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Detecting Unrecognized Myocardial Infarction: The Importance of Imaging

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Introduction

Prior studies have suggested that approximately half of all myocardial infarctions (MIs) are unrecognized and only diagnosed by Q waves on electrocardiogram (ECG) [1]. However, ECG is relatively insensitive for small MI or non-Q-wave MI. Late gadolinium-enhanced cardiac magnetic resonance imaging (CMR) quantifies scar or MI size as validated against histopathology in animal models of MI [2]. It correlates highly with levels of biomarkers in humans post-MI [3]. This technique is presently the most sensitive cardiac imaging technique available for sizing MI. It has been shown to be more sensitive than nuclear approaches, especially for smaller, non-Q-wave infarctions [4].

A recent study demonstrated the prognostic significance of identifying late gadolinium enhancement (LGE) as a marker of infarct scar in patients suspected of having underlying coronary artery disease (CAD) [5]. In this study, more than 20% of patients studied with suspected CAD but no known MI had LGE on CMR. LGE showed the strongest unadjusted association with major adverse cardiac events (MACE) and cardiac mortality, above ejection fraction, left ventricular (LV) end-diastolic and end-systolic index, segmental wall motion abnormality, noninvasive assessment of ischemia, and angiographic stenosis. A threshold effect was observed in which even very small scars (< 2% of LV mass) were associated with a more than sevenfold increase in MACE. This study suggested that any scar as demonstrated by LGE by CMR carries prognostic significance.

Aims

The aim of the present study was to examine the prevalence and prognostic significance of LGE in a cohort of patients with suspected CAD but without Q-wave MI by ECG. Thus, the authors aimed to demonstrate the significance of non-Q-wave MI as detected by LGE CMR.

Methods

Patients with suspected (and not known) CAD scheduled for elective x-ray coronary angiography were recruited into the study and thus had a high pretest likelihood of CAD. A previous history of MI was defined as a diagnostic ECG with or without elevated cardiac enzymes or chest pain and elevated enzymes. Exclusion criteria included prior revascularization, nonischemic myocardial disorders, a serious intercurrent illness, or a contraindication to CMR. One hundred eight-five patients were enrolled at two sites between 1998 and 2004. All enrolled patients underwent a questionnaire and a 12-lead ECG,

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and clinical follow-up was obtained annually. The primary end point was all-cause mortality, with cardiac mortality as a secondary end point.

CMR was performed in a standard fashion, including cine images and LGE images acquired 10–15 min after infusion of 0.15 mmol/kg of gadolinium-based contrast agents. Two blinded observers analyzed the CMR data, ECG, and coronary angiography. The presence and location of LGE were scored for transmural extent and segmental location and whether or not it was in a CAD or non-CAD-type of pattern. Non-Q-wave MI was defined as CAD-type LGE in patients without Q waves on ECG. Standard statistical analyses were performed.

Results

The patients' mean age was 60.4 years and LV ejection fraction was 59% ± 18%. The prevalence of non-Q-wave MI was 27% (50/185), whereas the prevalence of Q-wave MI was only 8%. Those with non-Q-wave MI were older, had more diabetes, a higher Framingham Risk Score, and a lower LV ejection fraction. Infarct size in those with non-Q-wave MI was small, averaging 8%±7% of the LV mass. Infarct size in those with Q waves on ECG was 14%±9% of the LV, including three without any LGE. The distribution of non-Q-wave MI and Q-wave MI was similar, approximately 40% left anterior descending, 50% right coronary artery, and 10% left circumflex artery. The prevalence of CAD in this population was 61%, but 96% in those with non-Q-wave MI (compared with 73% in those with Q-wave MI). Multivariate predictors of non-Q-wave MI included age, diabetes, and LV ejection fraction.

Follow-up averaged 2.2 years. Sixteen patients died (13 with non-Q-wave MI) and three had a nonfatal MI. Patients with non-Q-wave MI had a significantly reduced survival compared to those without an MI. Independent predictors of survival on multivariate analysis were LV ejection fraction and non-Q-wave MI (as detected by LGE CMR), even after adjusting for revascularization.

Discussion

This is the first study to systematically examine the prevalence and prognostic importance of non-Q-wave MI. Its presence predicted an 11-fold higher risk of death and a 17-fold higher risk of cardiac death compared to those without MI. These patients are sicker than those without MI as they were older, had more diabetes, and had a higher Framingham Risk Score. They also had more extensive CAD. Thus, non-Q-wave MI as identified by LGE CMR is a marker for those patients with CAD with particularly adverse outcomes. Part of the problem for these patients may be lack of appropriate medical therapy for underlying CAD in the absence of having ECG or other evidence of CAD. Study limitations include the fairly specific nature of the study population, namely those referred for symptoms or a positive stress test for coronary angiography and cannot be extrapolated to asymptomatic populations.

Comments

This is an important study that expands our understanding of the prevalence of unrecognized small MIs in patients with suspected CAD. The prevalence of 35% is strikingly high, as is the adverse prognosis in this patient subgroup compared to those without it, even after adjusting for other risk factors. Further studies are needed in different patient subgroups, such as diabetic patients and the elderly, to further understand the prevalence and significance of this finding.

Interestingly, a recent study of an elderly population in Iceland (> 70 years of age) demonstrated a prevalence of unrecognized MI of nearly 20% [6]. A study of LGE CMR added to a broader population such as the Framingham cohort might advance the understanding of the true prevalence of non-Q-wave MI unrecognized by ECG. Patients who have unrecognized MIs demonstrated by CMR should be candidates for secondary prevention therapy, even without a prior diagnosis of CAD. Cost/benefit analyses will be required to understand the efficacy of a screening approach in broader populations given the expense of CMR. With the recently recognized concern of nephrogenic systemic fibrosis, patients with stage IV or V chronic kidney disease are not candidates for gadolinium contrast and, thus, this kind of CMR study.

With this study comes the recognition of the limitations of the simple and cheap test that has been a mainstay of cardiology for decades—the electrocardiogram. Unfortunately, even in a patient population referred for x-ray angiography, the diagnostic accuracy of the ECG for small MIs is low. Whether infarct detection by CMR will become an important part of the armamentarium for assessing the extent and severity of underlying CAD and its adverse prognosis should be fodder for future study.

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