($p = 0.02$). Based on their findings, individuals under the age of 65 who complain of chest discomfort on angiography are more likely to have significant stenosis ($\geq 70\%$ diameter stenosis) in one or more main epicardial coronary arteries if MAC is present. In addition, the presence of MAC increases the likelihood of triple vascular disease or significant LM coronary artery stenosis in this patient population. Women under the age of 65 who did not have obstructive CAD were not more likely to have MAC. Furthermore, the multi-ethnic research of

atherosclerosis by Kanjanauthai et al. [13] discovered a correlation among MAC and cardiovascular risk factors such as advanced age, female gender, diabetes mellitus, and elevated body mass index. In a similar vein, Rahman et al. [11] demonstrated that the following four characteristics were shown to be significant predictors of severe CAD out of seven: MAC ($p=0.02$), a family history of CAD ($p=0.03$), smoking ($p=0.05$), and dyslipidemia ($p=0.05$). The corresponding odds ratios (ORs) were 2.84, 2.69, 2.68, and 2.67, respectively.

CONCLUSIONS

This study demonstrates that MAC is a significant and independent predictor of severe CAD. Patients with MAC were more likely to have adverse cardiovascular risk profiles, particularly a positive family history of CAD and smoking, both of which emerged as independent predictors of MAC. The existence and severity of MAC were strongly associated with critical CAD findings, including LM stenosis, single, double, and TVD, as well as increased mortality rates. These findings highlight the potential clinical value of assessing MAC as a noninvasive marker to aid in the early detection and risk stratification of patients at higher risk for severe CAD. Incorporating MAC evaluation into routine cardiovascular assessment may enhance early diagnosis and guide more targeted management strategies.

IMPLICATIONS

The findings of this study provide several important clinical and research implications. First, the demonstration that mitral annular calcification (MAC) is an independent predictor of severe coronary artery disease (CAD) underscores its potential role as a simple, non-invasive echocardiographic marker for cardiovascular risk stratification. Routine assessment of MAC during echocardiography could allow clinicians to identify high-risk patients, even in those younger than 65 years, who may otherwise appear to have a lower risk profile. Second, integrating MAC evaluation into clinical decision-making may improve the early detection of patients with significant CAD, thereby facilitating timely referral for coronary angiography or advanced imaging. This has the potential to optimize the allocation of healthcare resources by prioritizing invasive diagnostic procedures for

patients at greater risk. Third, the study's findings highlight the need for more aggressive preventive and therapeutic strategies in patients with MAC, especially those who present with additional risk factors such as hyperlipidemia, smoking, or a positive family history. Incorporating MAC into existing CAD risk prediction models may improve their predictive accuracy and support more personalized management approaches. Finally, these results suggest the necessity of larger, multicenter, and longitudinal studies to confirm the prognostic value of MAC across diverse populations. Future studies should also explore the pathophysiological mechanisms linking MAC with CAD progression, as well as the potential impact of interventions targeting modifiable cardiovascular risk factors on the natural history of MAC.

Conflict of Interest:

None

Financial Disclosures:

None

Availability of the data:

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

Author's contribution:

M.E.S. conceptualized and designed the study, contributed to data collection, and drafted the main manuscript text. N.H.M. and D.M.E. participated in data collection, statistical analysis, and manuscript writing. M.E.S performed additional statistical analysis, contributed to manuscript revision, and provided critical insights into data interpretation. All authors reviewed and approved the final manuscript.

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