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Can late gadolinium enhancement by cardiovascular magnetic resonance identify coronary artery disease as the etiology of new onset congestive heart failure?

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Abstract

Background—New left ventricular systolic dysfunction affects 500,000 Americans and coronary artery disease (CAD) is responsible for two-thirds of cases. Identifying CAD has both prognostic and therapeutic implications. We evaluated the ability of late gadolinium enhancement (LGE) by cardiovascular magnetic resonance imaging (CMR) to detect CAD as the etiology of recent onset congestive heart failure (CHF).

Methods—CMR and LGE were performed in 26 patients with new onset left ventricular systolic dysfunction. All patients received an x-ray angiography for identification of CAD. Patients with an acute coronary syndrome with troponin I > 1.0 ng/ml or a history of CAD were excluded. The presence and distribution of LGE was evaluated.

Results—Significant coronary stenoses were present in 5 of 26 patients (19%). LGE in an infarct pattern was found in 2 of the 5 patients with CAD. Of the 21 patients without CAD, 2 had midwall enhancement but none had evidence of LGE in an infarct pattern.

Conclusions—When present, LGE in an infarct pattern suggests CAD as the etiology of new onset CHF. However, the absence of LGE does not exclude CAD as the underlying etiology. A small proportion of patients with a nonischemic cause of new onset CHF have LGE limited to the midwall.

Keywords

Ischemic cardiomyopathy; Magnetic resonance imaging; Nonischemic cardiomyopathy

Introduction

The presentation of new congestive heart failure (CHF) with evidence of left ventricular (LV) systolic dysfunction is a common clinical scenario, affecting close to 500,000 Americans each year. Discerning the etiology of LV dysfunction is often the first step in management. Underlying coronary artery disease (CAD) and ischemia are responsible for approximately two-thirds of all systolic dysfunction [1] and delineating it from nonischemic causes has essential therapeutic [2,3,4] and prognostic [5,6,7,8] implications. The electrocardiogram (ECG)[9], presence of anginal chest pain [10], and elevated troponins [11] are unable to accurately differentiate ischemic from nonischemic cardiomyopathy (NICM).

X-ray coronary angiography provides a definitive evaluation of luminal coronary obstruction but is currently only an American Heart Association (AHA) Class IIB indication for systolic dysfunction without angina and is associated with a 1.7% risk of major complication [12]. To avoid the risks and complications of an invasive evaluation, a noninvasive strategy would be an attractive alternative. In the face of myocardial dysfunction, the diagnostic accuracy of noninvasive imaging suffers. Sixty-four percent of resting echocardiograms in NICM patients demonstrate segmental LV wall motion abnormalities suggestive of CAD yet lack obstructive coronary lesions [13]. Dobutamine echocardiography has a sensitivity of 80–83% with a specificity of 71–96% for detecting CAD in dilated cardiomyopathy [14,15]. Thallium perfusion defects are present in 94% of NICM patients and 48% will have wall motion abnormalities [16]. Positron emission tomography demonstrates high sensitivity for CAD in dilated cardiomyopathy but poor specificity and visual diagnostic accuracy [17,18,19].

Cardiovascular magnetic resonance imaging (CMR) continues to enjoy an expanding role in the assessment of myocardial structure, function, and viability [20]. Late gadolinium enhancement (LGE) is a magnetic resonance technique with proven efficacy for the detection of myocardial infarction [21] and identification of secondary causes of heart failure [22], including myocarditis [23,24]. Two recent studies have employed these techniques to evaluate the etiology of CHF [25,26]. Neither study specifically addressed patients with newly diagnosed heart failure. We hypothesized that in patients with new heart failure the presence of LGE in an infarct pattern would correctly identify CAD as the underlying cause of their LV dysfunction.

Methods

Patient population

CMR studies were performed in twenty six patients prospectively enrolled with new onset CHF and LV systolic dysfunction. The study was carried out using a protocol approved by the Human Investigation Committee at the University of Virginia Health System and all participants signed informed consent. New onset CHF was defined as a clinical presentation consistent with the diagnosis, LV systolic dysfunction documented by echocardiography, and symptom duration of less than three months prior to an inpatient cardiology hospitalization or an outpatient cardiology consultation. Patients were excluded from enrollment for: age <18 years old, known CAD by previous history of positive stress test or x-ray coronary angiography

with any identified coronary stenosis, acute coronary syndrome with troponin I > 1.0 ng/ml, prior coronary arterial revascularization, significant valvular heart disease, pregnancy, or contraindication to cardiovascular magnetic resonance imaging. All patients underwent x-ray coronary angiography. Patients were categorized into two groups based on their coronary angiogram. The NICM group was defined as having mild luminal irregularities or normal coronary arteries and those with coronary lesions $\geq 50\%$ stenotic were classified as having ischemic cardiomyopathy (ICM).

Cardiovascular magnetic resonance imaging

Studies were performed on a Siemens Sonata ($n = 19$) or Avanto ($n = 7$) 1.5-T scanner (Siemens Medical Solutions, Erlangen, Germany). After scout imaging, stacked short axes from apex to base and long-axis left ventricular cine images were obtained using an electrocardiogram-gated, steady-state, free-precession (SSFP) pulse sequence. Imaging parameters were repetition time (TR) 3.1 ms, echo time (TE) 1.6 ms, flip angle 60° , field of view 315–400 mm, matrix 164×256 , and slice thickness 8 mm with 2 mm interslice gaps. Ten minutes after intravenous infusion of 0.15–0.20 mmol/kg gadolinium-DTPA, a steady-state free-precession cine scout series with variable inversion times was performed to determine the optimal inversion time to null normal myocardium (TR 2.6 ms, TE 1.1 ms, trigger pulse of 2, flip angle 50° , and slice thickness 8 mm). Late gadolinium enhancement images were acquired contiguously through the LV short axis and in the standard two, three, and four chamber views using a segmented inversion-recovery gradient-echo pulse sequence with the previously determined optimal inversion time (generally 250–320 ms). Parameters included TR 8.0 ms, TE 4.3 ms, flip angle 30° , field of view 315–400 mm, matrix 148×256 , and slice thickness 8 mm.

Data analysis

Left ventricular volumes, function, and mass were calculated by an investigator (BJS) blinded to the clinical and angiographic data from the short axis cine using semi-automated contours and dedicated analysis software (Argus, Siemens Medical Solutions, Malvern, PA). Another blinded investigator (CMK) interpreted all cardiovascular magnetic resonance images qualitatively for LGE blinded to the clinical and angiographic data. Each region of late enhancement was defined by its location within the 17 segment AHA tomographic model [27] and position in the myocardial wall (subendocardial, midwall, epicardial).

Statistical analysis

Continuous variables were compared using an unpaired t test and reported as mean $\pm$ STD. Noncontinuous-dichotomous variables were compared with a Fischer's exact test or where appropriate a Mann–Whitney Rank Sum test. Statistical significance was established with a < 0.05 , 2-tailed probability test.

Results

Twenty-eight patients met criteria for inclusion in this study. Two subjects were excluded; one due to claustrophobia and the other due to the inability to adequately breath hold. The remaining 26 patients were included in all analyses (Table 1).

Five of 26 patients (19%) had coronary stenosis of $\geq 50\%$ by angiography and were diagnosed as having an ICM. The remaining 21 of 26 (81%) patients were classified as NICM. All ICM patients had significant coronary lesions of their left anterior descending arteries and at least one other major coronary artery, with 4 of 5 having three-vessel CAD. The ICM group was older and 100% were smokers ($P < 0.05$ for both). There were no other significant differences in patient characteristics, CAD risk factors, beta blocker or angiotensin converting enzyme

inhibitor use, physical exam findings, or ECG findings at presentation between the NICM and ICM groups.

The study population included 73% men, 62% caucasians, 46% diabetics, 54% smokers, and 46% with known hypertension. The most common presenting symptom was dyspnea on exertion, in 77% of all patients. At presentation, 39% of all patients had chest pain and this was not different between groups (8 of 21, 38% in NICM and 2 of 5, 40% in ICM). As part of their initial hospital evaluation all 5 ICM and 13 of 21 NICM patients had troponins levels drawn. Detectable troponins <1.0 ng/ml, were identified in 31% (4 of 13) of the NICM and 20% (1 of 5) of the ICM patients. In the NICM group, 15% (2 of 13) had both chest pain and a mildly elevated troponin whereas the single ICM patient with a positive troponin did not have chest pain, and both ICM patients with chest pain had negative troponins.

The systolic blood pressure, diastolic blood pressure, and heart rate were similar in the 2 groups (135 ± 32 mmHg in NICM vs. 150 ± 49 mmHg in ICM), (87 ± 22 mmHg in NICM and 77 ± 27 mmHg in ICM), (92 ± 17 bpm in NICM vs. 91 ± 24 bpm in ICM), respectively ($P = \text{NS}$ for all). Electrocardiogram on admission showed sinus rhythm in 89% of all patients. All patients in the ICM group were in sinus rhythm and 3 of 21 (14%) in the NICM group had atrial fibrillation. Electrocardiographic evidence of left ventricular hypertrophy was documented by the admitting physician in 6 of 21 (29%) in the NICM patients and 1 of 5 in the ICM group ($P = \text{NS}$). Left bundle branch block was only seen in the NICM group with an incidence of 6 of 21 (29%).

Cardiovascular magnetic resonance imaging was performed 48 ± 30 days from symptom onset and 14 ± 23 days from coronary angiography. There was no difference in timing of imaging between groups. The mean end diastolic volume, end systolic volume, left ventricular mass, and ejection fraction for the entire cohort was 222 ± 81 ml, 169 ± 75 ml, 175 ± 52 g, and $26 \pm 11\%$, respectively and the 2 groups were similar in all volumetric indices (Table 2).

Fifteen percent ($n = 4$) of all subjects had LGE (Table 3). The presence of enhancement did not differentiate the 2 groups. Subendocardial or transmural late gadolinium enhancement was only present in the ICM group (2 of 5) (Fig 1). Only one of these two ICM with LGE had a measurable troponin. Midwall or epicardial late enhancement was present only in the NICM group (2 of 21) (Fig. 2). No patient in the NICM group had subendocardial or transmural enhancement.

Discussion

We investigated the ability of LGE presence and distribution to detect CAD in patients presenting with new systolic dysfunction without a history of CAD or evidence of an acute coronary syndrome and troponin >1.0 . In this study, when present, the distribution of enhancement was helpful in characterizing the etiology of the underlying cardiomyopathy as ischemic or nonischemic. However, the absence of LGE in an infarct pattern did not help discriminate between the groups as three of five patients with ICM showed no evidence of late enhancement. The inability to identify these patients is a limitation of the use of this technique alone in this clinical setting.

In the ICM group, only 2 of 5 patients demonstrated subendocardial or transmural LGE. Our patients presented soon after symptom onset. This study took place in the acute setting with CMR on average 48 days from the patient reported onset of symptoms and 14 days from coronary angiography. Soriano et al [26] performed CMR 1.9 ± 3.6 months from the time of coronary angiography and found subendocardial or transmural LGE in 21 of 26 (81%) in ICM patients. McCrohon et al [25] investigated chronic heart failure patients from a heart failure

clinic at an even longer interval of 2.2–3.4 years from angiography and showed a 100% prevalence of LGE in a CAD pattern.

The absence of late enhancement in our 3 patients with ICM may well signify the presence of hibernating myocardium [28,29]. Failure to revascularize dysfunctional, hibernating segments can lead to myocardial apoptosis [30]. Increased connective tissue deposition and subendocardial apoptosis has been demonstrated in chronic hibernating animal models [29]. This may account for the increasing prevalence of subendocardial and transmural LGE in the studies with a prolonged interval prior to imaging [25,26].

Subendocardial or transmural LGE is a known consequence of CAD and myocardial infarction [21]. This pattern was only present in the ICM group. Soriano et al [26] showed that 4 of 45 (9%) of their NICM patients had subendocardial or transmural LGE, which lowered their specificity to 91%. Similarly, McCrohon et al demonstrated a prevalence of 13% of subendocardial late enhancement in NICM. These findings have been previously explained by either recanalized coronary arteries post-thrombotic infarct or embolic infarction. The patients in our study did not demonstrate this phenomenon. One potential explanation for the difference between our study and prior investigations is again the length of time from diagnosis to imaging.

Heterogeneous midwall or subepicardial LGE has been described in myocarditis [23,24] and NICM [25]. This pattern was present in 2 of 21 patients in the NICM group. This is consistent with the 10–20% found in prior studies [25,26]. McCrohon et al [25] first described this pattern of distribution in dilated NICM. Soriano et al [26] showed that 3 of 26 (11%) patients with LV dysfunction and CAD had midwall LGE and suggested that it represented coexistent NICM and CAD. With troponin I > 1.0 ng/ml as an exclusion criterion, patients with acute myocarditis may well have been excluded from our study. In the acute setting, myocarditis will have a LGE prevalence in the midwall of 44–88% and generally will not involve the subendocardium [23,24].

Limitations

Our study evaluated this modality in a carefully selected patient population, in particular excluding troponin I > 1.0 ng/ml. The exclusion of a significantly elevated troponin is most likely responsible for the low prevalence of ischemic cardiomyopathy and acute myocarditis in this study. CAD accounts for two-thirds of new CHF cases [1] but was only present in 19% of our study. However, an elevated troponin is not exclusive to CAD and can be present in 20% of acute CHF and 34% of myocarditis [11]. The addition of a noninvasive coronary assessment by multi-detector computed tomography or CMR or an ischemic evaluation by stress CMR [31] to the LGE evaluation might have provided a more comprehensive and conclusive evaluation of CAD in new onset CHF. Additional tools in CMR are available for the detection of both myocarditis [23] and transient ischemic injury [32] that might have improved our diagnostic accuracy in this setting had they been used. Our study population is limited in size, especially the number of patients with the diagnosis of ischemic cardiomyopathy. However, the finding that the absence of LGE in new onset CHF does not exclude CAD is an important one and relevant to clinical practice.

Clinical implications

In clinical practice, many physicians feel compelled to exclude CAD in recent onset LV systolic dysfunction with x-ray coronary angiography. The risk of catheterization, though present, is low and there is often a concern regarding missing a potentially reversible cause of heart failure with noninvasive testing. The detection of CAD as the cause of dilated cardiomyopathy by thallium scintigraphy, exercise echocardiography, persantine thallium and dobutamine

echocardiography have a sensitivity and specificity range of 85–89% and 79–91%, respectively [33]. Recent evaluation of myocardial contrast echo to distinguish ischemic from NICM in acute CHF showed a sensitivity of 82% [34]. Thus, to date, noninvasive imaging has not had the accuracy necessary to obviate the need for x-ray angiography in this setting.

This study evaluates the ability of LGE alone to differentiate NICM from ICM. LGE alone was unable to identify 3 of 5 of ICM patients, all of whom had multivessel disease. This is of significant clinical import as the lack of LGE in the presence of multivessel CAD signifies predominantly viable myocardium with potential to benefit from revascularization [35]. When present, however, LGE in an infarct pattern in new onset CHF was a marker of CAD as the etiology.

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Abbreviations

CMR	Cardiovascular magnetic resonance imaging
ICM	Ischemic cardiomyopathy
LGE	Late gadolinium enhancement
NICM	Nonischemic cardiomyopathy

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Fig. 1. Segmented inversion-recovery gradient-echo image in a 2 chamber long-axis orientation with late gadolinium enhancement in an anterior and inferior subendocardial distribution (**black arrows**). This patient had an ejection fraction of 23% and x-ray angiography subsequently demonstrated an occluded left anterior descending artery and a 90% stenosed circumflex artery

Fig. 2. Segmented inversion-recovery gradient-echo image in a 2 chamber long-axis orientation with late gadolinium enhancement in the anterior midwall (**black arrow**). This patient had an ejection fraction of <10% and x-ray angiography subsequently demonstrated normal coronary arteries

Table 1

Clinical characteristics of the study group

Characteristics	Entire group	NICM	ICM	<i>P</i> value
No. of patients (%)	26 (100)	21 (81)	5 (19)	-
Age (yrs)	49 ± 14	46 ± 12	64 ± 14	<0.05
Males, <i>n</i> (%)	19 (73%)	15 (71%)	4 (80%)	1.0
Caucasians, <i>n</i> (%)	16 (62%)	13 (62%)	3 (60%)	1.0
Hypertension, <i>n</i> (%)	12 (46%)	9 (43%)	3 (60%)	0.63
Diabetes, <i>n</i> (%)	3 (12%)	1 (5%)	2 (40%)	0.08
Hyperlipidemia, <i>n</i> (%)	2 (8%)	2 (10%)	0	1.0
Tobacco use, <i>n</i> (%)	14 (54%)	9 (43%)	5 (100%)	<0.05

Table 2

CMR volumetric findings in the group as a whole and in the 2 subgroups

Characteristics	Entire cohort (n = 26)	NICM (n = 21)	ICM (n = 5)	P value
Symptoms to scan (days)	49 ± 30	49 ± 30	35 ± 26	0.34
CA ^a to CMR ^b (days)	14 ± 23	15 ± 24	12 ± 20	0.75
End diastolic Volume (ml)	231 ± 83	831 ± 83	183 ± 62	0.25
End systolic volume (ml)	169 ± 75	177 ± 78	137 ± 54	0.29
Stroke volume (ml)	53 ± 23	54 ± 25	47 ± 14	0.54
LV mass (g)	175 ± 52	177 ± 55	166 ± 39	0.69
EF (%)	25 ± 11	25 ± 11	28 ± 9	0.66

^aCA = Coronary Angiogram^bCMR = Cardiovascular Magnetic Resonance Imaging

Table 3

Presence of late gadolinium enhancement

LGE	Entire Group (<i>n</i> = 26)	NICM (<i>n</i> = 21)	ICM (<i>n</i> = 5)
Any LGE, <i>n</i>	4	2	2
Subendocardial or Transmural, <i>n</i>	2	0	2
Midwall or Epicardial, <i>n</i>	2	2	0

LGE = late gadolinium enhancement