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Importance of papillary muscle infarction detected by cardiac magnetic resonance imaging in predicting cardiovascular events

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ABSTRACT

Background: Recent studies suggest that papillary muscle infarction (PMI) following recent myocardial infarction (MI) correlates with adverse cardiovascular outcomes. The purpose of this study is to determine the prevalence and prognostic significance of PMI by cardiac magnetic resonance (CMR) in a large cohort of patients.
Methods: Retrospective study of patients who underwent CMR between January 2007 and December 2009 were evaluated for the presence of PMI in one or both of the left ventricle papillary muscles. The primary outcome was a time to a combined endpoint of all-cause mortality and worsening heart failure. Secondary outcomes were time to individual components of the combined outcome.
Results: 419 patients were included in our analysis, 232 patients (55%) had ischemic cardiomyopathy. Patients were followed at six-month intervals for a median follow-up time of 3.7 (interquartile range (IQR): 1.6; 6.3) years after initial imaging. During this period 196 patients (46.8%) had a primary outcome and 92 patients (22%) died. PM infarct was identified in 204 (48.7%) patients with twice as many posteromedial (PRM) (27%) than anterolateral (ARL) lesions (11%) and a similar number with infarct in both (11%). There was no association between studied outcomes and the presence of PMI in either PRM or ARL PM. The presence of infarct in both PM was a predictor of both the primary outcome (HR 1.69, CI[1.01–2.86], $p < 0.049$.) and mortality (HR 1.69, CI[1.01–4.2], $p < 0.046$).
Conclusion: The presence of infarct in either papillary muscle was not associated with outcomes. However, infarct involving both papillary muscles was associated with worse outcomes.

1. Introduction

The left ventricle (LV) papillary muscles are essential components in mitral valve function and overall ventricle performance [1,2]. Papillary muscle infarct (PMI), most commonly from myocardial infarction (MI), but elicited as well in non-ischemic cardiomyopathies, is a marker for PM dysfunction contributive to mitral valve regurgitation (MR), LV systolic dysfunction, and ventricular dyssynchrony [3,4]. Also, fibrotic scarring in the PM can serve as a substrate for the development of reentry ventricular arrhythmias [5].

PMI following myocardial infarction was initially demonstrated from prior intra-operative and autopsy descriptive studies [6]. The prevalence of PM infarction from autopsy data was 10–25%, however, with the advent of myocardial tissue imaging by ultrasound, nuclear, and magnetic resonance, the prevalence is upwards to 40% and the in vivo pathological correlations more substantive [7,8]. Of the modern myocardial imaging modalities, cardiac magnetic resonance (CMR) offers the best tissue resolution and hence, demarcations of PM scar extent and borders [9].

Nonetheless, studies specific to PM evaluation by CMR are thus far limited and based on relatively small sample sizes. Furthermore, outside of recent myocardial infarction (MI) patients, few studies are linking CMR detected PMI and long-term cardiovascular outcomes [3,8,10,11].

Abbreviations: ARL, anterolateral; CI, confidence interval; CMR, cardiac magnetic resonance; HR, hazard ratio; ICM, ischemic cardiomyopathy; IQR, interquartile range; LGE, late gadolinium enhancement; LV, left ventricle; LVEF, left ventricular ejection fraction; MI, myocardial infarction; MR, mitral regurgitation; NICM, nonischemic cardiomyopathy; PM, papillary muscle; PRM, posteromedial; ROC, receiver operator characteristics; SSFP imaging, steady-state free precession imaging.

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Therefore, the purpose of this study was to investigate the prevalence of PMI in a broad population of patients referred for CMR and impact on long-term cardiovascular morbidity and mortality.

2. Materials and methods

This retrospective study of patients referred for CMR at our institution. Our study is approved by the institutional review board. Every patient enrolled in this study provided informed consent for inclusion of CMR, demographic, and outcomes data to the registry. There was no external funding used to support this work. The authors are responsible for the design and conduct of this study, all data analysis, drafting, editing of the paper and its final content.

2.1. Study population

Patients referred to a Brooklyn, New York community hospital CMR center between January 2007 and December 2009. In this analysis we included all patients over 18 years of age, who did not carry a diagnosis or referral for complex congenital heart disease, did not have a history or were scheduled for valve surgery and did not have significant arrhythmias during CMR exam. All included patients underwent gadolinium contrast enhanced imaging. Demographic and clinical data was obtained at the time of CMR via patient interview, notes from referring physicians and electronic medical record.

2.2. Magnetic resonance image protocol

All CMR studies were performed on a 1.5-T CMR system Magnetom Avanto™ (Siemens Healthcare®) using standard pulse sequences. Before contrast administration, short-axis steady-state free precession (SSFP) images covering the LV from the mitral valve annulus to the apex were obtained. Two, three and four chamber SSFP images were also acquired to visualize all 17 segments of the LV according to the American Heart Association imaging recommendation [12]. The CMR parameters of the cine SSFP sequence were as follows: bandwidth 125 kHz, flip angle 45°, repetition time to echo time 3.7/1.6 ms, a field of view 32 cm, image matrix 256 × 192, and slice thickness 8 mm. Delayed contrast enhancement images corresponding to positions of already obtained SSFP images were acquired using inversion recovery sequence 10 to 20 min after an intravenous bolus injection of 0.1 to 0.15 mmol/kg of gadopentetate dimeglumine (Magnevist®, Bayer Schering Pharma) with the inversion time varying from 300 to 400 ms, depending on the null point of healthy myocardium. In order to limit imaging artifacts (such as slow flow areas around and between double headed PM), we performed imaging of suspicious areas at different time points (ex 10–15–20 min) in addition to acquiring multiple images in the orthogonal planes.

2.3. Analysis of CMR study and definitions of the cardiomyopathy

Visual assessment of the images was conducted independently by two physicians with level 2 or higher CMR competency and blinded to the clinical data. Further delineation into ischemic and non-ischemic cardiomyopathies was made based on patient's history and review of available medical records. We defined non-ischemic cardiomyopathy (NICM) as left ventricular ejection fraction (LVEF) <50 and an the absence of stenosis ≥50% of left main or ≥70% of a major epicardial coronary artery on prior CA [12]. If no CA performed, then NICM was defined by CMR criteria which was defined as: mid-myocardial or epicardial hyperenhancement or hyperenhancement in a non-coronary artery distribution, or LVEF less than 50% in the absence of hyperenhancement.

2.4. Analysis of papillary muscle infarct

A presence of myocardial infarct was determined by the presence of LGE in the LV myocardium on delayed contrast enhancement sequences by visual assessment and designated as sub-endocardial, mid-myocardial, epicardial, or transmural [13,14] (Fig. 1). PM infarct was defined as the presence of LGE in either or both of the anterolateral and posteromedial papillary trunks [15,16]. Matching corresponding SSFP and delayed enhancement images were reviewed side by side to differentiate papillary muscle scar from the surrounding blood pool (Fig. 2). A presence of MR was assessed visually using SSFP long axis images.

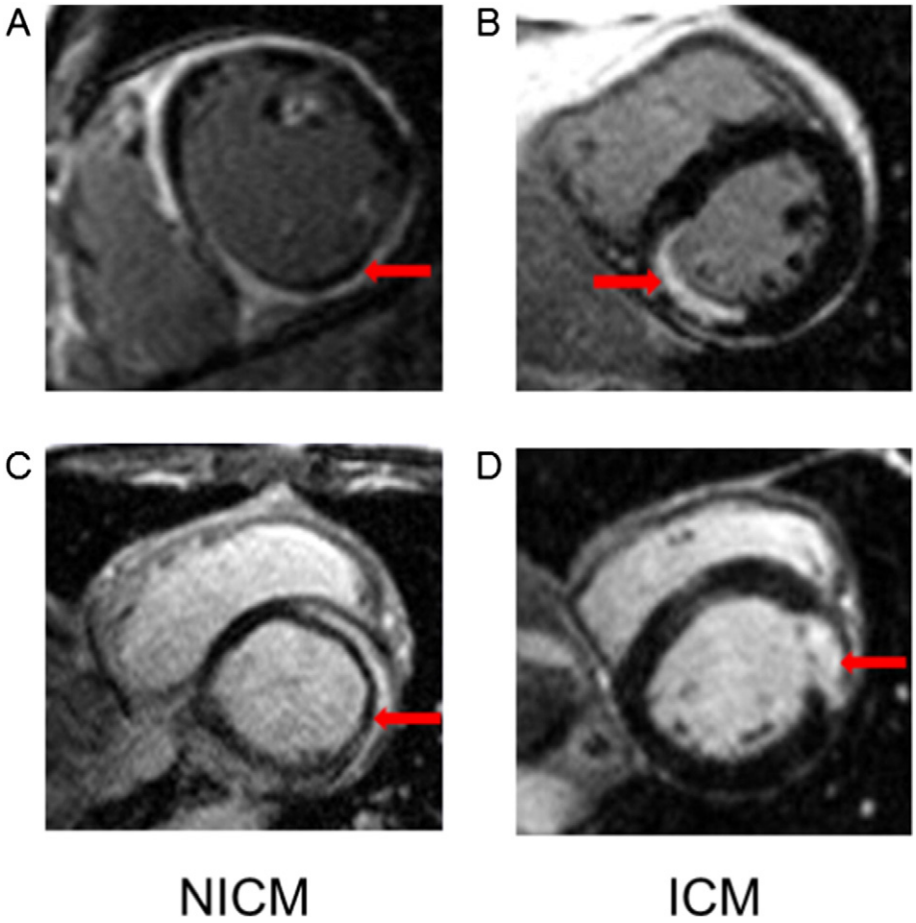

Fig. 1. Short-axis delayed enhancement CMR images with scar designated by red arrows. Nonischemic cardiomyopathy (NICM) is distinguished by epicardial (A) and mid-myocardial (C) scar pattern. Ischemic cardiomyopathy (ICM) is typically distinguished by subendocardial (B) and transmural (D) scar pattern.