Comparison of delayed enhancement patterns on multislice computed tomography immediately after coronary angiography and cardiac magnetic resonance imaging in acute myocardial infarction

M Habis,[1] A Capderou,[2] A Sigal-Cinqualbre,[3] S Ghostine,[1] S Rahal,[1] J Y Riou,[3] P Brenot,[3] C Y Angel,[3] J F Paul[3]

[1] Department of Cardiology, Centre Chirurgical Marie Lannelongue Le Plessis Robinson, France; [2] Department of Physiology Univ Paris-Sud, France; [3] Department of Radiology, Centre Chirurgical Marie Lannelongue, Le Plessis Robinson, France

Correspondence to:
Dr Michel Habis, Centre Chirurgical Marie Lannelongue 133 avenue de la Resistance, 92350 Le Plessis Robinson, France; mmchabis@free.fr

Accepted 11 November 2008
Published Online First 3 December 2008

ABSTRACT

Objective: Recent experimental and limited clinical studies have demonstrated the usefulness of delayed enhancement multislice computed tomography (MSCT) for assessing myocardial infarct size (IS) and transmurality. The aim of this study is to compare MSCT enhancement patterns immediately after coronary angiography (CAG) in an acute myocardial infarction (AMI) setting with cardiac magnetic resonance (CMR) enhancement during the second week follow-up.

Methods: 26 patients admitted for an AMI were evaluated by MSCT immediately after CAG without iodine re-injection. All but three were reperfused. The same patients had delayed enhancement CMR imaging at 10 (SD 4)-day follow-up. Myocardial enhancement was considered transmural (non-viable) when involving >75% of myocardial thickness, subendocardial (1 − ⩽75%) or normal (viable for the two latter). Two or more >75% enhanced segments were required to define transmurality on patient-level or culprit artery-level analysis. A semi-quantitative scale score was defined for the 17 left ventricular segments. IS was computed from these scores.

Results: On segment analysis, sensitivity, specificity, accuracy, positive and negative predictive values of MSCT for transmurality assessment were 84%, 96%, 94%, 85% and 96%, respectively, compared to CMR. On patient analysis, these respective values were 90%, 80%, 88%, 95% and 67%. IS assessed by the two methods were highly correlated ($r = 0.94$, $p<0.0001$) and the regression line did not statistically differ from the identity line.

Conclusion: MSCT enhancement immediately following CAG without iodine re-injection for an AMI is a reliable method for evaluating transmurality and IS. This very early evaluation could be an interesting alternative to CMR.

Transmurality and infarct size (IS) assessments in the setting of acute myocardial infarction (AMI) are a real and essential challenge because they provide important prognostic information. Left ventricular remodelling and its deleterious consequences are predicted by transmurality extent[1 2] and IS.[3–5] Preliminary studies have also demonstrated a correlation between infarct (but not the myocardium at risk) size assessed by technetium-99m sestamibi and mortality.[6 7] Stunning of the area at risk and the unpredictable contractility of the remote myocardium are responsible for the mismatch between left ventricular function and IS.[8 9] Differentiation between stunned and irreversibly damaged myocardium is the aim of all methods of viability assessment. Cardiac magnetic resonance (CMR) has progressively been considered the reference method[10–15] but lacks diffuse availability. Two recent experimental studies have demonstrated that IS assessment by multislice computed tomography (MSCT), histomorphometry[16 17] and CMR[17] are well correlated. On the other hand, two clinical studies have highlighted the correlation between delayed enhancement 16-slice computed tomography (CT) and CMR both performed within two weeks after an AMI.[18 19] A recent study has also shown that delayed enhancement 64-slice CT immediately after coronary angiography (CAG) for an AMI is reliable for early viability assessment compared to low-dose dobutamine echocardiography.[20] Finally in a recent study, this latter method of assessing transmurality was able to predict left ventricular remodelling at follow-up.[21] We aim to compare very early delayed enhancement patterns on MSCT and CMR performed during the second week follow-up, for assessing IS and transmurality in AMI.

MATERIALS

Over a 9-month period (from September 2006 to May 2007), 28 patients admitted for an AMI to our hospital during daytime hours were enrolled. Inclusion criteria were: ST-segment elevation and/or long-lasting spontaneous chest pain within the preceding 3 days and/or acute coronary occlusion, with more than twice normal creatine kinase release. Exclusion criteria were previous myocardial infarction and renal impairment (creatinine >200 μmol/l). Two patients admitted in cardiogenic shock died during coronary angioplasty. The remaining 26 completed the study. CAG was performed as soon as the patient arrived at the hospital in cases of unrelieved chest pain. The contrast agent used was Iomeprol 350 mg/ml (Bracco, Milan, Italy). When thrombolytic agent was perfused, CAG was performed 90 minutes later with continuing chest pain or the following day in cases of relieved chest pain. The culprit coronary artery was defined on the combination of electrocardiogram (ECG) at admission and CAG result. Angioplasty was immediately attempted in cases of cardiogenic shock, unrelieved chest pain and/or TIMI 2 or less flow downstream from the culprit stenosis. All 26 patients underwent MSCT immediately following CAG. Contrast agent volume and the time elapsed from the last

Table 1 Baseline characteristics of the study population

Patients	Gender	Age (years)	BMI (kg/m²)	ST	Entry status*	MI	CK IU	Contrast Volume (ml)	Time to scan (min)	Heart rate (beats/min)	CA	Status of CA†	SSCA	DIC¶ (h)	DIT§ (h)
1	M	64	34.7	ST+	FA	ant	3625	160	17	58	LAD	ss	1	6	4
2	M	57	29.9	ST+	PA	inf	628	180	45	55	RCA	occl	2	8.5	3.5
3	M	68	25.2	ST−	A	lat	964	210	23	47	Cx	occl	2	6.5	5
4	M	45	23.1	ST+	PA	inf	5385	100	10	58	RCA	occl	2	8	8
5	F	59	21.4	ST+	PA	ant	5856	130	22	95	LAD	occl	1	2	2
6	M	76	26.0	ST+	A	inf	2224	80	15	70	RCA	ss	3	96	9
7	F	83	25.8	ST+	PA	inf	710	180	17	51	RCA	occl	3	32	12
8	M	57	34.7	ST+	PA	inf	743	160	20	93	RCA	occl	2	7	6
9	M	76	27.2	ST+	A	lat	549	60	16	69	Cx	occl	3	85	72
10	F	39	21.9	ST+	A	lat	828	80	17	88	Cx	occl	3	24	6.5
11	M	64	26.7	ST+	PA	lat	3900	200	6	95	Cx	occl	3	1.5	1
12	F	49	27.9	ST+	PA	lat	8173	190	19	88	Cx	occl	1	4	3
13	M	54	30.5	ST+	PA	ant	1156	150	45	61	LAD	ss	2	43	2
14	M	62	23.5	ST+	FA	inf	518	240	28	80	RCA	ss	1	14	1
15	M	73	36.7	ST+	PA	lat	6619	180	20	51	Cx	occl	2	19	4
16	M	52	25.9	ST+	AT	ant	436	60	30	68	LAD	ss	3	24	2
17	M	64	25.0	no ST	PA	ant	580	190	30	60	LAD	ss	1	39	27
18	F	83	31.3	ST+	PA	inf	456	70	30	65	RCA	ss	2	6	2
19	M	62	27.6	ST+	PA	ant	6112	360	32	52	LAD	occl	3	11	9
20	M	52	24.8	ST+	PA	inf	1786	210	15	58	RCA	occl	3	28	7
21	F	77	26.0	ST+	PA	ant	5586	240	37	63	LAD	ss	1	1	1
22	M	65	26.8	ST+	PA	inf	871	285	25	65	RCA	occl	3	6.5	6
23	M	72	22.9	ST+	A	ant	763	100	20	65	LAD	ss	2	2.5	1
24	M	57	27.8	ST+	PA	inf	1744	220	5	88	RCA	occl	1	6	4.5
25	M	65	19.7	ST+	FA	inf	2721	175	15	50	RCA	ss	3	72	3.5
26	M	53	24.2	ST+	PA	ant	3734	160	15	65	LAD	ss	1	18	2.5

ST-segment (ST) variation: elevation (ST+), depression (ST−), no variation (no ST).
*Entry status: coronary angiography without previous thrombolytic therapy (A), angiography after thrombolytic therapy (AT), facilitated angioplasty (FA), primary angioplasty (PA).
†Status of CA: occl, occluded; ss, significant stenosis.
‡SSCA, number of coronary arteries with significant stenosis.
¶DIC, delay (h) from onset of myocardial infarction to coronary angiogram.
§DIT, delay (h) from onset of myocardial infarction to treatment.
CA, culprit artery; LAD, left anterior descending coronary artery; CK, creatine kinase; Cx, circumflex; MI, myocardial infarction (ant, anterior; inf, inferior); lat, lateral; RCA, right coronary artery.

coronary injection to MSCT scanning were recorded. Creatine kinase samples were repeated every 12 hours after admission until their peak. Patients had CMR evaluation 10 (SD 4) days after AMI. The study was approved by our institution committee and all patients gave their informed consent.

METHODS

Multislice computed tomography

All images were acquired without contrast agent re-injection. The evaluation was performed on a 64-slice (seven first cases) then dual-source CT (19 last patients) (Somatom Definition, Siemens, Erlangen, Germany) using ECG-gated acquisition with ECG-tube current modulation. Low kilovoltage setting was applied (80 kV in patients below 70 kg, 100 kV above) with a tube current of 700 mAs in both cases. Mean dose length product was 240 mGy × cm corresponding to a radiation dose of 4 (SD 1) mSv. Images were reconstructed at the mid diastolic phase with an initial 3-mm slice thickness. Sections of the left ventricle in the short axis (basal, mid-cavity and apical), apical four-chamber and two-chamber views were obtained by 8-mm slice reformatted images. Delayed enhanced, remote myocardium and left ventricle cavity densities were recorded.

CMR protocol

CMR examinations were performed with a 1.5-T MR system (Siemens Avanto, Erlangen, Germany) using a dedicated six-element cardiac phased-array surface coil. All patients were examined in the supine position while holding their breath at inspiration. The imaging protocol included (a) gradient echo imaging in the three orthogonal planes of the chest (scout localisation imaging) to locate the long axis, then the short axis and four chamber views, (b) short axis breath-hold contrast-enhanced steady-state free precession cine study, and (c) 3D short axis contrast-enhanced gradient-echo T1-weighted acquisitions (TR = 700 ms, TE = 1.44 ms) with an inversion time determined previously by a scout-TI sequence (TI 180–240 ms) to null the signal of normal myocardium with 8-mm slice thickness (d) 2D two-chamber and four-chamber contrast-enhanced gradient-echo T1-weighted acquisitions (TR = 833 ms, TE = 4.3 ms) with 8-mm slice thickness and the same TI as in (c). All the acquisitions were prospectively cardiac-gated. Delayed perfusion imaging sequences were performed in diastole, 15 minutes after bolus infusion via a power injector of 0.2 mmol/kg of gadopentetate dimeglumine (Magnevist; Schering, Berlin, Germany).

Image analysis

MSCT and CMR images were assessed by two independent observers unaware of each other's data. The same 17-segment model[22] was used to assess contrast enhancement patterns by both techniques; 75% myocardial thickness enhancement was considered the best threshold to differentiate subendocardial and transmural involvement of each diseased segment.

Transmural, subendocardial and normal segments were quoted 4, 3, 2, 1 and 0 when enhancement involved >75%, 51–75%, 26–50%, 1–25% and 0% of myocardial thickness respectively. Myocardial IS (percentage of left ventricle) was assessed semi-quantitatively as the sum of 17-segment myocardial enhancement scores divided by 68 (17×4). No or subendocardial enhancement was expected to reflect a viable myocardium. Transmural enhancement was expected to reflect non-viability. Patients were considered having a transmural infarction by either method whenever ⩾2 adjacent segments were >75% enhanced. In addition, right ventricular wall enhancement was assessed by both techniques.

Statistics

Statistical analysis was performed with StatView 5.0 software. Percentages are expressed with 95% confidence interval (95% CI). Continuous variables were expressed as mean (SD). Differences between CMR and 64-slice or dual-source CT infarct size were compared by a non-paired Student *t* test. IS determined by MSCT versus CMR were compared by a paired Student *t* test.

A linear regression model was used to assess the relation between those two IS values obtained for each patient, and the regression line was compared to identity line by testing slope and intercept against 1 and 0, respectively, by Student *t* tests. The two IS values were also compared by a Bland and Altman analysis. Correlations were tested after transformation by z value of Fisher to evaluate the probability of rejecting the null hypothesis ($r = 0$). We reported correlation coefficient (r) and the assessed probability from z transformation (p). A correlation between CAG to MSCT delay, creatine kinase value, body mass index (BMI), contrast volume injected and the difference between MSCT and CMR infarct sizes was assessed. Enhancement on MSCT was compared to that on CMR accepted as the reference method. Classifications of enhancement as normal, subendocardial and transmural were compared by a Cohen's kappa test. MSCT transmurality assessment was depicted as sensitivity, specificity, accuracy, positive and negative predictive values on segment, patient and culprit artery level. Statistical tests were two-tailed and p value <0.05 was regarded as statistically significant.

RESULTS

Our study population comprised 20 males and six females (table 1).

Mean age was 63 (11) years. Twenty-five patients were in sinus rhythm and one had atrial fibrillation. Twenty-four patients had ST-segment elevation, one had ST depression and one no ST variation. Myocardial infarction was anterior (9), inferior (11), and lateral (6). Seventeen patients had a primary angioplasty when entering the study. Three had a facilitated angioplasty following a thrombolytic therapy. The remaining had a CAG following (1) or not (5) a thrombolytic therapy. The median delay between onset of myocardial infarction and first therapy was 4 hours (range 1–72 hours). The median delay between onset of myocardial infarction and CAG was 10 hours (range 1–96 hours). Left anterior descending (LAD) (9), right (11) and circumflex (6) were the culprit coronary arteries. Coronary angiography depicted one significant left main stenosis. LAD was occluded (2 patients) or significantly stenosed (19). These respective values were 7 and 9 for the circumflex and 7 and 10 for the right coronary artery. This yielded 10 trivascular, 8 bivascular and 8 monovascular patients. Median creatine kinase level was 1450 IU/l (range 436–8173). No patient had recurrent ischaemia or additional revascularisation procedures between MSCT and CMR evaluation. The culprit coronary artery was reperfused at the time of the two evaluations for all except three patients. Two of the latter had delayed bypass surgery.

Multislice computed tomography

All examinations achieved sufficient diagnostic quality for the assessment of the myocardial contrast changes. Densities of enhanced, remote myocardium and left ventricle cavity were 116 (52), 58 (11) and 69 (16) Hounsfield units, respectively. No significant difference was found between 64-slice and dual-source CT IS comparisons with CMR ($p = 0.12$). Iomeprol volume injected during CAG was 168 (72) ml. The time elapsed from the end of CAG to MSCT scanning was 22 (10) minutes. Heart rate during scanning was 68 (15) beats/min. Delayed enhancement was transmural (88 segments) and subendocardial (16 segments). Three hundred and thirty-eight segments were normally enhanced. Myocardial IS on MSCT was 21% (15%) of the left ventricle.

Cardiac magnetic resonance imaging

CMR evaluation was performed 10 (4) days after myocardial infarction. Delayed enhancement was transmural (89 segments) and subendocardial (21 segments). Three hundred and thirty-two segments were normally enhanced. Myocardial IS on CMR was 23% (15%) of the left ventricle.

Comparison between MSCT and CMR

Comparison of delayed enhancement segments provided by MSCT and CMR is characterised by a kappa value of 0.76 (95% CI 0.69 to 0.83) relating a good agreement between both methods (table 2). A kappa value is 0.81 (95% CI 0.74 to 0.88) for transmurality assessment comparisons.

The difference between IS assessed by the two methods was not significant ($p = 0.28$). The two IS were highly correlated ($r = 0.94$, $p<0.0001$) and the regression line did not statistically differ from the identity line (fig 1A). Bland and Altman analysis showed a non-significant bias of 1.1% (95% CI −0.8 to 3.0) and a precision of 10% (95% CI −8.7 to 10.9) (fig 1B). IS assessed by both methods was correlated to peak creatine kinase value ($r = 0.74$, $p<0.0001$ and $r = 0.72$, $p<0.0001$ for MSCT and CMR, respectively). No correlation was found between the difference in both IS and BMI ($r = 0.215$; $p = 0.29$), delay between CAG and MSCT ($r = 0.07$; $p = 0.74$), peak creatine kinase value ($r = 0.15$; $p = 0.46$) and contrast volume injected ($r = 0.104$; $p = 0.62$). On segment analysis, sensitivity, specificity, accuracy, positive and negative predictive values of MSCT transmurality assessment were 84%, 96%, 94%, 85% and 96%, respectively, compared to CMR. On patient analysis, these respective values were 90%, 80%, 88%, 95% and 67%. The values for each culprit artery are presented in table 3.

These results were unchanged (on patient and culprit artery level) when the threshold of transmurality was decreased to 50% and were very similar on segment level (82%, 96%, 93%, 84%, 95%). MSCT misclassified three patients (two false negatives and one false positive) for whom the infarct-related artery was the right coronary: patient 22 was considered to have a transmural myocardial infarction by MSCT though CMR showed subendocardial enhancement. He had an atrial fibrillation and his peak creatine kinase value (871 IU/l) suggested a limited infarct size. Two others (patients 20 and 24) were

Table 2 Cross-table comparison of multislice computed tomography and cardiac magnetic resonance scoring for the 442 segments

		Cardiac magnetic resonance				
Multislice computed tomography		0	1	2	3	4
	0	320	1	7	2	8
	1	3	2	0	1	0
	2	1	0	1	0	6
	3	0	0	2	0	0
	4	8	0	4	1	75

suspected of having subendocardial infarction on MSCT despite transmural involvement on CMR (fig 2). No relation was found between the discrepancy of the two methods and contrast agent volume, delay between CAG and MSCT, peak creatine kinase value and BMI. Three other patients (patients 14, 16 and 23) with limited peak creatine kinase values were suspected of having no subendocardial infarction on MSCT. Patient 16 had none, and the other two (patients 23 and 14) had only one subendocardial enhanced segment each on MSCT, despite four, three and two subendocardial enhanced segments on CMR, respectively. However those three patients were correctly classified based on the previous definition of transmurality at patient level. The culprit coronary artery was the right for patient 14 and the left anterior descending for patients 16 and 23. Finally, two patients (patients 4 and 7) presenting with right ventricular concomitant enhancement on CMR were also detected by MSCT.

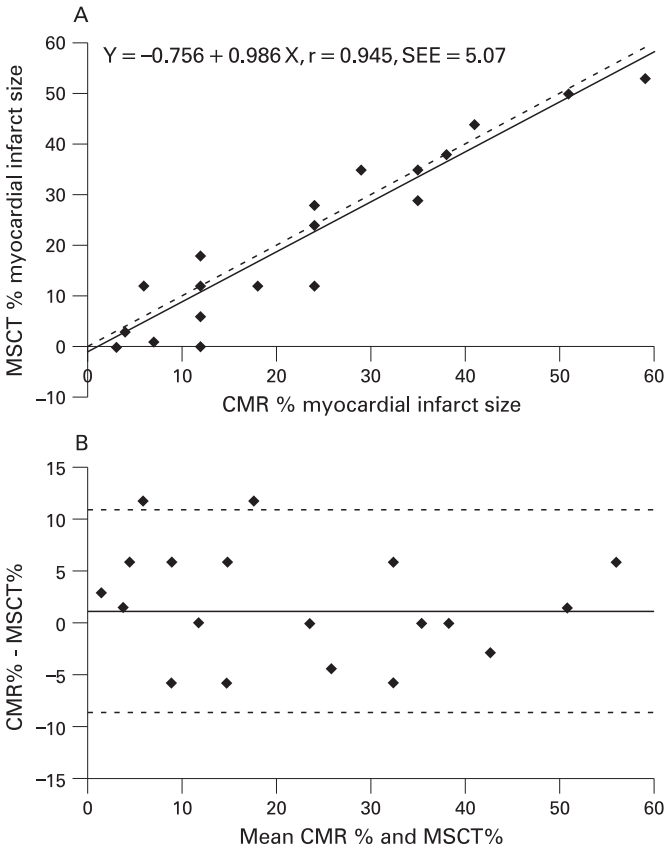

Figure 1 (A) Relation between myocardial infarct size (percentage of left ventricle) determined by multislice computed tomography (MSCT) versus cardiac magnetic resonance (CMR) as reference. Solid line, regression line; broken line, identity line. SEE = standard error of estimate. Regression line and identity line are not statistically different. (B) Comparison between myocardial infarct size (percentage of left ventricle) assessed by MSCT (MSCT%) and CMR (CMR%). Bland and Altman plots of the difference between MSCT% and CMR% as a function of their mean value. The solid line is the bias and broken lines are 95% limits of agreement. The bias (1.1%) is non-significant, the precision is around 10%.

DISCUSSION

Transmurality and non-viability assessment

Computed tomography equipment is close to the catheterisation laboratory in our institution allowing for just a short delay between CAG and MSCT. The highly concentrated iodinated contrast agent injected during angiography and the slow wash-in and wash-out from the damaged myocytes provide myocardial infarction enhancement without additional contrast. This method of viability assessment is reliable even immediately after the patient's admission, as previously demonstrated when dobutamine echocardiography was considered the reference method.[20] In addition, this transmurality assessment was also recently found to be reliable in predicting left ventricular remodelling.[21]

We chose a 75% thickness enhancement threshold for defining transmurality based on previous published studies. When it was 50% for MSCT[20] or CMR,[13] 20% of transmurally enhanced segments still had a contractile reserve. The percentage of viable segments decreased to 5% when the enhancement threshold was upgraded to 75%.[10] Therefore, the latter better delineates the infarct core in an AMI setting. In the present study, the results were nearly the same when the transmurality threshold was decreased to 50% thickness enhancement.

Transmurality (>75% thickness enhancement) assessment is concordant by the two methods (kappa value = 0.81). Disagreement concerned one false-positive and two false-negative patients. An increase in IS between the two evaluations[23] could explain the two false negative patients. However, even when performed at the same time, 29%[18] and 14%[19] of transmurally enhanced segments on CMR were underestimated by 16-slice CT, although the MSCT protocol differed from the present one. The false-positive patient had an atrial fibrillation, which could explain this misinterpretation. Finally, in the present study, though missing limited infarcts compared to CMR, MSCT enhancement did not alter the binary viability results since subendocardial and no enhancement were equally considered viable.

Myocardial infarct size

IS is a prognostic indicator after an AMI.[3–7 24–26] In a previous report, IS assessed by CMR enhancement was found to be more sensitive than by SPECT defect, especially for smaller infarcts.[27] The low spatial resolution of SPECT probably explains most of the missed cases. In the present study, the greater sensitivity for the detection of subendocardial infarcts is probably related to

Table 3 Comparison of MSCT and CMR enhancement on segment, patient and culprit artery level

	No	TP	TN	FP	FN	Sensitivity	Specificity	Accuracy	PPV	NPV
Patients	26	19	4	1	2	90 (70–99)	80 (28–99)	88 (70–98)	95 (75–100)	67 (22–96)
All segments	442	75	340	13	14	84 (75–91)	96 (94–98)	94 (91–96)	85 (76–92)	96 (93–98)
RCA	11	7	1	1	2	78 (40–97)	50 (1–99)	73 (39–94)	88 (47–100)	33 (1–91)
Cx	6	6	0	0	0	100 (54–100)	–	100 (54–100)	100 (54–100)	–
LAD	9	6	3	0	0	100 (54–100)	100 (29–100)	100 (66–100)	100 (54–100)	100 (29–100)

Cx, circumflex; FN, false negative; FP, false positive; LAD, left anterior descending coronary artery; NPV, negative predictive value; PPV, positive predictive value; RCA, right coronary artery; TN, true negative; TP, true positive.

the better CMR signal-to-noise ratio. This highly sensitive determination of damaged myocytes is confirmed in a study correlating modest troponin release after coronary angioplasty with CMR-enhanced myocardium.[28] No correlation was found in the present study between BMI, creatine kinase value, contrast volume, CAG to MSCT delay and the difference between IS assessed by both methods. MSCT and CMR IS were highly correlated and the regression line comparing them was not significantly different from the identity line (fig 1A). This correlation was also found in a previous study.[19] We found a good correlation with both techniques despite a substantial delay (10 (4) days) between the two image acquisitions. For most of our patients the infarct size did not vary at least in the first two weeks after angioplasty. These data will need to be confirmed on a larger scale. It is well established that myocardial IS shrinks during the first 6 months.[21 29 30] Our data suggest that this is more likely to happen after the second week.

Study limitations

Iodinated contrast agent volume per patient weight and the delay between CAG and MSCT were not held constant through the study as it was for CMR precluding standardisation of the primer technique. On the other hand, delayed enhancement intensity was found very variable with a large standard deviation compared to both remote myocardium and left ventricular cavity. The difference in attenuation may be related to the extent of myocardial damage but this would require further evaluation. Though it would be attractive in the future for a more automated assessment of the real IS, this was not applicable in the present study. Three-dimensional (3D) evaluation of IS would have been more precise, but the choice of a threshold to determine hyperenhanced areas may still be a limiting factor. Dedicated 3D software are welcomed for volume evaluation in the future. In some cases, the difference between contrast-enhanced necrotic areas and salvaged myocardium was difficult to assess, especially for small subendocardial MI. Dual-energy scanning could improve contrast resolution by extracting contrast information from two datasets acquired at the same time with two different energy levels (iodine attenuation is higher at lower kV). This is a very promising new field for cardiac perfusion and delayed imaging. However, it implies lower temporal resolution (165 ms instead of 83 ms). Appropriate dual-energy protocols need to be established for cardiac acquisition in the future. The delays between CAG and MSCT or CMR were quite different, which could explain some discrepancies in IS. This delay was the result of the unsafety of performing a longer evaluation (CMR) earlier in the follow-up of a patient presenting a large AMI. In addition, our study comprised 23/26 (88%) actively reperfused patients. Therefore, our results could not be extrapolated to non-reperfused patients admitted later after AMI onset. Finally, though quicker and easier than CMR our MSCT protocol fails to evaluate microvascular obstruction that is reliably evaluated by first-pass imaging. This latter evaluation was previously demonstrated to predict functional recovery at follow-up.[26 29 30]

Clinical implications

This study demonstrates that, when performed immediately after CAG without iodinated contrast agent re-injection, MSCT is reliable for assessing two out of three important prognostic indicators in AMI:IS and transmurality. The third, microvascular obstruction is not assessable because of the lack of first-pass imaging. MSCT tended to be less sensitive than CMR for limited AMI, but these latter have usually a good prognosis. However, MSCT is easier and quicker to perform, more diffusely available and an interesting alternative for patients with a pacemaker or suffering from claustrophobia or breathlessness, but adds radiation dose to CAG.

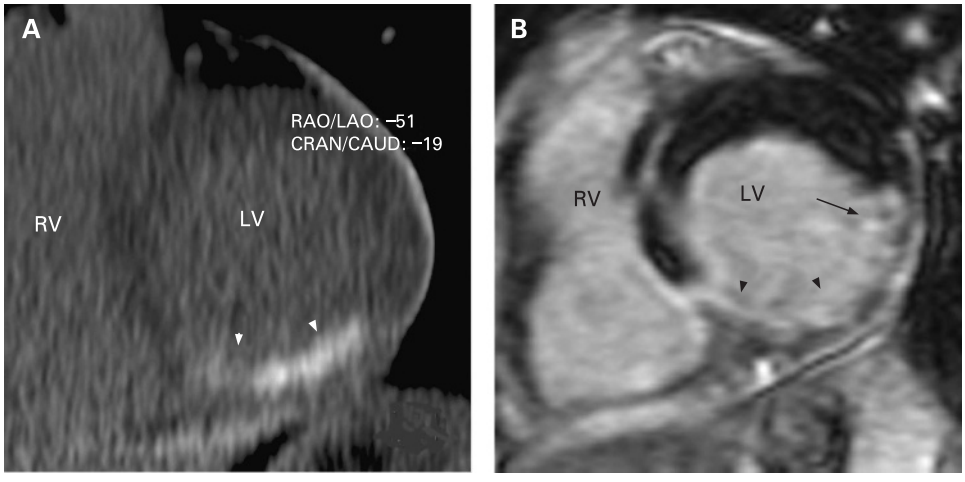

Figure 2 False-negative case: (A) computed tomography scan 15 minutes after primary angioplasty of a right coronary artery for an acute myocardial infarction of a 52-year-old patient (patient 20). Short-axis view (8-mm slice thickness) shows subendocardial enhancement of segments 10 and 11 (arrowheads). (B) Delayed enhancement cardiac magnetic resonance performed 21 days later shows clearly transmural enhancement of segments 10 and 11 with extension of the myocardial damage to the segment 9, suggesting worsening of the lesions between the two evaluations. LV, left ventricle; RV, right ventricle.

Any advances for decreasing left ventricular remodelling and cardiac in-hospital and late adverse events will aim to avoid large transmural myocardial infarctions. Therefore, when the MSCT set-up is next to the catheterisation laboratory, this reliable method could be used in the future as a surrogate for comparing different methods of successful coronary reperfusion of AMI and provide a rationale for therapy aimed at avoiding left ventricular remodelling.

CONCLUSION

MSCT enhancement immediately following CAG without iodinated contrast agent re-injection for an AMI is a reliable method for evaluating transmurality and IS. This very early evaluation could be an interesting alternative to CMR.

Funding: None.

Competing interests: None.

REFERENCES

1. **Tarantini G,** Razzolini R, Cacciavillani L, *et al*. Influence of transmurality, infarct size, and severe microvascular obstruction on left ventricular remodeling and function after primary coronary angioplasty. *Am J Cardiol* 2006;**98**:1033–40.
2. **Bolognese L,** Cerisano G, Buonamici, *et al*. Influence of infarct-zone viability on left ventricular remodeling after acute myocardial infarction. *Circulation* 1997;**96**:3353–9.
3. **Ørn S,** Manhenke C, Anand IS, *et al*. Effect of left ventricular scar size, location, and transmurality on left ventricular remodeling with healed myocardial infarction. *Am J Cardiol* 2007;**99**:1109–14.
4. **Chareonthaitawee P,** Christian TF, Hirose K, *et al*. Relation of initial infarct size to extent of left ventricular remodeling in the year after acute myocardial infarction. *J Am Coll Cardiol* 1995;**25**:567–73.
5. **Kaandorp TAM,** Lamb HJ, Viergever EP, *et al*. Scar tissue on contrast-enhanced MRI predicts left ventricular remodelling after acute infarction. *Heart* 2007;**93**:375–6.
6. **Miller TD,** Hodge DO, Sutton JM, *et al*. Usefulness of technetium-99m sestamibi infarct size in predicting posthospital mortality following acute myocardial infarction. *Am J Cardiol* 1998;**81**:1491–3.
7. **Miller TD,** Christian TF, Hodge DO, *et al*. Comparison of acute myocardial infarct size to two-year mortality in patients <65 to those >65 years of age. *Am J Cardiol* 1999;**84**:1170–5.
8. **Lima JAC,** Becker LC, Melin JA, *et al*. Impaired thickening of nonischemic myocardium during acute regional ischemia in the dog. *Circulation* 1985;**71**:1048–59.
9. **Yang Z,** Berr SS, Gilson WD, *et al*. Simultaneous evaluation of infarct size and cardiac function in intact mice by contrast-enhanced cardiac magnetic resonance imaging reveals contractile dysfunction in noninfarcted regions early after myocardial infarction. *Circulation* 2004;**109**:1161–7.
10. **Choi KM,** Kim RJ, Gubernikoff G, *et al*. Transmural extent of acute myocardial infarction predicts long-term improvement in contractile function. *Circulation* 2001;**104**:1101–7.
11. **Gerber BL,** Garot J, Bluemke DA, *et al*. Accuracy of contrast-enhanced magnetic resonance imaging in predicting improvement of regional myocardial function in patients after acute myocardial infarction. *Circulation* 2002;**106**:1083–9.
12. **Wu KC,** Lima JAC. Nonivasive imaging of myocardial viability. Current techniques and future developments. *Circ Res* 2003;**93**:1146–58.
13. **Nelson C,** McCrohon J, Khafagi F, *et al*. Impact of scar thickness on the assessment of viability using dobutamine echocardiography and thallium single-photon emission computed tomography. A comparison with contrast-enhanced magnetic resonance imaging. *J Am Coll Cardiol* 2004;**43**:1248–56.
14. **Kim RJ,** Wu E, Rafael A, *et al*. The use of contrast-enhanced magnetic resonance imaging to identify reversible myocardial dysfunction. *N Engl J Med* 2000;**343**:1445–53.
15. **Mahrholdt H,** Klem I, Sechtem U. Cardiovascular MRI for detection of myocardial viability and ischaemia. *Heart* 2007;**93**:122–9.
16. **Lardo AC,** Cordeiro MAS, Silva C, *et al*. Contrast-enhanced multidetector computed tomography viability imaging after myocardial infarction. Characterisation of myocyte death, microvascular obstruction, and chronic scar. *Circulation* 2006;**113**:394–404.
17. **Baks T,** Cademartiri F, Moelker AD, *et al*. Multislice computed tomography and magnetic resonance imaging for the assessment of reperfused acute myocardial infarction. *J Am Coll Cardiol* 2006;**48**:144–52.
18. **Mahnken AH,** Koos R, Katoh M, *et al*. Assessment of myocardial viability in reperfused acute myocardial infarction using 16-slice computed tomography in comparison to magnetic resonance imaging. *J Am Coll Cardiol* 2005;**45**:2042–7.
19. **Gerber BL,** Belge B, Legros GJ, *et al*. Characterization of acute and chronic myocardial infarcts by multidetector computed tomography. Comparison with contrast-enhanced magnetic resonance. *Circulation* 2006;**113**:823–33.
20. **Habis M,** Capderou A, Ghostine S, *et al*. Acute myocardial infarction early viability assessment by 64 slice computed tomography immediately following coronary angiography. Comparison with low-dose dobutamine echocardiography. *J Am Coll Cardiol* 2007;**49**:1178–85.
21. **Sato A,** Hiroe M, Nozato T, *et al*. Early validation study of 64-slice multidetector computed tomography for the assessment of myocardial viability and the prediction of left ventricular remodelling after acute myocardial infarction. *Eur Heart J* 2008;**29**:490–8.
22. **Cerqueira MD,** Weissman NJ, Dilsizian V, *et al*. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart. A statement for healthcare professionals from the cardiac imaging committee of the council on clinical cardiology of the American Heart Association. *Circulation* 2002;**105**:539–42.
23. **Rochitte CE,** Lima JAC, Bluemke DA, *et al*. Magnitude and time course of microvascular obstruction and tissue injury after acute myocardial infarction. *Circulation* 1998;**98**:1006–14.
24. **Gibbons RJ,** Miller TD, Christian TF. Infarct size measured by single photon emission computed tomographic imaging with 99mTc-sestamibi. A measure of the efficacy of therapy in acute myocardial infarction. *Circulation* 2000;**101**:101–8.
25. **Gibbons RJ,** Valeti US, Araoz PA, *et al*. The quantification of infarct size. *J Am Coll Cardiol* 2004;**44**:1533–42.
26. **Wu KC,** Zerhouni EA, Judd RM, *et al*. Prognostic significance of microvascular obstruction by magnetic resonance imaging in patients with acute myocardial infarction. *Circulation* 1998;**97**:765–72.
27. **Ibrahim T,** Bülow HP, Hackl T, *et al*. Diagnostic value of contrast-enhanced magnetic resonance imaging and single-photon emission computed tomography for detection of myocardial necrosis early after acute myocardial infarction. *J Am Coll Cardiol* 2007;**49**:208–16.
28. **Selvanayagam JB,** Porto I, Channon K, *et al*. Troponin elevation after percutaneous coronary intervention directly represents the extent of irreversible myocardial injury. Insights from cardiovascular magnetic resonance imaging. *Circulation* 2005;**111**:1027–32.
29. **Baks T,** Van Geuns RJ, Biagini E, *et al*. Effects of primary angioplasty for acute myocardial infarction on early and late infarct size and left ventricular wall characteristics. *J Am Coll Cardiol* 2006;**47**:40–4.
30. **Koyama Y,** Matsuoka H, Mochizuki T, *et al*. Assessment of reperfused acute myocardial infarction with two-phase contrast-enhanced helical CT: prediction of left ventricular function and wall thickness. *Radiology* 2005;**235**:804–11.

Heart

Comparison of delayed enhancement patterns on multislice computed tomography immediately after coronary angiography and cardiac magnetic resonance imaging in acute myocardial infarction

M Habis, A Capderou, A Sigal-Cinqualbre, S Ghostine, S Rahal, J Y Riou, P Brenot, C Y Angel and J F Paul

Heart 2009 95: 624-629 originally published online December 3, 2008
doi: 10.1136/hrt.2008.144097