Original Article

Prevalence and Prognostic Value of Papillary Muscle Infarction in Patients with Previous Myocardial Infarction

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Objective: To determine the prevalence and prognostic value of papillary muscle infarction.

Materials and Methods: The authors studied patients who were referred for cardiac magnetic resonance [CMR] for assessment of myocardial ischemia or myocardial viability and found to have previous myocardial infarction. CMR was performed for assessment of cardiac function and late gadolinium enhancement [LGE], including detection and quantification of mitral regurgitation. Patients were followed for cardiac events, including cardiac death, nonfatal myocardial infarction, and hospitalization due to heart failure.

Results: The authors studied 785 patients with previous myocardial infarction. Average age was 64.8 years. Papillary muscle infarction was found in 305 patients (38.9%). Papillary muscle infarction was associated with an increase in left ventricular volumes, mass, wall motion score, LGE percentages, and mitral regurgitation. Patients with papillary muscle infarction had increased risk of cardiac event during follow-up. Multivariable analysis showed that left ventricular end-systolic volume index and mitral regurgitation were independent predictors for cardiac event.

Conclusion: Papillary muscle infarction is not an uncommon condition. It was found to be associated with more severe left ventricular dysfunction. The increased risk associated with papillary muscle infarction was mediated by more severe myocardial damage and mitral regurgitation.

Keywords: Papillary muscle, Myocardial infarction, Late gadolinium enhancement, Cardiac magnetic resonance

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Papillary muscle infarction or dysfunction is a known complication of myocardial infarction. Serious complications of papillary muscle infarction include papillary muscle rupture or mitral regurgitation[1]. Association between papillary muscle infarction and mitral regurgitation remains unclear, however, a decrease in mitral regurgitation has been reported to be associated with papillary muscle dysfunction[2,3]. Many mechanisms have been proposed to be more important than papillary muscle dysfunction in the development of mitral regurgitation including displacement of papillary muscle[4], a more leaning papillary muscle or shorter chordae tendineae[5] and impaired basal rotation[6]. Prevalence of mitral regurgitation during myocardial infarction has been reported to be as high as 35%[7]. Even a mild degree of mitral regurgitation after myocardial infarction may be associated with increased risk of cardiac event[7]. Very limited data is available on the prevalence of papillary muscle infarction during or after myocardial infarction, given that it is difficult to diagnose or visualize infarction of the papillary muscle. However, late gadolinium enhancement [LGE] technique by cardiac magnetic resonance [CMR] facilitates accurate identification of the area and estimate the extent of myocardial infarction[8] including the visualization of papillary muscle infarction[9,10]. CMR can detect and estimate the extent and severity of mitral regurgitation as well[11].

Although LGE can be detected in different forms of cardiomyopathy, LGE in the subendocardial region and the presence of a transmural pattern has been reported to be ischemic in origin[12]. Patients with LGE have an increased risk of cardiovascular events, such as patients with documented or suspected coronary artery disease[13]. Data regarding the prevalence and prognostic value of papillary muscle infarction are limited.

The objectives of this study were to determine the prevalence of papillary muscle infarction in patients with previous myocardial infarction as defined by CMR definition[14] and evaluate the prognostic value of papillary muscle infarction.

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Materials and Methods

Patient population

The authors enrolled patients above 30 years of age who were referred for CMR for assessment of myocardial ischemia or myocardial viability and found to have positive LGE associated with abnormal wall motion, which is consistent with previous myocardial infarction, which defined as positive delayed enhancement without evidence of non-ischemic cause[14]. Since a significant number of patients with myocardial infarction may go unrecognized and electrocardiogram [ECG] criteria might not be sensitive[15], the authors used this imaging definition for previous myocardial infarction to identify both symptomatic and silent myocardial infarction. Patients were excluded if they had one or more of the following conditions 1) contraindication for CMR such as pacemaker, internal defibrillator implantation or intracranial clip, 2) unstable clinical conditions including history of acute coronary syndrome within 1 month, 3) pregnancy, 4) inability to complete CMR examination, 5) history of allergy to gadolinium, 6) poor image quality, 7) history of claustrophobia, 8) known disease other than ischemic cardiomyopathy that may cause LGE including hypertrophic cardiomyopathy, amyloidosis, dilated cardiomyopathy, and myocarditis[12], and 9) unable to obtain follow-up data.

Study protocol

The protocol of the present study was approved by the Siriraj International Review Board [SIRB] and all participants provided the written informed consent.

CMR protocol

CMR was performed using 1.5 Tesla Gyroscan NT Intera Philips scanner (Philips Medical Systems, Best, the Netherlands) for assessment of cardiac function and LGE. Steady-state free precession [SSFP] technique with cardiac gating sequence was used for assessment of cardiac function in the long axis, multiple slice short axis and 4-chamber view. The following parameters were used for cardiac function, repetition time/echo time/number of excitations (TR/TE/NEX) = 3.7/1.8/2, 390×312 mm field of view, 256×240 matrix, 1.52×1.3 reconstruction pixel, 8 mm slice thickness, and 70-degree flip angle.

LGE was evaluated by 3D segmented-gradient-echo inversion-recovery sequence in long axis short axis slices from mitral valve plane to left ventricular apex at a similar position as the functional study and 4-chamber view. Images were acquired 10 minutes after intravenous injection of 0.15 mmol/kg of gadolinium (Magnevist, Schering AG, Berlin, Germany). The following parameters were used: TR/TE = 4.1/1.25 ms, 303×384 mm field of view, 240×256 matrix, 1.26×1.5 mm reconstruction pixel, 8 mm slice thickness, 15-degree flip angle, and 1.5 sensitivity encoding [SENSE] factor.

Analysis of CMR images

CMR images were analyzed on a ViewForum workstation (Philips Medical Systems, Best, the Netherlands) with reviewer blinded patient clinical data. Endocardial and epicardial borders were automatically detected from the short-axis series, followed by manual editing. Left ventricular end-diastolic volume [LVEDV], left ventricular end-systolic volume [LVESV], and left ventricular mass [LVMASS] was automatically calculated. Left ventricular ejection fraction [LVEF] was calculated from LVEDV-LVESV/LVEDV and adjusted to percentage. Left ventricular end-diastolic volume index [LVEDVI], left ventricular end-systolic volume index [LVESVI] and left ventricular mass index [LVMASSI] were calculated by adjustment of the body surface area. The authors applied the 17-segment model for myocardial segmentation as recommended by the American Heart Association [AHA][16] with the exception of segment 17. Wall motion of each myocardial segment was graded, as 1 = normal, 2 = hypokinesia, 3 = akinesia, and 4 = dyskinesia or aneurysm. An average of wall motion score was also calculated.

Analysis of LGE was performed by visual assessment. Extent of LGE area was divided by visual assessment into five grades as 0 = no scar, 1 = 1% to 25%, 2 = 26% to 50%, 3 = 51% to 75%, and 4 = 76% to 100%. Total infarct size was calculated from the sum of all extent of infarction scores divided by four times the total number of segments[8]. Papillary muscle infarction was determined by LGE of short axis views and confirmed by 2-chamber and 4-chamber views.

LGE was analyzed by visual assessment and determined by consensus of 2 readers. Intra-observer and inter-observer agreement for the presence of a LGE area in our center were k = 0.94, $p<0.001$ and k = 0.97, $p<0.001$, respectively[17]. Intra-observer and inter-observer variability of the extent of LGE in our center were 1.9±5.1% and 2.8±9.2%, respectively. Intra-observer and inter-observer agreement for detection of papillary muscle infarction was 0.98 and 0.95, respectively.

Grading of mitral regurgitation was performed by calculation of the regurgitation fraction from the ratio of the area of the regurgitant jet and the area of the left atrium during end-systole by tracing the left atrium area, excluding the pulmonary vein. Regurgitation fractions of the long-axis and 4-chamber views were averaged and graded as mild, moderate or severe. Intra-observer and inter-observer agreement of the detection of mitral regurgitation was 0.99 and 0.96, respectively. This technique was validated by proximal isovelocity surface area [PISA] method using echocardiogram in 43 consecutive patients with mitral regurgitation in our center with statistically significant correlation ($r = 0.72$, $p<0.001$)[18].

Clinical follow-up

Clinical follow-up was performed by review of the medical record and scripted telephone interview. Primary outcome was a composite outcome that included cardiac death, nonfatal myocardial infarction, and hospitalization due to heart failure. Outcomes were determined by consensus of two cardiologists using the best available information.

Statistical analysis

Continuous data were expressed as mean ± standard deviation [SD] and categorical variables as number and percentages. Comparisons of continuous data were made by Student's t-test for unpaired data and Chi-square test for comparisons of categorical data. Cox regression analysis was performed to assess univariate and multivariate outcomes as predictor for cardiovascular outcome. Kaplan-Meier survival curves were used to describe survival probability. The data were analyzed by SPSS version 20.0. A *p*-value of less than 0.05 was considered statistically significant.

Results

Seven hundred eighty-five patients with evidence of previous myocardial infarction from CMR were enrolled. There were 580 males (73.9%) and the average age of the study population was 64.8±10.5 years. Transmural infarction was demonstrated in 529 patients (67.4%). Papillary muscle infarction was detected in 305 patients (38.9%), with anterior papillary muscle infarction in 174 (22.2%) and posterior papillary muscle infarction in 269 (34.3%). Infarction of both anterior and posterior papillary muscles was demonstrated in 138 patients (17.6%). Images of papillary muscle infarction are shown in Figure 1. Baseline characteristics of patients with and without papillary muscle infarction are shown in Table 1. Patients with papillary muscle infarction were more likely to be male, have prior revascularization, and dyspnea, be on nitrate or antiplatelets and less likely to be hypertensive, have angina, or be on calcium antagonist. CMR findings in patients with and without papillary muscle infarction are shown in Table 2. Patients with papillary muscle infarction had a lower LVEF, greater left ventricular volumes and mass, greater average wall motion score and percentages of LGE, and more likely to have mitral regurgitation. Only two patients with severe mitral regurgitation required mitral valve surgery. Both patients had papillary muscle infarction.

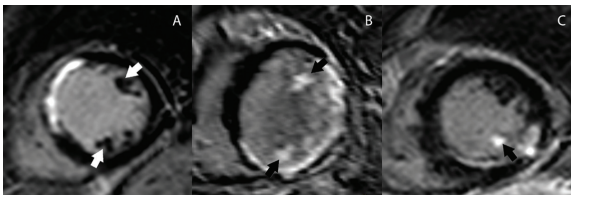

Figure 1. A) LGE images shows normal papillary muscles (white arrow), B) LGE of anterior and posterior papillary muscles (black arrow), and C) LGE of posterior papillary muscle (black arrow).

Average follow-up duration was 37±21 months. Cardiac events during follow-up were cardiac death in 36 (4.6%), nonfatal myocardial infarction in 51 (6.5%), hospitalization due to heart failure in 75 (9.6%), hospitalization due to unstable angina in 39 (5.0%), percutaneous coronary intervention in 116 (14.8%), coronary bypass surgery in 138 (17.6%) and stroke in five (0.6%). Table 3 shows univariate and multivariable analysis of the predictors of cardiac events. Significant predictors from univariate analysis included use of beta blocker, use of statin, LVEF, LVEDVI, LVESVI, LVMASSI, percentages of LGE, papillary muscle infarction, and the presence of mitral regurgitation. From multivariate analysis, LVESVI, and the presence of mitral regurgitation were identified as independent predictors of cardiac event whereas statin use is a protective factor. Survival curves stratified by mitral regurgitation, LVESVI, and papillary muscle infarction are shown in Figure 2.

Discussion

The results of the present study showed that papillary muscle infarction was presented in 38.9% of patients with previous myocardial infarction. Papillary muscle infarction was associated with more severe left ventricular systolic dysfunction, presence of mitral

Table 1. Baseline characteristic of patients with and without papillary muscle infarction

Variables	All patients (n = 785)	Patients with PMI (n = 305)	Patients without PMI (n = 480)	*p*-value
Male	580 (73.9)	255 (83.6)	325 (67.7)	<0.001
Age	64.8±10.5	64.7±10.1	64.9±10.8	0.782
Smoker	236 (30.1)	101 (33.1)	135 (28.1)	0.137
Hypercholesterolemia	542 (69.0)	214 (70.2)	328 (68.3)	0.589
Diabetes mellitus	314 (40.0)	126 (41.3)	188 (39.2)	0.550
Hypertension	466 (59.4)	165 (54.1)	301 (62.7)	0.017
Prior revascularization	226 (28.8)	108 (35.4)	118 (24.6)	0.001
Angina	443 (56.4)	158 (51.8)	285 (59.4)	0.037
Dyspnea	375 (47.8)	160 (52.5)	215 (44.8)	0.036
Medications				
Beta-blocker	436 (55.5)	166 (54.4)	270 (56.3)	0.616
Calcium antagonist	99 (12.6)	24 (7.9)	75 (15.6)	0.001
Nitrate	408 (52.0)	181 (59.3)	227 (47.3)	0.001
Antiplatelet	614 (78.2)	253 (83.0)	361 (75.2)	0.010
ACEI or ARB	422 (53.8)	175 (57.4)	247 (51.5)	0.105
Statin	539 (68.7)	215 (70.5)	324 (67.5)	0.378

PMI = papillary muscle infarction; ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker

Variables are presented with count (percentages) or mean ± SD

Table 2. Comparisons of CMR findings in patients with and without PMI

Variables	All patients (n = 785)	Patients with PMI	Patients without PMI	*p*-value
LVEF (%)	44.5±19.2	35.4±16.8	50.2±18.5	<0.001
LVEDVI (ml/m^2)	100.6±46.9	118.7±51.8	89.0±39.3	<0.001
LVESVI (ml/m^2)	62.2±47.4	82.2±52.2	49.4±39.0	<0.001
LVMASSI (g/m^2)	67.7±23.3	72.7±23.2	64.4±22.8	<0.001
Wall motion score	1.75±0.57	1.97±0.52	1.60±0.55	<0.001
Extent of LGE (%)	27.8±18.1	37.1±18.3	21.8±15.3	<0.001
Mitral regurgitation	244 (31.1)	125 (41.0)	119 (24.8)	<0.001
Severity of mitral regurgitation				0.924
Mild	184 (75.4)	94 (75.2)	90 (75.6)	
Moderate	55 (22.5)	28 (22.4)	27 (22.7)	
Severe	5 (2.0)	3 (2.4)	2 (1.7)	

CMR = cardiac magnetic resonance; PMI = papillary muscle infarction; LVEF = left ventricular ejection fraction; LVEDVI = left ventricular end-diastolic volume index; LVESVI = left ventricular end-systolic volume index; LVMASSI = left ventricular mass index; LGE = late gadolinium enhancement

Variables are presented with count (percentages) or mean ± SD

regurgitation and higher percentages of LGE. Although papillary muscle infarction was a significant predictor of cardiac events, it was not found to be an independent predictor in multivariate analysis.

Prevalence of papillary muscle dysfunction has been reported to be as high as 40% among patients after acute myocardial infarction[19] and up to 53% in patients with previous myocardial infarction[20], although some studies reported a lower prevalence of papillary muscle infarction[21] in patients with acute myocardial infarction. Patients with papillary infarction may present with mitral regurgitation immediately after myocardial infarction. However, mitral regurgitation that develops after myocardial infarction in the majority of patients are not related to papillary infarction[4]. The authors have little data on the proportion of patients with papillary muscle infarction. As such, papillary muscle infarction may be under-recognized given that an accurate tool for diagnosis is not yet available. Echocardiogram including tissue Doppler imaging is the initial investigation for assessment of mitral regurgitation and papillary muscle dysfunction or rupture[22]. The present study showed prevalence of papillary muscle infarction to be 38.9%. The 3D image acquisition technique used in this study has been shown to be superior to conventional 2D imaging for papillary scar measurement[23].

The use of a short inversion time may improve

Table 3. Univariate and multivariable associations with cardiac events

Variables	Univariate analysis		Multivariable analysis	
	Adjusted HR (95% CI)	*p*-value	Adjusted HR (95% CI)	*p*-value
Male gender	1.07 (0.74 to 1.53)	0.725		
Age (10 years increment)	1.13 (0.92 to 1.31)	0.122		
Smoker	1.16 (0.70 to 1.87)	0.516		
Hypercholesterolemia	1.04 (0.62 to 1.71)	0.826		
Diabetes mellitus	1.10 (0.80 to 1.51)	0.565		
Hypertension	1.26 (0.91 to 1.75)	0.169		
Prior revascularization	0.97 (0.68 to 1.37)	0.844		
Angina	0.77 (0.56 to 1.06)	0.104		
Dyspnea	1.08 (0.79 to 1.48)	0.628		
Medications			0.72 (0.52 to 0.99)	0.046
Beta-blocker	0.76 (0.55 to 1.04)	0.085		
Calcium antagonist	0.88 (0.53 to 1.48)	0.631		
Nitrate	1.18 (0.86 to 1.62)	0.319		
Statin	0.72 (0.52 to 0.99)	0.049		
Antiplatelet	0.81 (0.56 to 1.17)	0.261		
ACEI or ARB	0.95 (0.69 to 1.30)	0.728		
CMR parameters			1.57 (1.19 to 2.06)	0.001
LVEF (per 10% increment)	1.23 (1.13 to 1.35)	<0.001		
LVEDVI (per 30 ml/m^2 increment)	1.18 (1.07 to 1.29)	0.001		
LVESVI (per 30 ml/m^2 increment)	1.22 (1.12 to 1.33)	<0.001		
LVMASSI (per 30 g/m^2 increment)	1.28 (1.11 to 1.47)	0.001		
Wall motion score	1.58 (1.20 to 2.09)	0.001		
Percentages of LGE	1.15 (1.05 to 1.26)	0.002		
Papillary muscle infarction	1.70 (1.05 to 2.54)	0.014		
Presence of MR	2.05 (1.49 to 2.81)	<0.001	1.76 (1.24 to 2.48)	0.001

HR = hazard ratio; ACEI = angiotensin converting enzyme inhibitor; ARB = angiotensin receptor blocker; CMR = cardiac magnetic resonance; LVEF = left ventricular ejection fraction; LVEDVI = left ventricular end-diastolic volume index; LVESVI = left ventricular end-systolic volume index; LVMASSI = left ventricular mass index; LGE = late gadolinium enhancement

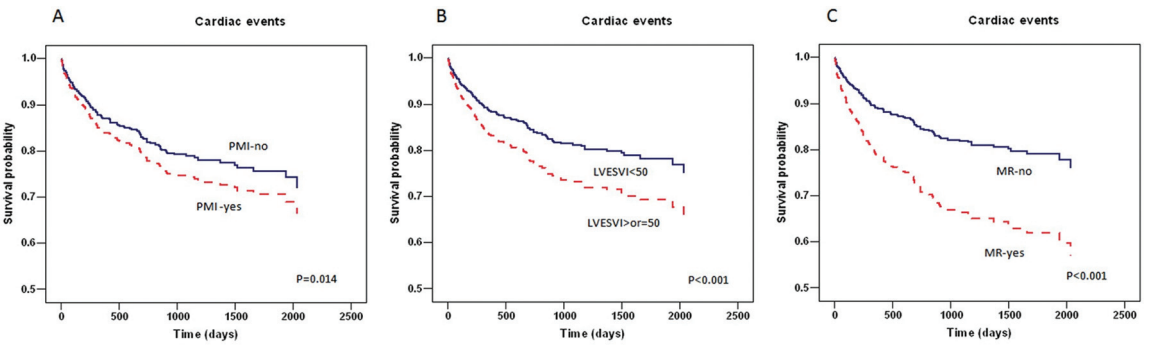

Figure 2. Kaplan-Meier curves of death or nonfatal myocardial infarction stratified by A) the presence or absence of papillary muscle infarction [PMI], B) high and low LVESVI, and C) the presence or absence of mitral regurgitation.

spatial resolution and signal-to-noise ratio, which may increase the sensitivity of detection of papillary muscle infarction[24]. Posterior papillary muscle was involved more often than anterior papillary muscle in the present study. This finding is similar to previous reports[19,20] and may be related to the single blood supply to the posterior papillary muscle by the right coronary and dual blood supply by the left anterior descending artery and left circumflex artery to the anterior papillary muscle[25]. The present study showed that, among patients with mitral regurgitation, 75% were mild, 23% were moderate, and 2% were severe, which is similar to previous reports[7,19]. However, the present study was not in patients with papillary muscle

infarction immediately after myocardial infarction. This meant that patients with significant papillary muscle dysfunction or severe mitral regurgitation early after myocardial infarction may not be included in this study.

The authors showed that papillary muscle infarction was associated with increased left ventricular volumes, more severe left ventricular dysfunction, higher LGE percentages, and more severe wall motion score. Similar to previous report[21], these findings indicate that patients with papillary muscle infarction had more myocardial damage than patients without papillary muscle infarction.

The present study also showed that patients with papillary muscle infarction had an increased risk of cardiac event. However, multivariate analysis revealed that papillary muscle infarction was not an independent predictor. Independent predictors for cardiac event were presence of mitral regurgitation, LVESVI, and the use of statins. This finding suggests that a larger area of infarction may be associated with increased risk of papillary muscle infarction and mitral regurgitation and is the major determinant of prognosis. LVESVI has been shown to be the most significant predictor of cardiac event in patients with coronary artery disease[26] and post myocardial infarction[27] and has been reported to be the most significant predictor for recovery of cardiac function after revascularization[28]. Besides common mechanisms of increased cardiovascular risk in post myocardial infarction patients, papillary muscle infarction may be associated with arrhythmogenesis of post myocardial infarction ventricular arrhythmia and may even lead to ventricular fibrillation[29]. Resection of scarred papillary muscles has been shown to improve outcomes after surgery for ventricular tachycardia[30]. The present result also demonstrated that the presence of mitral regurgitation was associated with an increased risk of cardiac events, which is consistent with previous study that demonstrated a prognostic significance of mild mitral regurgitation after myocardial infarction after adjustment for other factors[7].

The present study had several limitations. First, the authors studied patients who were referred for CMR for assessment of cardiac function and myocardial viability. This patient population may not be an accurate representation of most post myocardial infarction patients. More to the point, patients with acute myocardial infarction or recent myocardial infarction may not have been included. Second, although CMR can be used for assessment of mitral regurgitation, there are several other methods that can be used. Although the method used in this study is easy to use in clinical practice, it may not be the most accurate available technique. Quantitative measurement of mitral regurgitation by CMR may require the calculation of forward flow through the aorta and left ventricular volume during systole and diastole[11].

In conclusion, papillary muscle infarction is not an uncommon condition. Papillary muscle infarction was found to be associated with the increased risk of cardiac event in univariate analysis. In multivariate analysis, LVESVI and presence of mitral regurgitation were associated with adverse outcome.

What is already known on this topic?

Papillary muscle infarction occurs in the setting of myocardial infarction and can lead to papillary muscle dysfunction and mitral regurgitation. The prevalence was unknown, and the significance is difficult to determine.

What this study adds?

Prevalence of papillary muscle infarction in patients after myocardial infarction is 38.9%. It was found to be associated with more severe left ventricular dysfunction. The increased risk associated with papillary muscle infarction was mediated by more severe myocardial damage and mitral regurgitation.

Potential conflicts of interest

The authors declare no conflict of interest.

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