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Contrast-Enhanced Cardiovascular Magnetic Resonance Imaging of Right Ventricular Infarction

Andreas Kumar, MD,*† Hassan Abdel-Aty, MBBCH, MSC,*† Ilka Kriedemann,† Jeanette Schulz-Menger, MD,† C. Michael Gross, MD,† Rainer Dietz, MD,† Matthias G. Friedrich, MD, FESC*†

Calgary, Alberta, Canada; and Berlin, Germany

OBJECTIVES We assessed the role of late enhancement cardiovascular magnetic resonance imaging (LE-CMR) for the diagnosis of right ventricular infarction (RVI).

BACKGROUND Right ventricular infarction occurs in about one-half of patients with inferior myocardial infarction (MI). It is associated with an unfavorable prognosis, but established methods often lack the diagnostic accuracy to detect it. Late enhancement cardiovascular magnetic resonance imaging accurately detects left ventricular MI.

METHODS Thirty-seven patients with acute inferior MI were included. To test for RVI, they prospectively underwent a physical examination, an electrocardiogram (ECG) for ST-segment elevation in the V_4r right precordial lead, and an echocardiogram. After coronary reperfusion, LE-CMR was performed for assessing presence and extent of late enhancement in the right ventricular (RV) wall. The LE-CMR data were compared with the other results; interobserver variability was assessed. The LE-CMR was repeated after 13 months.

RESULTS Late enhancement cardiovascular magnetic resonance imaging detected RVI in 21 of 37 (57%) patients with acute inferior MI. Interobserver variability was very good (kappa 0.83); physical exam was positive for RVI in 7 of 37 (19%) patients, V_4r ECG in 13 of 37 (35%) patients, and echocardiogram in 6 of 37 (16%) patients. The LE-CMR findings for RVI showed only mild agreement with findings for RVI on physical exam (kappa 0.30), V_4r ECG (kappa 0.38), and echocardiography (kappa 0.32). Irreversible injury of the RV persisted at 13 months (kappa 0.85).

CONCLUSIONS In patients with acute inferior MI, RVI is more frequently detected by LE-CMR than by current standard diagnostic techniques. Further CMR studies might allow for analyzing its clinical and prognostic relevance. (J Am Coll Cardiol 2006;48:1969–76) © 2006 by the American College of Cardiology Foundation

Right ventricular infarction (RVI) occurs in approximately 50% of patients with acute inferior myocardial infarction (MI) (1–4), but isolated forms have been reported as well. In a clinical setting, however, RVI are detected less often, although early and accurate detection of RVI might be necessary to identify patients at increased risk for complications and in-hospital mortality (4–8). Furthermore, RVI patients might require an intensive treatment regimen in terms of volume loading (9), a therapy potentially harmful in acute isolated left ventricular (LV) infarction.

"Late enhancement" cardiovascular magnetic resonance imaging (LE-CMR) can be performed safely in patients with acute coronary syndromes (10–12) and visualizes in vivo areas of necrosis in acute LV MI and fibrous scar in chronic LV MI (13–15). Its high spatial resolution allows for detection of very small irreversibly injured areas of the LV (15,16) and, as recently shown, small fibrous lesions in the right ventricular (RV) wall (17). Case reports suggested the ability of LE-CMR to detect RVI (18,19). However, the clinical role of contrast enhanced CMR imaging for the detection of RVI remains undefined. We hypothesized that RVI in acute inferior MI can be visualized by LE-CMR.

METHODS

Study design. We prospectively enrolled patients admitted to our emergency department with acute inferior MI, irrespective of presence or absence of RVI. The diagnosis of acute inferior MI was established on clinical grounds, enzyme changes, and ST elevation in at least 2 of the inferior leads II, III, and aVF of a 12-lead electrocardiogram (ECG). Patients with a history of previous MI were excluded. During the acute phase, 4 diagnostic tests for RVI were performed: physical examination, ECG with right precordial leads, echocardiography, and a LE-CMR examination (for details, see Table 1). The results were compared with agreement statistics. A follow-up study was performed in 27 patients. The internal ethical review board approved

From the *Stephenson CMR Centre at the Libin Cardiovascular Institute of Alberta, University of Calgary, Calgary, Alberta, Canada; and the †Franz-Volhard-Klinik, Helios Klinikum Berlin, Kardiologie, Charité Universitätsmedizin Campus Berlin-Buch, Humboldt Universität zu Berlin, Berlin, Germany. Drs. Kumar and Abdel-Aty are Canadian Institute of Health Research Strategic Training Fellows in TORCH (Tomorrow's Research Cardiovascular Health Professionals) at the University of Calgary; both are also supported by a non-restricted research grant from Siemens Medical Solutions, Erlangen, Germany. Data of this study were the subject of an oral presentation at the 2005 American College of Cardiology Annual Meeting in Orlando, Florida.

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Abbreviations and Acronyms

CMR = cardiovascular magnetic resonance imaging
ECG = electrocardiogram
LE-CMR = late enhancement cardiovascular magnetic resonance imaging
LV = left ventricle/ventricular
MI = myocardial infarction
RCA = right coronary artery
RV = right ventricle/ventricular
RVI = right ventricular infarction

the study and written informed consent was obtained from all patients.

Non-CMR diagnostic tests. The admitting physician initially evaluated the patients and entered physical findings into a standardized form. Clinical evidence was rated positive for RVI if, on physical examination, the patient revealed a triad of arterial hypotension (systolic blood pressure ≤90 mm Hg), clear lung fields by auscultation, and distended jugular veins (20). All patients obtained an ECG with 6 right precordial leads before revascularization. Those ECGs were photocopied and interpreted by an observer (I.K.) who was blinded to all other results. Right ventricular infarction was rated present by ECG criteria, if the patient had ST-segment elevation of ≥0.1 mV in the V_4r right precordial lead.

Two-dimensional echocardiography was performed with a commercially available system (Acuson Sequoia, Siemens Medical Solutions, Erlangen, Germany), with a 3.2-MHz transducer. The LV and RV chambers were imaged in standard apical and parasternal long- and short-axis views. Right ventricular free wall motion was classified as normal or abnormal; the latter comprised hypokinesis, akinesis, and dyskinesis. Right ventricular dilatation and abnormal interventricular septal motion were assessed qualitatively. Right ventricular infarction was rated present if any of these criteria was present. X-ray coronary angiography was performed on a standard angiography suite (Hicor, Siemens Medical Solutions) in all patients. One observer unaware of the CMR data (M.G.) evaluated the angiographic studies for the infarct-related artery and culprit lesion, on the basis of the location of ECG changes during chest pain and the angiographic characteristics of the lesion (21).

CMR. After reperfusion, the patients were studied on a 1.5-T system (Signa CV/*i*, General Electric, Cleveland, Ohio; or Sonata, Siemens). During CMR, ECG, respiration, pulse, and blood pressure of the patients were monitored. Localization was performed with breath-hold real-time and steady-state free precession images of true anatomical cardiac axes. Systolic contrast-enhanced inversion-recovery gradient-echo images ("late enhancement": repetition time 7.1 ms, echo time 3.1 ms, inversion time individually optimized to 200 to 300 ms to null remote myocardium, matrix 256 × 192, field of view 32 to 35 cm, slice thickness 10 mm) were obtained in a short-axis stack covering the RV completely (n = 24) or with 3 short axes

Table 1. Patient Characteristics

Parameter	All Patients	LE-CMR RVI Positive	LE-CMR RVI Negative	p Value
n	37	21	16	
Age (yrs)	53.8 ± 11.1	55.3 ± 12.2	51.8 ± 9.6	0.346
Gender, M/F	28/9	14/7	14/2	
Body mass index	26.8 ± 3.5	26.5 ± 3.8	27.1 ± 3.1	0.605
Culprit lesion*				
LCX	6	0	6	**0.002**†
RCA				
Total	30	21	9	**0.002**†
Proximal	14	11	3	0.346
Mid	10	7	3	1.0
Distal	6	3	3	0.240
Perfusion pattern				
Dominant right	25	17	8	**0.049**†
Intermediate	8	5	3	0.221
Dominant left	4	1	3	0.181
STEMI	35	21	14	0.100
CK max (U/I)	2,100 ± 3,220	2,627 ± 4,129	1,639 ± 1,250	0.310
LVEF‡	52.3 ± 10.1	51.2 ± 9.9	54.4 ± 10.6	0.440
Time to reperfusion (h)§	8.8 ± 5.4	10.0 ± 5.3	7.3 ± 5.2	0.170
Time—symptoms to ECG (h)§	7.8 ± 5.5	9.2 ± 5.6	6.0 ± 5.0	0.095
Time—symptoms to CMR (days)§	2.9 ± 1.6	3.00 ± 1.71	2.9 ± 1.6	0.912
Time—echocardiography to CMR (days)	1.97 ± 1.96	2.10 ± 2.08	1.81 ± 1.87	0.665
Time—ECG to CMR (days)	2.54 ± 1.63	2.42 ± 1.68	2.70 ± 1.59	0.607
Stay at ICU (days)	3.24 ± 1.40	3.76 ± 1.5	2.56 ± 0.89	**0.005**†

All values are mean ± SD. The p values were calculated with the Mann-Whitney and chi-square tests comparing the late enhancement cardiovascular magnetic resonance imaging (LE-CMR) right ventricular infraction (RVI) positive versus LE-CMR RVI negative subgroups. *In 1 patient, no culprit lesion could be defined at coronary angiography after successful thrombolysis. †Significant p-values. ‡Left ventricular ejection fraction (LVEF) values were measured by echocardiography or CMR with different methods in 35 patients. §The exact time of onset of symptoms could not be determined in 4 patients who were not included in this analysis.

CK = creatine kinase; ECG = electrocardiogram; ICU = intensive care unit; LCX = left circumflex coronary artery; RCA = right coronary artery; STEMI = ST-segment–elevation myocardial infarction.

(n = 13) 10 min after intravenous injection of 0.2 mmol/kg Gadolinium-DTPA (Magnevist, Schering, Germany). Functional and visual signal analysis was performed on an off-line workstation with validated software (MASS Medis, Leiden, the Netherlands). Infarcts were assessed on 2 separate days by 2 separate observers (A.K., H.A.A.). Both were blinded to each other's results, the results of all other diagnostic tests, and the functional analysis.

The area of late enhancement was first identified visually and the study was rated RVI positive, if the late enhancement extended from the inferior LV myocardium or the inferior interventricular septum into the RV free wall in any 1 or more of the CMR images (2,3). Both observers were asked to rate "positive" or "negative" for RVI. In cases of disagreement, agreement was reached by consensus. Regions of interest were drawn within the area of late enhancement (excluding areas of no reflow), in the remote myocardium (remote), and in the air outside the chest (noise) to measure signal intensities (SI) and calculate the contrast-to-noise ratio (CNR) with the following formula:

$$\mathrm{CNR} = \left[\text{mean SI}(\text{late enhancement}) - \text{mean SI}(\text{remote})\right] / \text{mean SI}(\text{noise})$$

In a second step, the amount of infarcted RV myocardium was quantified. On the basis of the work by Isner et al. (3). Three short-axis images (1 basal, 1 midventricular, and 1 apical) were divided into 4 RV segments per slice (12-segment model of the RV), and segments with late enhancement were counted (3). The RVIs were categorized as "small" when 4 or fewer segments were involved and "large" when more than 4 segments showed late enhancement. In a third step, more than 4 months after assessing late enhancement, both observers performed a consensus evaluation of all complete short-axis cine CMR studies for presence or absence of RV wall motion abnormality. Those data were compared with LE-CMR findings and echocardiography.

In the absence of a single accepted gold standard to identify RVI in vivo, we used an "aggregate standard" for analysis, taking into consideration the information provided by physical examination, ECG, and echocardiography. By definition, the aggregate standard was considered positive in patients who demonstrated RVI by at least 1 non-CMR method of assessment, and it was considered negative only in those patients whose non-CMR methods of assessment were all negative for RVI.

Patients were re-examined with a similar CMR protocol in the chronic phase several months after the acute infarction to assess LE-CMR findings. In addition, RV end-diastolic volumes and ejection fractions were measured in the chronic phase and correlated with LE-CMR findings in the acute phase.

Statistical analysis. Values are presented as mean ± SD. A p value <0.05 was considered significant. Statistical analysis was performed with commercially available software (SPSS 11.0 for Macintosh, SPSS, Chicago, Illinois). All statistical tests were 2-tailed. Agreement between methods and inter-observer agreement was measured with Kappa statistics. Continuous variables were compared with the paired *t* test when normally distributed and the Mann-Whitney *U* test when not normally distributed. Non-continuous data were compared with the chi-square test.

RESULTS

Forty-two consecutive patients with acute inferior ST-segment elevation MI were initially included, but 5 patients had to be excluded because of insufficient image quality on the echocardiogram (n = 4) or arrhythmia causing ECG trigger problems during the CMR examination (n = 1). The demographic characteristics of the study population (n = 37) are presented in Table 1. Reperfusion therapy (thrombolysis and/or percutaneous coronary intervention) was successful in 36 of 37 patients. Cardiovascular magnetic resonance imaging was performed without complications in all patients 2.9 ± 1.6 days after onset of symptoms. Late enhancement was readily visualized in 36 of 37 (97%) patients with a contrast-to-noise ratio of 6.5 ± 3.8. Late enhancement cardiovascular magnetic resonance imaging was positive for RVI in 21 of 37 patients (59%). The 2 blinded CMR observers reached independent agreement on presence or absence of RVI in 34 patients, and consensus reading was required in 3 patients (kappa value for inter-observer agreement: 0.83). Eight of 21 patients had a small RVI involving 4 segments or less, and 13 had a large RVI. Time to reperfusion, maximum creatine kinase elevation, and LV ejection fraction did not differ significantly between the RVI positive and RVI negative groups (Table 1).

An overview of the results of the LE-CMR study compared with the results of ECG, physical exam, and echocardiography is given in Table 2. Figures 1 and 2 show examples of patients with different combinations of positive or negative RVI findings.

Comparison of physical examination and LE-CMR. On physical examination, the triad of hypotension, distended neck veins, and clear lung sounds was observed in 7 of 37 (19%) patients; all those were positive for RVI on the LE-CMR study. Three of these patients had a large RVI

Table 2. Overview of the Results of the Conventional Diagnostic Tests, Compared With the Results of LE-CMR

	LE-CMR RVI + (n = 21)	LE-CMR RVI − (n = 16)	Kappa
Physical examination RVI −	14	16	0.302
Physical examination RVI +	7	0	
ECG RVI −	10	14	0.376*
ECG RVI +	11	2	
Echocardiography RVI −	16	15	0.323
Echocardiography RVI +	5	1	
Aggregate standard† −	5	14	0.623
Aggregate standard† +	16	2	

All numbers are absolute numbers of patients. *Kappa was 0.49 when the analysis was restricted to electrocardiograms (ECGs) obtained within 10 h after onset of symptoms. †For definition of the aggregate standard, see Methods section of the text. + = positive; − = negative; other abbreviations as in Table 1.

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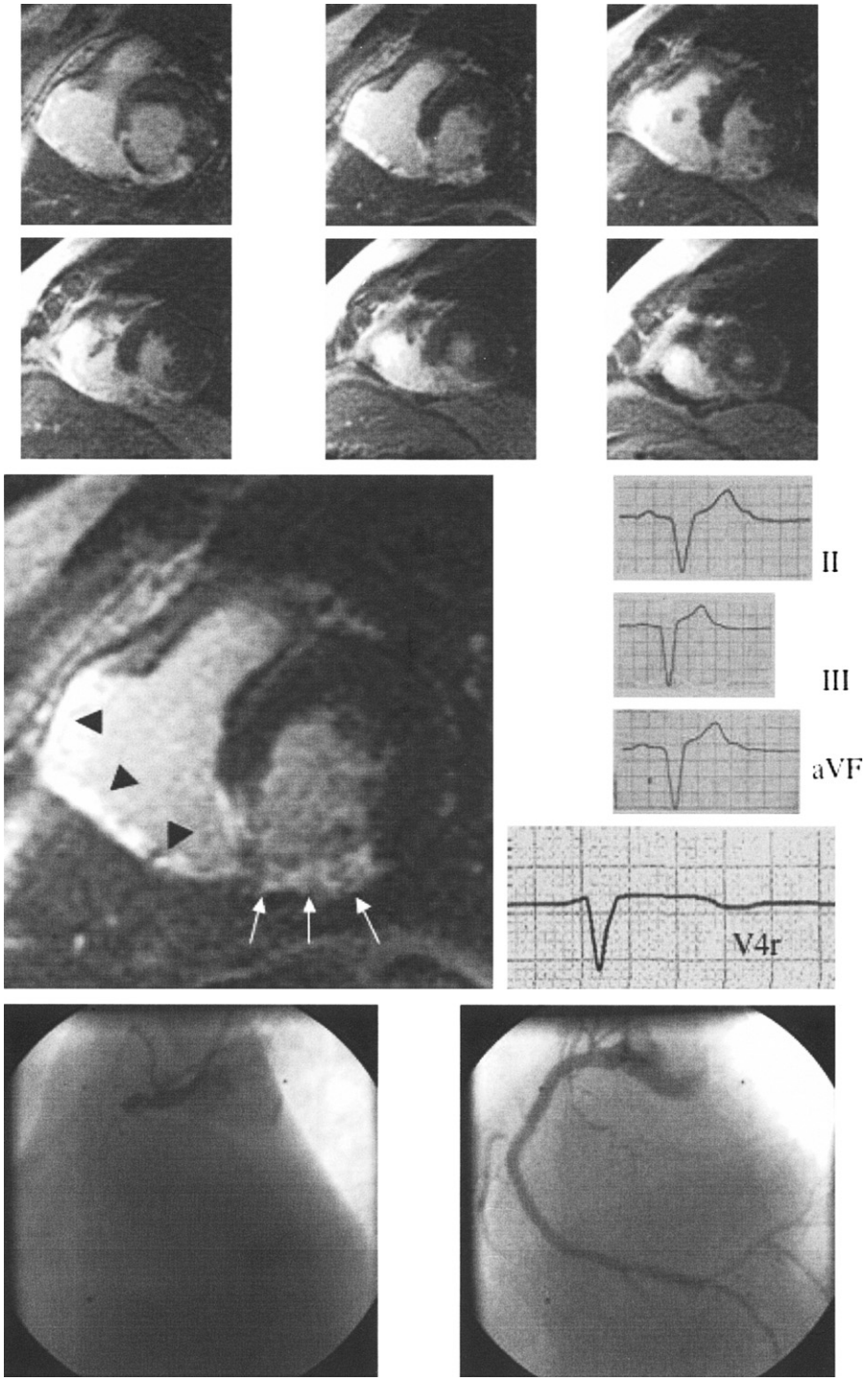

Figure 1. Patient with acute inferior and right ventricular (RV) infarction on late enhancement cardiovascular magnetic resonance imaging (LE-CMR). **(Upper panels)** Short-axis LE-CMR images showing contrast enhancement of the RV wall. **(Middle panels, left)** Enlarged short-axis view with infarction of the RV wall **(black arrowheads)** and the inferior left ventricle **(white arrows)**. **(Middle panels, right)** Electrocardiogram with ST-segment elevation in V_4r. **(Lower panels)** Culprit right coronary artery lesion in a right dominant perfusion pattern before **(left)** and after **(right)** angioplasty. Echocardiography revealed RV hypokinesis and dilatation.

and 4 had a small RVI by LE-CMR. Of 30 patients who were not positive for RVI on physical examination, 14 were still positive on LE-CMR (Table 2). The kappa test showed mild agreement between the results of physical examination and LE-CMR (kappa = 0.302).

Comparison of ST-segment elevation in V_4r and LE-CMR. Thirteen of 37 (35%) patients had ST-segment elevation of at least 1 mm in V_4r, including 1 patient with left bundle branch block; 11 of those 13 had a LE-CMR study positive for RVI. Of the 24 patients negative for RVI by

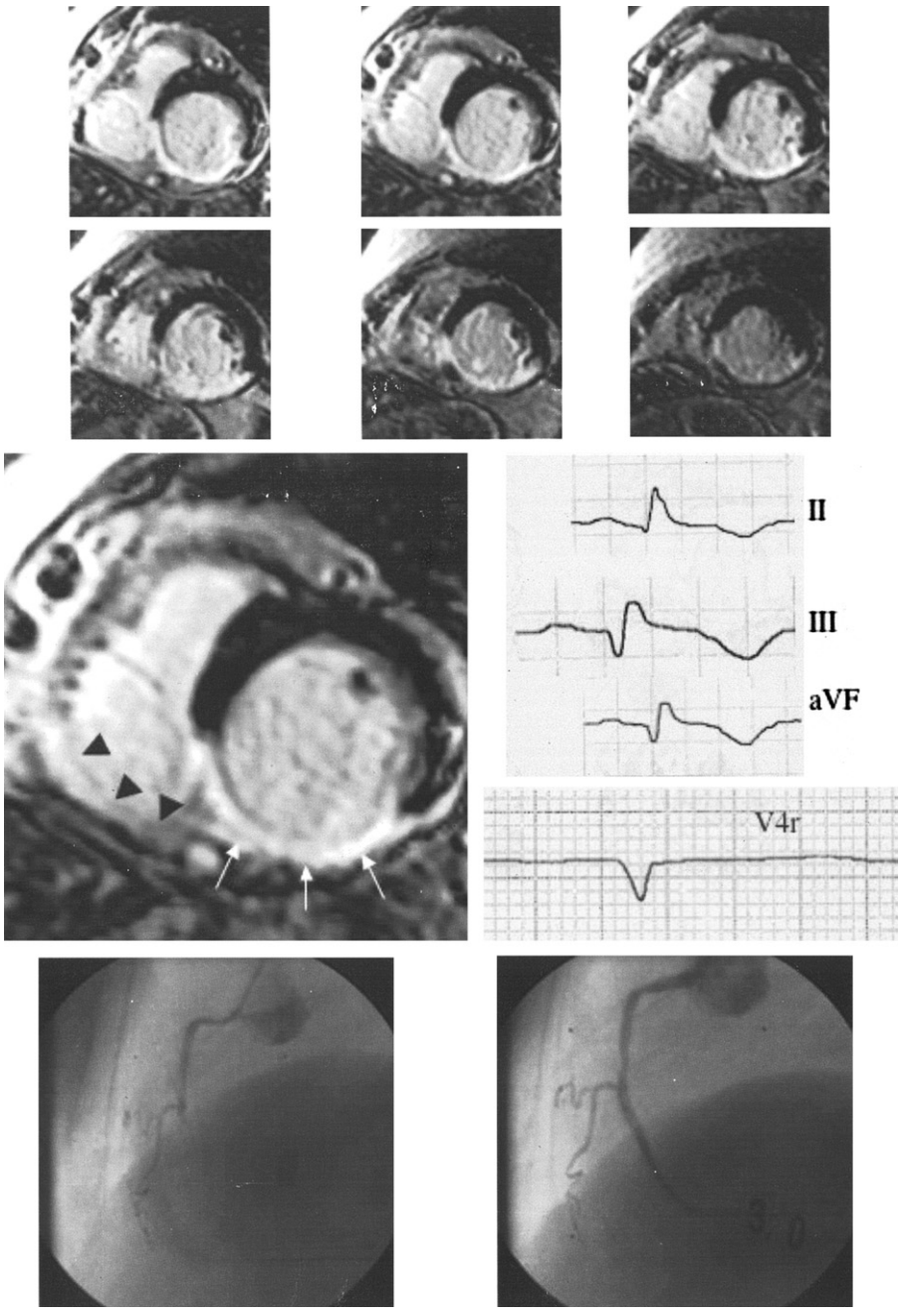

Figure 2. Patient with acute inferior infarction and right ventricular (RV) involvement on late enhancement cardiovascular magnetic resonance imaging (LE-CMR). **(Upper panels)** Short-axis LE-CMR images showing contrast enhancement of the RV wall. **(Middle panel, left)** Enlarged short-axis view with infarction of the RV wall **(black arrowheads)** and the inferior left ventricle **(white arrows)**. **(Middle panels, right)** Electrocardiogram showing absence of ST-segment elevation in V_4r. **(Lower panels)** Culprit right coronary artery lesion in a right dominant perfusion pattern before **(left)** and after **(right)** angioplasty. The echocardiogram showed no evidence of RV infarction.

ECG criteria, 10 had a positive LE-CMR study (Table 2). Time from onset of symptoms to recording of the ECG did not differ significantly between the LE-CMR RVI positive and negative groups (Table 1). In those 11 patients with both positive ECG and positive LE-CMR, 6 had a small RVI and 5 had a large RVI. There was a mild agreement between the RVI results of V_4r ECGs and LE-CMR (kappa = 0.38). A better agreement was found when the analysis was restricted to a subgroup of patients who had their ECGs obtained within ≤10 h after onset of chest pain (kappa = 0.49).

Comparison of echocardiography and LE-CMR. Echocardiography diagnosed RVI in 6 patients (hypokinesis of the RV free wall [n = 2], hypokinesis in conjunction with RV dilatation [n = 4]), and LE-CMR was positive in 5 of those. All of those 5 patients had a large RVI by LE-CMR, involving more than 4 segments of RV myocardium. There was a mild agreement between echocardiography and LE-

CMR findings for RVI (kappa = 0.323). Echocardiography was negative in all 8 patients with a small RVI by LE-CMR criteria.

Comparison of LE-CMR to the aggregate standard. The results of the LE-CMR study showed moderate agreement with the results of the combined non-CMR tests, the aggregate standard (kappa = 0.623). There were 5 patients in whom LE-CMR was the only method to diagnose RVI; in contrast, there were 2 patients who were positive by non-CMR methods (ECG [n = 1], ECG and echocardiography [n = 1]) but negative on LE-CMR.

Comparison of the non-CMR methods with small and large RVI, as defined by LE-CMR. The detailed overview of these data is given in Table 3. There was no significant correlation between LE-CMR–defined RVI extent and the results of physical exam or ECG; however, the correlation between RVI extent on LE-CMR and echocardiography findings for RVI was significant ($p = 0.044$): echocardiography was negative in all patients with small RVI (0 of 8), and it was positive in 5 of 13 (38%) patients with large RVI. Four of the 8 patients with small RVI developed findings consistent with RV failure on physical exam.

CMR wall motion analysis. Of 14 patients with a complete short-axis cine package study, 9 were LE-CMR negative and 5 were LE-CMR positive; 11 had a RV wall motion abnormality. All patients with RVI by LE-CMR also had a RV wall motion abnormality, but of 9 patients without RVI by LE-CMR, 6 still had a RV wall motion abnormality. The CMR wall motion data did not correlate significantly with the data obtained from echocardiography.

X-ray coronary angiography data. All 21 patients with RVI detected by LE-CMR had culprit lesions in the right coronary artery (RCA), as opposed to 9 of 16 patients who were LE-CMR RVI negative ($p < 0.05$). Of the 21 patients with RVI on LE-CMR, 11 (52%) had a proximal RCA lesion, 7 had a mid RCA lesion, and 3 had a distal RCA lesion; 17 of 21 (81%) had a right dominant perfusion pattern as compared with only 8 of 18 (50%) in the LE-CMR RVI negative group ($p < 0.05$). All 6 patients with a culprit lesion in the left circumflex artery did not have RVI on LE-CMR. No culprit lesion could be identified in 1 patient after successful thrombolysis (Table1).

Table 3. Results of the Non-CMR Tests of Patients With Small Versus Large Right Ventricular Infarcts, as Defined by LE-CMR

	LE-CMR Small RVI (n = 8)	LE-CMR Large RVI (n = 13)	p Value for Correlation*
Physical examination RVI −	4	10	0.204
Physical examination RVI +	4	3	
ECG RVI −	2	8	0.104
ECG RVI +	6	5	
Echocardiography RVI −	8	8	**0.044**†
Echocardiography RVI +	0	5	

*Pearson chi-square test. †Significant p values.
Abbreviations as in Tables 1 and 2.

Follow-up data. At 13 ± 9 months after the acute event, LE-CMR findings for irreversible RV injury persisted in 25 of 27 (93%) of the patients (kappa = 0.85). The LE-CMR RVI positive and negative groups did not differ in their RV end-diastolic volumes and RV ejection fraction.

DISCUSSION

This study was performed to test the feasibility of LE-CMR to diagnose RVI. The results might be summarized as follows:

- Late enhancement CMR can diagnose RVI with very good interobserver variability.
- Findings consistent with RVI were more frequently observed on LE-CMR than on ECG, physical examination, or echocardiography.
- Late enhancement cardiovascular magnetic resonance imaging findings for irreversible injury of the RV in the acute phase persisted at 13 months of follow-up.

The LE-CMR findings for RVI showed mild agreement with findings consistent with RVI on physical exam, ECG, and echocardiography. Interesting discrepancies were found between the LE-CMR and the non-CMR methods.

As opposed to all other investigated methods, LE-CMR specifically diagnoses irreversible injury and therefore infarction per se (13), whereas all other methods investigate other facets of pathophysiology.

Electrocardiography measures ischemic currents directed toward the RV wall, which can be a result of ischemia as well as infarction; echocardiography examines wall motion abnormalities due to RV failure, which might be caused by irreversible as well as reversible RV injury; the physical exam relies on hemodynamic consequences of a failing RV, which might be due to reversible as well as irreversible RV injury or might even be secondary to severe LV failure. One might therefore argue that LE-CMR seems to be the most specific test for irreversible injury of the RV and therefore the most specific test to diagnose true infarction of the RV.

There were 5 patients with LE-CMR findings consistent with RVI (large RVI [n = 2], small [n = 3]) but in whom all other methods indicated the absence of RVI. For this discrepancy, two explanations seem plausible:

- Late enhancement CMR was false positive in these patients, or
- The non-CMR methods were false negative, especially in smaller RV infarcts.

By defining positive RVI as extending from the inferior LV wall, a feature described on histopathology, we aimed at limiting false positive studies due to partial volume effects with epicardial fat tissue. The power of LE-CMR to assess irreversible injury of the RV wall was recently shown in the context of arrhythmogenic right ventricular cardiomyopathy (17). Furthermore, all of these 5 patients had culprit lesions of the proximal RCA, making RVI probable (1,22). We

therefore believe that false positive LE-CMR studies are the less likely of the two explanations.

The possibility that the infarcts were too small to be diagnosed by other methods, with non-CMR methods being false negative, seems to be more plausible, for the following reasons: 3 of the 5 LE-CMR positive yet otherwise undetected RVI were small, involving a maximum of 4 segments of RV myocardium or less; 2 of those 5 patients had their ECG recorded more than 10 h after onset of chest pain; and ECG changes for RV ischemia have been shown to vanish frequently after more than 10 h after onset of chest pain (23). One patient had stuttering chest pain. Therefore, the ECG findings might have been "false" negative. Overall, the time from onset of symptoms to ECG was rather long in our population (7.8 ± 5.5 h).

The echocardiogram relies on wall motion abnormalities. However, RV wall motion abnormalities in the setting of acute inferior LV infarction evolve from the inferior RV wall (2,3), an area that often is difficult to visualize on echocardiography. Some echocardiograms might therefore be read false negative, especially when the underlying infarcts are small.

Overall, the results and these considerations suggest that LE-CMR might be more sensitive to diagnose RVI than other tests. However, in the absence of a true gold standard (e.g., histopathology), verification of this statement requires further investigation. The good interobserver variability and persistence of LE-CMR findings at long-term suggest that LE-CMR is a robust test for the diagnosis of RVI.

Clinical implications. This is a feasibility study not designed to assess clinical or prognostic impact. However, some potential scenarios can be imagined where LE-CMR could be helpful: it could for example fill a diagnostic gap in patients who present with inferior MI and in whom ECG changes for RVI have vanished because of late presentation and the echocardiogram is non-diagnostic or negative. This situation was the case in 24% (5 of 21) of RVI patients in our study population, even after 4 patients had been excluded at study entry owing to an insufficient echocardiography window. The fact that 5 of 16 patients with RVI on LE-CMR and not diagnosed by echocardiography decompensated and developed clinically overt RV failure supports the hypothesis that LE-CMR could be of clinical value. Larger and appropriately designed studies are necessary to assess whether or not RVI, diagnosed by LE-CMR only, are of prognostic significance. Although the intensive care physicians were not systematically blinded to the LE-CMR results, it is important to note that LE-CMR RVI positive patients spent significantly more time on the intensive care unit than LE-CMR RVI negative patients (Table 1). This could indicate a clinical relevance of the LE-CMR findings.

Study limitations. We achieved a pixel size of 1.7 × 1.3 mm, which—in systole—covers the 5-mm-thick RV wall with 3 to 4 pixels (24), and this together with the lack of full coverage of the RV with LE-CMR in some patients might have led to "false negative" results. We believe, however, that this did not significantly affect our results, because we observed a relatively high prevalence of RVI.

We cannot exclude false positive results due to partial volume effects with epicardial fat signal. However, on the basis of autopsy reports, we defined RVI by a continuous extension of the myocardial injury from the inferior LV (not affected by partial volume effects) to the RV, making a relevant bias less likely.

Our CMR data were obtained in reperfused infarcts, and thus, the results and conclusion might not be valid for non-reperfused infarcts. The sample size was small with n = 37.

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Reprint requests and correspondence: Dr. Matthias G. Friedrich, Department of Cardiac Sciences, University of Calgary, Stephenson Cardiovascular MR Centre, Foothills Medical Centre, Suite 0700-SSB, 1403–29th Street NW, Calgary, Alberta, T2N 2T9, Canada. E-mail: matthias.friedrich@ucalgary.ca.

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