Right Ventricular Ischemic Injury in Patients With Acute ST-Segment Elevation Myocardial Infarction
Characterization With Cardiovascular Magnetic Resonance

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Background—Experimental data show that the right ventricle (RV) is more resistant to ischemia than the left ventricle. To date, limited data are available in humans because of the difficulty of discriminating reversible from irreversible ischemic damage. We sought to characterize RV ischemic injury in patients with reperfused myocardial infarction using cardiovascular magnetic resonance.

Methods and Results—In 3 tertiary centers, 242 consecutive patients with reperfused acute ST-segment elevation myocardial infarction were studied with cardiovascular magnetic resonance at 1 week and 4 months after myocardial infarction. T2-weighted and postcontrast cardiovascular magnetic resonance scans were used to depict myocardial edema and late gadolinium enhancement, respectively. Early after infarction, RV edema was common (51% of patients), often associated with late gadolinium enhancement (31% of patients). Remarkably, RV edema and late gadolinium enhancement were found in 33% and 12% of anterior left ventricular infarcts, respectively. Baseline regional and global RV functions were inversely related to the presence and extent of RV edema and RV late gadolinium enhancement. At follow-up, a significant decrease in frequency (25/242 patients; 10%) and extent of RV late gadolinium enhancement was observed (P<0.001). With the use of multivariable analysis, the presence of RV edema was an independent predictor of RV global function improvement during follow-up (β-coefficient=0.221, P=0.003).

Conclusions—Early postinfarction RV ischemic injury is common and is characterized by the presence of myocardial edema, late gadolinium enhancement, and functional abnormalities. RV injury is not limited to inferior infarcts but is commonly found in anterior infarcts as well. Cardiovascular magnetic resonance findings suggest reversibility of acute RV dysfunction with limited permanent myocardial damage at 4-month follow-up. (Circulation. 2010;122:1405-1412.)

Key Words: magnetic resonance imaging ■ myocardial infarction ■ right ventricle

Although autopsy studies1–3 have demonstrated frequent right ventricular (RV) infarction in patients dying of acute myocardial infarction (MI), the occurrence and impact of RV ischemic injury in patients who survive acute MI are less well known. Several clinical studies have shown that in patients with reperfused left ventricular (LV) MI and RV ischemic injury, RV function recovers more rapidly and better than LV function despite similar initial dysfunction.4–6 These observations, along with data on experimentally induced RV ischemia,7,8 have led us to hypothesize that acute postinfarct RV dysfunction is the expression of viable rather than irreversibly damaged myocardium. A better understanding of the mechanisms of RV ischemic dysfunction in patients with acute MI is crucial because impaired RV recovery is associated with persistent hemodynamic compromise and a high mortality rate.4,9 Unfortunately, to date, our understanding of the pathophysiological mechanisms of RV ischemic dysfunction has been hampered by lack of an accurate, noninvasive diagnostic technique enabling full characterization of RV ischemic injury.

Clinical Perspective on p 1412

Cardiovascular magnetic resonance (CMR) allows accurate characterization of ischemic myocardial injury. T2-weighted, short-inversion time, inversion-recovery imaging (T2w imaging) and late gadolinium enhancement (LGE)
imaging enable visualization of reversibly and irreversibly damaged myocardium, respectively.\textsuperscript{10,11} Recent studies\textsuperscript{12,13} have shown that LGE imaging is accurate and reproducible for detection of acute and chronic RV infarction. To date, a combined T2w imaging/LGE imaging CMR approach, a well-accepted imaging tool to study jeopardized LV myocardium, has never been used to investigate RV ischemic injury. Using a comprehensive CMR approach in a large cohort of consecutive acute MI patients obtained in 3 tertiary hospitals, we sought to characterize the pattern of RV ischemic injury and its impact on RV function early after infarction and during 4 months of follow-up.

Methods

Study Population

Between May 2006 and September 2008, 271 consecutive acute ST-segment elevation MI patients (143 at UZ Leuven, Leuven, Belgium [center A]; 80 at La Sapienza University, Rome, Italy [center B]; and 48 at Fondazione G. Monasterio, Pisa, Italy [center C]) treated by primary percutaneous coronary intervention (PCI) were prospectively studied by CMR at 1 week and 4 months after onset. Inclusion criteria were as follows: (1) chest pain suggestive of myocardial ischemia lasting >30 minutes but <12 hours and (2) ECG showing ST-segment elevation >0.1 mV in ≥2 limb leads or >0.2 mV in ≥2 contiguous precordial leads or presumed new left bundle-branch block. Exclusion criteria were as follows: prior MI or revascularization, atrial fibrillation, cardiogenic shock, renal failure (plasma creatinine >2 mg/dL), contraindications to CMR, and any known clinical condition that might affect RV function, including severe chronic obstructive or interstitial pulmonary disease, primary pulmonary hypertension, congenital heart disease, and moderate or severe preexisting valvular heart disease. The local ethics review boards approved the protocol, and written informed consent was obtained from each patient.

CMR Protocol

CMR studies were performed at center A with a 1.5-T unit (Intera CV, Philips Medical Systems, Best, Netherlands), at center B with a 1.5-T unit (Avanto Siemens, Erlangen, Germany), and at center C with a 1.5-T unit (Cvi, GE-Healthcare, Milwaukee, Wis). All studies were performed with the use of dedicated cardiac software, phased-array surface receiver coil, and ECG triggering. A similar CMR study protocol was followed in all centers (see the online-only Data Supplement for sequence details). After determination of cardiac axes with localizers, breath-hold steady state free-precession cine CMR was performed in cardiac vertical and horizontal long-axis orientation and in short-axis orientation. In cardiac short-axis orientation, both ventricles were completely encompassed by a stack of contiguous slices. Next, myocardial edema imaging was performed with the use of breath-hold black-blood T2-weighted, short-inversion time, inversion-recovery, fast spin-echo imaging (T2w imaging) in cardiac short-axis orientation. Finally, a breath-hold T1-weighted, 2-dimensional (Avanto Siemens; Cvi, GE-Healthcare) or 3-dimensional (Intera CV, Philips) contrast-enhanced, inversion-recovery, segmented gradient-echo sequence was used to depict the presence, location, and extent of MI (LGE imaging) and the concomitant presence of microvascular obstruction. An intravenous contrast agent dose of 0.1 to 0.2 mmol/kg gadolinium-BOPTA (Multihance, Bracco, Milan, Italy) or gadolinium-DOTA (Dotarem, Guerbet Roissy, France) was used. LGE imaging was performed between 10 and 20 minutes after contrast administration. Inversion time was individually adapted to nullify the signal of remote myocardium (usual range, 220 to 300 ms). At 4-month follow-up, a similar CMR protocol was used, except for T2w imaging that was discontinued after evaluation of the first 70 patients, showing normalization of increased signal intensity (SI) in the jeopardized myocardium.

Image Analysis

All CMR studies were analyzed off-line with the use of in-house developed cardiac vendor-independent software (CardioViewer), by the consensus of 2 experienced observers (J.B., J.G.). Both operators were unaware of clinical and angiographic data. Analysis was started by scoring T2w imaging quality with a 4-grade score, as follows: (1) poor, (2) moderate, (3) good, and (4) excellent. Only examinations scored >1 were considered for further analysis. For regional RV analysis, the Isner classification, dividing the ventricle into 12 segments (4 basal, 4 midventricular, and 4 apical), was used.\textsuperscript{11,12} For LV analysis, we used the 17-segment model proposed by the American Heart Association.\textsuperscript{14} Segment 17 (ie, LV apex) was excluded from analysis. On T2w imaging, LV edema was considered present if SI of hypointense myocardium was >2 SD above the mean SI of remote myocardium.\textsuperscript{15} Next, extent of LV edema toward RV free wall (FW) was evaluated. To discriminate RV edema from intracavitary slow flow or pericardial effusion and optimize the accuracy of RV edema detection, we adopted the following approach: (1) SI of LV edema was taken as reference to evaluate RV edema, and (2) we performed a side-by-side analysis of T2w and cine imaging in short-axis orientation. A RV segment was rated positive when the hypointense signal involved ≥50% of its circumferential length. Extent of RV edema was calculated as the number of involved segments.

Cine CMR was used to derive RV and LV volumes, ejection fraction (EF), regional wall motion, and LV mass. Contours of short-axis cine images were traced manually at end-diastole and end-systole. Papillary muscle and trabeculations were included in the cavity. In basal slices, particular care was taken to exclude the atrium from the contours. Regional wall motion per segment was scored 1 to 5 (1, normal/mild hypokinesia; 2, moderate hypokinesia; 3, severe hypokinesia; 4, akinesia; 5, dyskinesia). Wall motion score index (WMSI) was determined as the sum of segmental scores divided by number of segments. Left atrial dimension was measured as the distance from midatrial septum to midlateral wall on the end-systolic horizontal long-axis cine image. This parameter was used as a marker of chronic LV diastolic function.\textsuperscript{16} Follow-up change (Δ) of RV EF, LV volume, LV EF, and left atrial dimension were also calculated as the difference between 4-month and baseline results.

For LGE imaging, LV myocardial enhancement was considered present if the SI of hypoenhanced myocardium was >5 SD above the mean SI of remote myocardium,\textsuperscript{17} whereas microvascular obstruction was defined as the hypoenhanced region within the infarcted myocardium. LV LGE size was obtained by manually drawing regions of interest and was expressed as LV percentage. For RV LGE, criteria similar to those for RV edema were used to define a segment as LGE positive or negative, and RV LGE extent was expressed as number of segments involved.

To assess intraobserver and interobserver agreement for detection of RV edema and LGE, in 20 randomly chosen patients, T2w imaging and LGE images were analyzed twice by the same observer with a 2-week interval between readings and independently by 2 operators, respectively.

Statistical Analysis

Continuous variables were expressed as mean±SD or median (25th to 75th percentiles). Categorical variables were expressed as frequency with percentage. Student independent t test or Mann–Whitney test was used as appropriate to compare continuous variable differences between patients with and without RV edema. Student paired t test or Wilcoxon test was used as appropriate to compare continuous variable differences between baseline and follow-up. In RV edema patients, comparison of continuous variables between groups with or without RV LGE at baseline and/or follow-up was achieved by 1-way ANOVA, followed by Bonferroni post hoc analysis. Comparison between categorical variables was performed by χ² test or by Fisher exact test if an expected cell count was <5. Correlation between continuous variables was tested with the use of Pearson or Spearman ρ correlation coefficient as appropriate. The k statistic was used to assess intraobserver and interobserver agreement for the identification of RV edema and LGE.
Pattern of Ischemic Injury

RV edema was detected in 123 of 242 patients (51%). Seventy-four RV edema patients (31%) showed concomitant RV LGE at baseline. However, at follow-up only 25 patients (10%) showed residual RV LGE. Conversely, myocardial edema was present in the jeopardized LV myocardium in all patients, of whom 235 (97%) showed LV LGE at baseline and follow-up. Follow-up T2w imaging (first 70 patients) showed normalization of SI in jeopardized LV and RV myocardium.

When present, RV abnormalities were contiguous with the jeopardized LV myocardium and involved a variable portion of RV FW. At baseline, extent of RV edema was significantly larger than extent of RV LGE (ie, 4.5±2.9 versus 2.9±1.8 segments; P<0.001; Figure 2). At follow-up, not only was there a significant reduction in the number of patients with RV LGE, but the extent of LGE decreased significantly in patients with persistent RV LGE (ie, from 4.2±2.3 to 3.1±2.1 segments; P<0.001; Figure 2). Although baseline LV infarct size was not significantly different between RV edema and non–RV edema patients (18±12% versus 16±11%; P=0.24), RV edema patients showed greater infarct transmurality (81±20% versus 65±32%; P=0.001) and higher frequency of microvascular obstruction (n=76 [63%] versus n=47 [39%]; P<0.001) than non–RV edema patients.

RV Ischemic Injury: Relationship to Infarct Location

In the 113 patients (47%) with an inferior LV MI, RV edema and RV LGE were observed at baseline in 85 (75%) and 61

Results

Study Population

Twenty-nine patients (11%) were excluded from analysis because of insufficient T2w imaging quality, yielding a total of 242 patients (203 men, aged 59±10 years) (Figure 1). The presence of RV edema was used to dichotomize patients. Baseline characteristics are summarized in Table 1. Mean blood pressure and heart rate were lower in RV edema patients than in non–RV edema patients. Preprocedural Thrombolysis in Myocardial Infarction flow was lower in RV edema patients than in non–RV edema patients. Preprocedural arteries were successfully stented with bare metal or drug-eluting stents, and all patients received double antiplatelet therapy. During follow-up, 4 patients were hospitalized because of heart failure, and 5 underwent PCI for recurrent angina; no cardiac deaths or reinfarction occurred. All patients underwent 4-month CMR.

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<td>Medication at discharge, n (%)</td>
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Continuous variables are expressed as mean±SD or median (25th to 75th percentiles). Categorical variables are expressed as frequency with percentage. LAD indicates left anterior descending coronary artery; RCA, right coronary artery; LCx, left circumflex coronary artery; TIMI, Thrombolysis in Myocardial Infarction; and ACE, angiotensin-converting enzyme.
patients (54%), respectively. At follow-up, 20 patients (18%) showed residual RV LGE (Figure 3). In the 116 patients (48%) with an anterior LV MI, RV edema and RV LGE were observed at baseline in 38 (33%) and 13 patients (11%), respectively (Figure 4). At follow-up, only 5 patients (4%) showed residual RV LGE. In the 13 patients (5%) with a pure lateral LV MI, no RV edema or RV LGE was detected.

At baseline, extents of RV edema and LGE were significantly larger in inferior than anterior MI (ie, $4.5\pm2.5$ versus $2.9\pm1.4$ segments for RV edema; $P<0.001$; $3.2\pm2.0$ versus $2.3\pm0.9$ segments for RV LGE; $P=0.008$). At follow-up, RV LGE extent was slightly but not significantly larger in inferior than anterior MI patients (ie, $3.3\pm2.4$ versus $2.3\pm0.5$ segments; $P=0.11$).

Retrospective analysis of post-PCI coronary angiograms of anterior LV MI patients (105 studies useful for analysis) showed an RV branch originating from the left anterior descending coronary artery in 21 patients; 16 of them showed RV edema.

**RV Ischemic Injury: Ventricular Volumes and Function**

At baseline, RV WMSI was more compromised in RV edema patients than in non–RV edema patients and was positively related to the number of segments with myocardial edema and LGE (Spearman $\rho$ correlation coefficient $=0.71$ and Spearman $\rho$ correlation coefficient $=0.64$, respectively; both $P<0.001$) (Table 2). RV edema patients showed larger RV volumes and lower RV EF than non–RV edema patients. RV

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**Figure 2.** Inferior LV infarction in a 56-year-old man with proximal right coronary artery occlusion. Short-axis T2w imaging at baseline (A) shows myocardial edema in inferoseptal and inferior LV segments (*') widely extending to RV inferior and part of lateral FW (arrows). Small pericardial effusion (arrowheads) is visible along inferior RV FW. Short-axis LGE imaging at baseline (B) shows transmural LGE of inferoseptal and inferior LV segments, with evidence of microvascular obstruction (black arrowhead), extending to inferior RV FW (white arrow). At follow-up T2w imaging (C), myocardial edema has resolved completely, whereas on LGE imaging (D), the LV infarcted region has a thinned appearance (black arrowhead), and RV LGE is no longer present.

**Figure 3.** Inferior LV infarction in a 68-year-old man with proximal right coronary artery occlusion. Short-axis T2w imaging at baseline (A), LGE imaging at baseline (B), and follow-up (C) are shown. Hyperintense myocardium (A) in LV inferoseptum (arrow) extending to RV inferior FW (arrowheads) is shown. At baseline, LGE imaging (B) shows transmural inferoseptal LV enhancement (arrow) with enhancement of adjacent RV inferior FW (arrowhead). At follow-up LGE imaging (C), inferoseptal LV enhancement has become subendocardial (arrow), and RV LGE has disappeared completely.

**Figure 4.** Baseline short-axis T2w imaging at 4 short-axis levels (A through D; from apex to base) in a 68-year-old man with proximal occlusion of left anterior descending coronary artery. Hyperintense appearance of jeopardized anteroseptal and anterior LV myocardium (arrowheads), extending to adjacent RV lateral FW (arrows), is shown.
EF was inversely related to extent of RV ischemic injury expressed by number of segments with myocardial edema (Spearman $\rho$ correlation coefficient = $-0.31$, $P<0.001$) or with LGE (Spearman $\rho$ correlation coefficient = $-0.35$, $P<0.001$) but did not correlate with baseline LV EF ($r=0.06$, $P=0.39$). RV edema patients had larger baseline LV volumes, although LV WMSI and LV EF were similar between the 2 groups.

At follow-up, RV edema patients showed a significant improvement in RV WMSI, decrease in RV end-systolic volume, and increase in RV EF (all $P<0.001$), whereas in non–RV edema patients, there were no changes in RV WMSI, volumes, or global function. Differences in LV end-diastolic volume between groups persisted, although both groups showed a slight but significant increase in LV EF ($P\leq 0.001$). In both groups, left atrial size increased to a similar extent at follow-up ($P<0.001$).

### Pattern of RV Ischemic Injury: Functional Consequences

To assess the impact of RV ischemic injury pattern on RV function and recovery, presence or absence of RV LGE at baseline and follow-up was considered (Figure 1). In the RV edema group ($n=123$), 49 patients showed RV edema exclusively, 49 patients had RV LGE only at baseline, and 25 patients showed baseline RV LGE persisting at 4 months (Table 3). At baseline, patients with persistent RV LGE showed more extensive RV ischemic injury than the other 2 groups, and this pattern was associated with worse regional function. Patients with RV LGE had larger RV end-systolic volume and lower EF than patients with only RV edema. At follow-up, lack of persistent RV LGE was associated with decreased RV end-systolic volume and improvement in RV WMSI and EF (all $P<0.001$). In patients with persistent RV LGE, although there was an improvement in RV WMSI ($P<0.001$), the increase in RV EF at follow-up ($P=0.033$) was associated with an increase of RV end-diastolic volume ($P=0.089$) but not with a reduction of end-systolic volume ($P=0.79$), likely indicating adverse RV remodeling. Compared with patients with only RV edema, patients with persistent RV LGE showed larger end-systolic volume and lower RV EF at follow-up.

### Determinants of RV Functional Recovery at Follow-Up

With the use of univariable analysis, increase of RV EF during follow-up was associated with right coronary artery occlusion, presence of postinfarction RV edema, presence of baseline RV LGE, lower baseline RV EF, decrease in LV end-systolic volume, and improvement in LV EF during follow-up (Table 4). Multivariable analysis revealed that absence of RV LGE at follow-up remained associated with RV EF improvement even after correction for other important determinants, namely, baseline RV EF, presence of
In this prospective cohort of 242 consecutive patients studied with CMR in 3 centers, we have demonstrated that RV edema and LGE, reflecting ischemic myocardial injury, are often present early after reperfusion of ST-segment elevation MI. RV abnormalities are contiguous to the jeopardized LV myocardium and do not occur exclusively in inferior LV infarcts but are found in up to 33% of anterior LV infarcts as well. The presence of RV ischemic injury is associated with early RV dysfunction as well as with RV functional recovery at follow-up. Another remarkable finding of the present study is the reduction in frequency and extent of RV LGE at 4 months after infarction. In contrast to patients with transient forms of RV ischemic injury, patients with persistent RV LGE show adverse RV remodeling and worse function at follow-up.

Use of T2w imaging to depict infarct-related myocardial edema and to determine the area at risk in acute LV MI has been validated by several groups.10,11 Although preliminary data8 suggest the potential of T2w imaging in identifying infarct-related RV edema, to date, the value of T2w imaging has never been evaluated in a large cohort of consecutive MI patients. Additionally, LGE imaging is an established technique in depicting myocardial necrosis and/or fibrosis in the acute and chronic settings of MI.19,20 Therefore, combined T2w imaging/LGE imaging CMR is likely the best approach to identify and characterize ischemic myocardial injury. Although it is definitely more challenging to apply these CMR sequences in the thin-walled RV, the present study shows good intraobserver and interobserver agreement for RV edema and LGE detection.

Early postreperfusion RV edema and RV LGE are found in approximately one half and one third of patients, respectively, emphasizing frequent ischemic RV involvement in patients without clinical evidence of hemodynamic RV compromise. RV abnormalities are typically contiguous to the jeopardized LV myocardium and involve a variable portion of the adjacent RV wall. This is a well-known phenomenon in inferior LV infarcts, and our data are in agreement with published results.1–3,12 Remarkably, as shown clearly in this study, RV ischemic injury occurs in a considerable portion of patients with anterior LV infarcts, although the magnitude is less compared with inferior LV infarcts. When it is taken into account that in pigs 28% of RV mass is supplied by the left anterior descending coronary artery21 and that in approximately one fourth of patients >30% of the RV FW is supplied by left anterior descending coronary artery branches,22 the finding of concomitant RV ischemic injury in anterior infarcts may not be so surprising, but until now we lacked the appropriate means to accurately depict its presence. Retrospective analysis of coronary angiograms in anterior LV MI patients showed RV branches originating from the left anterior descending coronary artery in 21 patients, 16 of whom had evidence of associated RV ischemic injury. Although this percentage is lower than that reported by James,22 it should be emphasized that angiographic visualization of RV branches immediately after primary PCI may be hampered by the small size of these vessels (<1 mm), exceeding the spatial resolution of x-ray coronary angiography, the nonoptimal orientation of the angiographic projections, and possible occlusion caused by distal embolization or plaque shift during PCI.

The presence of early postinfarction RV ischemic injury is associated with regional and global RV dysfunction. In fact, patients with RV edema have consistently lower regional and global RV performance than patients without RV ischemic injury. Nonetheless, patients with RV ischemic injury demonstrate consistent regional and global functional recovery during follow-up. Remarkably, univariable analysis shows that RV function improvement is associated with the presence of postinfarction RV edema, and this result remains unchanged after correction for other important determinants such as reduction of LV end-systolic volume and improvement of LV EF during follow-up. Overall, these findings...
support the concept that acute RV dysfunction and subsequent functional recovery are caused primarily by RV ischemic injury, arguing against a significant influence of LV dysfunction on RV performance through interventricular mechanical dependence or the pulmonary vascular system. Moreover, our results agree nicely with animal studies showing that dysfunctional RV ischemic myocardium consists predominantly of viable or only modestly necrotic tissue, indicating that the RV is more resistant to ischemic injury than the LV. This can be attributed to a more favorable oxygen demand/supply profile for the RV, as follows: (1) by facing lower stroke work (2) by extracting less oxygen at rest and thereby having greater oxygen reserve during stress; and (3) by resulting in more efficient circulation due to high systolic/diastolic flow ratio and protective anatomic collaterals from the left coronary system.

Although LGE imaging is an established technique for depicting LV myocardial necrosis and/or fibrosis, this technique has been much less validated for RV MI assessment. In our study, only 33% of patients with initial RV LGE showed LGE at follow-up, and, overall, 10% of patients had evidence of postinfarction RV myocardial fibrosis. The latter finding is in good agreement with the study of Larose et al demonstrating RV LGE in 13 of 144 patients (9%) 30 days after infarction but differs from the study by Kumar et al describing persistence of RV LGE in 25 of 27 inferior MI patients at 13±9 months after infarction. This discrepancy may be explained by the difference in study population, considering that in the latter study 62% of patients had extensive baseline RV LGE involvement, defined as >4 segments with LGE, in contrast to only 17% of patients in our series. In fact, infarct volume may shrink up to 24% of its initial size as irreversibly damaged myocardium is replaced by scar, resulting in a smaller LGE size at follow-up. This implies that limited baseline RV LGE may result in a very small extent of LGE at follow-up, likely excluding the spatial resolution of current LGE sequences. Thus, it is conceivable that LGE imaging was unable to detect RV LGE at follow-up in a certain proportion of our study patients. In fact, the current LGE technique, optimized for the detection of LV LGE, encounters several problems when applied to the thin and trabeculated RV FW, primarily because of insufficient spatial resolution and related partial volume effects. Use of thinner slices, fat-saturation pre pulses, and optimized inversion times may help to improve RV LGE detection. Although some reports suggest that infarct-related edema within the viable portion of the jeopardized LV myocardium can contribute to early postinfarction LGE, further research is warranted to assess whether peri-infarct edema plays a role in the reduction of frequency and extent of RV LGE at follow-up.

**Study Limitations**

Although this was a 3-center study in which different vendor CMR units were used, a similar study protocol was used with centralized data analysis. T2w imaging is susceptible to signal loss in cardiac structures distant from the surface coil. However, all CMR units used a SI correction algorithm to homogenize signal throughout the field of view. Correct identification of myocardial edema on T2w images is challenging particularly in thin anatomic structures, such as the RV FW. In particular, discrimination between RV edema and slow flow or adjacent pericardial effusion may be troublesome. Although computer-aided RV edema detection is recommendable, we performed side-by-side visual analysis of T2w and cine imaging, excluding patients with suboptimal T2w imaging from analysis, using the increased SI of LV edema as reference to determine RV edema. Slow flow and pericardial fluid typically have higher SI than edematous myocardium, allowing differentiation from RV edema. New flow-independent T2w sequences are appealing to improve discrimination between slow-flow artifacts and myocardial edema. Depiction of ischemic-related RV myocardial injury may further benefit CMR sequences with improved spatial resolution and optimized inversion time for RV LGE. In the present study, ECG right precordial leads were not routinely recorded. Kumar et al reported only mild agreement between ECG and CMR in detecting RV infarctions. The use of CMR-derived left atrial diameter may not be ideal to accurately assess diastolic (dys)function. Finally, occurrence of microvascular obstruction was detected on LGE images, reflecting pronounced damage of coronary microvasculature.

**Conclusion**

Our study sheds new light on RV ischemic injury in patients with reperfused MI. First, temporary RV dysfunction is frequently present early after infarction and is determined primarily by RV ischemic involvement. Second, although the RV is preferentially involved in inferior LV infarcts, it is not uncommon to find similar, albeit less extensive, abnormalities in anterior LV infarcts as well. Third, a significant reduction in frequency and size of RV LGE is observed at follow-up. Finally, persistent RV LGE occurs in a minority of patients, most likely representing postinfection myocardial fibrosis, and is associated with adverse RV remodeling and worse function at follow-up.

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**Disclosures**

None.

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LV infarction, ischemia may extend toward the adjacent RV free wall and lead to transient RV dysfunction. Moreover, it is important to realize that in patients with anterior infarction, ischemia may also extend to the right coronary artery occlusion. Circulation. 1994;90:1398–1409.

Follow-up CMR showed reversibility of acute RV dysfunction with limited permanent myocardial damage on late enhancement (31% of patients) of RV free wall adjacent to jeopardized LV myocardium, and functional abnormalities.

Ischemic RV infarction. CMR included assessment of myocardial edema with the use of T2-weighted imaging, myocardial enhancement imaging of viable rather than irreversibly damaged myocardium. Moreover, it is important to realize that in patients with anterior infarction, ischemia may extend toward the adjacent RV free wall and lead to transient RV dysfunction.
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SUPPLEMENTAL MATERIAL
### Supplemental Tables

Parameters of CMR sequences used by diverse 1.5 T scanners in the 3 centers.

<table>
<thead>
<tr>
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<th>SSFP Cine CMR</th>
<th>T2-weighted STIR FSE</th>
<th>CE T1-w IR fast GE</th>
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</thead>
<tbody>
<tr>
<td><strong>Intera CV, Philips</strong></td>
<td>FOV: 350-400 mm; TR/TE: 3.6/1.8 ms; (\alpha): 60°; matrix: 256x160; ST: 8 mm; no interslice gap</td>
<td>FOV: 380-400 mm, TR/TE: 2 R-to-R intervals/100 ms; TI: 150 ms, matrix: 256x256; ST: 8 mm; no interslice gap</td>
<td>FOV: 350-400 mm; TR/TE: 4.5/1.3 ms; (\alpha): 15°; matrix: 256x128, ST: 5 mm; no interslice gap</td>
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<td><strong>Avanto, Siemens</strong></td>
<td>FOV: 340-400 mm, TR/TE: 5.2/1.2 ms, (\alpha): 80°; matrix: 256x256, ST: 8 mm; no interslice gap</td>
<td>FOV: 340-400 mm, TR/TE: 2 R-to-R intervals/75 ms; TI: 170 ms, matrix: 256x256; ST: 8 mm; no interslice gap</td>
<td>FOV: 380-420 mm; TR/TE: 6.0/3.8 ms; (\alpha): 25°; matrix: 256x192; ST: 8 mm; no interslice gap</td>
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<tr>
<td><strong>CVi, GE Healthcare</strong></td>
<td>FOV: 350-400 mm, TR/TE: 3.2/1.6 ms, (\alpha): 60°; matrix: 256x256, ST: 8 mm; no interslice gap</td>
<td>FOV: 380-400 mm, TR/TE: 2 R-to-R intervals/100 ms; TI: 150 ms, matrix: 256x192; ST: 8 mm; no interslice gap</td>
<td>FOV: 380-420 mm; TR/TE: 4.6/1.3 ms; (\alpha): 20°; matrix: 256x192; ST: 8 mm; no interslice gap</td>
</tr>
</tbody>
</table>

**Table Legend:** \(\alpha\): flip angle; CE: contrast enhanced; CMR: cardiovascular magnetic resonance; FOV: field of view; FSE: fast spin echo; GE: gradient echo; SSFP: steady state free precession; IR: inversion recovery; ST: slice thickness; STIR: short inversion-time inversion-recovery; TE: echo time; TI: inversion time; TR: repetition time.
Video Legends

Mid-ventricular short-axis cine CMR (same patient as in Figure 1) at baseline (movie a) and follow-up (movie b). Dysfunctional right ventricle at baseline (end-diastolic volume 167 ml, EF 41%) with severe hypokinesis of inferior and lateral RV FW, showing functional improvement at follow-up (end-diastolic volume 145 ml, EF 65%).