CARDIOVASCULAR MEDICINE

Microvascular perfusion 1 week and 6 months after myocardial infarction by first-pass perfusion cardiovascular magnetic resonance imaging

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Objective: To characterise the evolution of myocardial perfusion during the first 6 months after myocardial infarction by first-pass perfusion cardiovascular magnetic resonance imaging (CMR) and determine its significance.
Design: Prospective cohort design.
Setting: Single-centre study in a teaching hospital in Spain.
Patients: 40 patients with a first ST-elevation myocardial infarction, single-vessel disease and thrombolysis in myocardial infarction (TIMI) grade 3 flow (stent in 33 patients) underwent rest and low-dose dobutamine CMR 7 (SD 1) and 184 (SD 11) days after infarction. Microvascular perfusion was assessed at rest by visual assessment and quantitative analysis of first-pass perfusion CMR. Of the 640 segments, 290 segments subtended by the infarct-related artery (IRA) were focused on.
Results: Both 1 week and 6 months after infarction, segments with normal perfusion showed more wall thickening, contractile reserve and wall thickness, and less transmural necrosis, $p < 0.05$ in all cases. Of 76 hypoperfused segments at the first week, 47 (62%) normalised perfusion at the sixth month. However, 42 segments (14% of the whole group) showed chronic abnormal perfusion; these segments showed worse CMR indices in the late phase ($p < 0.05$ in all cases).
Conclusions: In patients with an open IRA, more than half of the segments with abnormal perfusion at the first week are normally perfused after six months. First-pass perfusion CMR shows that in a small percentage of segments, abnormal perfusion may become a chronic phenomenon—these areas have a more severe deterioration of systolic function, wall thickness, contractile reserve and the transmural extent of necrosis.

The primary objective of treatment after myocardial infarction is to restore tissue perfusion.[1] In patients with an open infarct-related artery (IRA), a lack of perfusion at the microvascular level is related to worse outcome.[1–5] The extent of abnormal perfusion may change in the months after myocardial infarction.[5 6]

Cardiovascular magnetic resonance imaging (CMR) allows state-of-the-art analysis of the heart in different clinical scenarios.[7] Basically, two methods have been described for evaluating perfusion using CMR: first-pass perfusion imaging (which analyses myocardial enhancement immediately after contrast injection)[7–11] and late-enhancement imaging (which evaluates the presence of microvascular obstruction in the middle of the necrotic area several minutes after contrast administration).[4 12 13 14] Several authors[13 14] have shown (using late-enhancement imaging) that microvascular obstruction after myocardial infarction is always a transient phenomenon.

Little is known about the evolution of microvascular perfusion after myocardial infarction analysed with first-pass perfusion CMR. We conducted this study to evaluate by means of this technique the evolution of microvascular perfusion as well as its influence on systolic function, wall thickness, contractile reserve (evaluated with stress dobutamine CMR) and the extent of necrosis (analysed with late-enhancement imaging) in the first 6 months after myocardial infarction in patients with an open IRA.

PATIENTS AND METHODS

Study group

We prospectively included 60 consecutive patients with a first ST-elevation myocardial infarction treated with thrombolytic therapy (time to reperfusion 229 (standard deviation (SD) 115) min) within the first 6 h after onset of chest pain. To avoid several confusing factors that could alter the interpretation of the microvasculature (occlusion or severe stenosis in the IRA, previous necrosis, multivessel disease, reinfarction, restenosis, etc), the inclusion criteria were as follows:

a. Stable clinical course without complications during the first 6 months
b. Single-vessel disease and a patent (thrombolysis in myocardial infarction grade (TIMI) 3 flow and residual stenosis <50%) IRA at the end of pre-discharge cardiac catheterisation and at the sixth month
c. No contraindications to CMR.

We excluded 20 patients who had multivessel disease (n = 10), TIMI flow <3 (n = 2), restenosis (n = 5), claustrophobia (n = 2) and reinfarction (n = 1). Therefore, the final study group comprised 40 patients (fig 1).

The local ethics committee approved the research protocol. Informed consent was obtained from all patients.

Cardiac catheterisation

Cardiac catheterisation was carried out 4 (SD 1) days after myocardial infarction. TIMI grade 3 flow was observed in 20 (50%) patients; a stent was placed in 33 (82%) patients in

Abbreviations: CMR, cardiovascular magnetic resonance imaging; IRA, infarct-related artery; TIMI, thrombolysis in myocardial infarction; True FISP, true fast imaging in steady-state precession

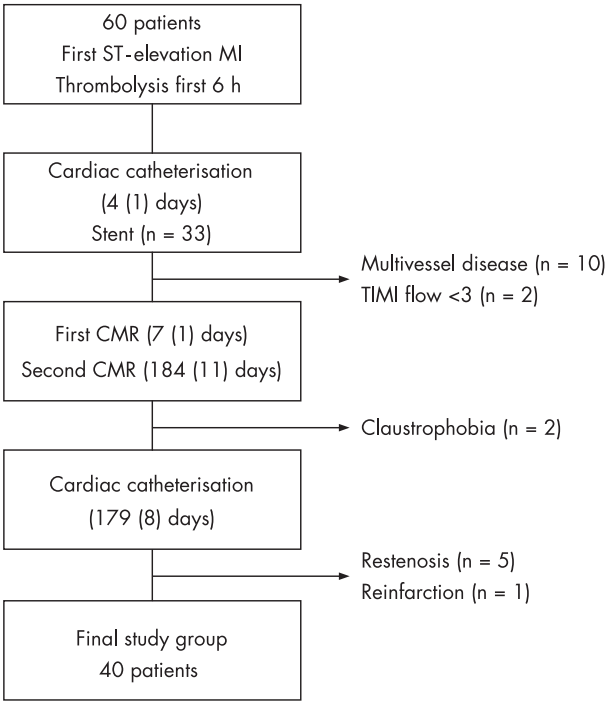

Figure 1 The final study group. CMR, cardiovascular magnetic resonance imaging; MI, myocardial infarction.

whom luminal narrowing in the IRA was >50%. At the end of the pre-discharge study, all patients showed TIMI grade 3 flow and residual stenosis <50%. Angiographic data were evaluated in a core laboratory (ICICOR, Valladolid, Spain). Cardiac catheterisation was repeated 179 (SD 8) days after myocardial infarction and TIMI grade 3 flow and residual stenosis <50% were confirmed in all patients.

Cardiovascular magnetic resonance imaging

CMR (1.5-T CMR scanner; Sonata Magnetom, Siemens, Erlangen, Germany) was carried out 7 (SD 1) days (at least 48 h after cardiac catheterisation) and 184 (SD 11) days after myocardial infarction, according to our laboratory protocol.[8 9] All images were acquired by a phased-array body surface coil during breath holds and were electrocardiogram triggered. Cine images (true fast imaging in steady-state precession (TrueFISP), repetition time/echo time 3.2/1.6 ms, flip angle 61°, matrix 256×128 and slice thickness 6 mm) were acquired in 2-chamber, 3-chamber, 4-chamber views and every 1 cm in short-axis views at rest and during intravenous infusion of low-dose (10 µg/kg/min) dobutamine.

After cine images, at least five views, a minimum of three short-axis views (basal, midventricular and apical) and two long-axis views, were recorded for rest first-pass perfusion imaging (TrueFISP, inversion time 110 ms, repetition time/echo time 190/1 ms, flip angle 49°, matrix 128×72 and in-plane spatial resolution 2.7×3.6 mm) after giving 0.1 mmol/kg of gadolinium-diethylenetriaminepentaacetic acid (Magnograf, Juste SAQF, Madrid, Spain) at a flow rate of

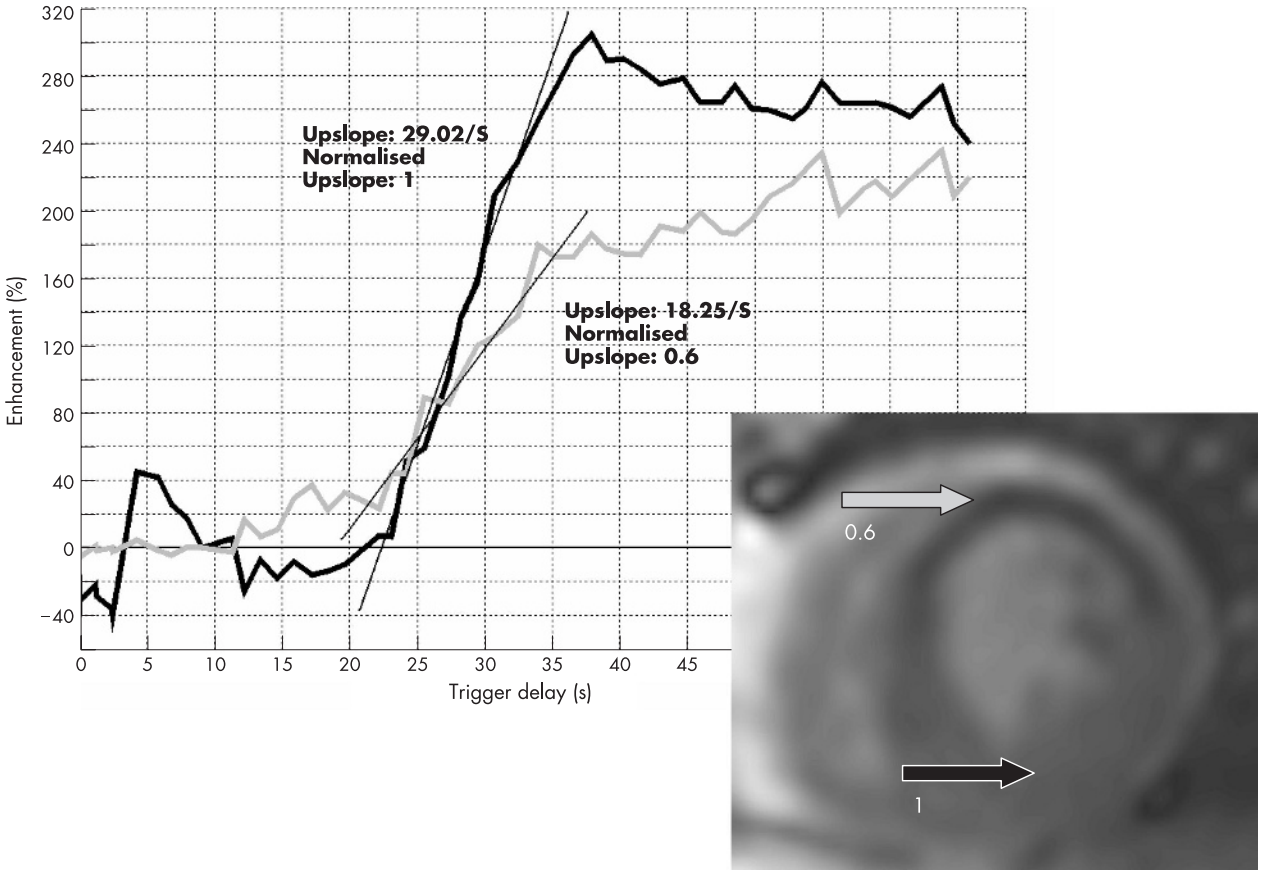

Figure 2 Quantitative analysis of perfusion. The maximal upslope of the time–intensity curves was normalised by the maximal upslope of a remote non-infarcted segment. The example used is that of a patient with an anterior infarction, normal perfusion in the remote area and delayed contrast arrival in the anterior area.

3 ml/s, and acquiring images every other beat sequentially in all views during 60–90 s. Analysis of perfusion was carried out at rest (not under stress conditions). As the perfusion sequence lasted 60–90 s, patients were instructed to hold their breath for as long as possible, taking a further breath when required and then holding their breath again.

Late-enhancement imaging was carried out 10 min after contrast injection, using a segmented inversion recovery TrueFISP sequence (repetition time/echo time 2.5/1.1 ms, slice thickness 6 mm, flip angle 50°, matrix 195×192 and in-plane spatial resolution 1.8×1.4 mm) and nullifying the myocardial signal.

CMR data analysis

CMR studies were analysed by an experienced observer blinded to all patient data and to whether the scans were performed at baseline or at the 6-month follow-up. The observer assessed the scans at least 6 months after they were carried out. Customised software (QMASS MR V.6.1.5; Medis, Leiden, The Netherlands) was used. The 16-segment model was applied.[15] The location of segments was established in cine image sequences and (to avoid mismatch) the same projections were recalled for analysing response to dobutamine, perfusion and late enhancement. The observer evaluated perfusion images on the first day and conducted the rest of the analysis the next day.

End-diastolic thickness (abnormal if $\leqslant$5.5 mm)[8 9 12] and wall thickening at rest (abnormal if $\leqslant$2 mm) and after low-dose dobutamine (abnormal if $\leqslant$2 mm)[8–10] were quantified in cine images. In our laboratory, the rationale for using a cut-off value of $\leqslant$2 mm for abnormal wall thickening[8 9] was based on receiver–operating characteristic analysis of its ability to detect dysfunctional segments (on the basis of visual assessment).

Abnormal perfusion was defined (on the basis of visual assessment) as regions showing hypoenhancement (both in short-axis and long-axis views) compared with remote non-infarcted segments in the same slice at the end of the 60–90-s acquisition period in first-pass perfusion imaging.[7–9 11] Using this method,[8] we have reported a similar ability to detect late-systolic improvement in comparison with studies conducted using a quantitative assessment method.[16]

A quantitative analysis of the perfusion was also carried out. The endocardial and epicardial contours were traced several pixels from the outer and inner borders to ensure myocardial sampling. The mean signal intensity before injection of the contrast agent was subtracted from all post-contrast data, and the upslope of the resulting time–intensity curves was calculated.[17] The maximal upslope in each segment was normalised by the maximal upslope in a remote segment (without delayed hyperenhancement and uniform wash-in over all slices), resulting in a normalised maximal upslope between 0 and 1 (fig 2). A cut-off value of <0.6 for abnormal perfusion was based on receiver–operating characteristic analysis of its ability to predict segmental recovery.

Late enhancement was considered in the case of signal intensity >2SD with respect to a remote non-infarcted area in late-enhancement imaging.[16] The transmural extent of necrosis was defined as $\geqslant$50% of wall thickness showing late enhancement.[7–11]

In the sixth-month CMR study, all indices were re-evaluated. A dysfunctional segment was considered to show a marked improvement in contractility (systolic recovery) in the case of an increase in wall thickening >2 mm from the first week to the sixth month.

In each patient, both at the first week and at the sixth month, the number of segments with abnormal CMR indices was quantified (table 1).

Table 1 Characteristics of the study group

Age (years)	57 (10)
Men	37 (92%)
Hypertension	16 (40%)
Smoking	30 (75%)
Diabetes	7 (17%)
Hypercholesterolaemia	15 (37%)
Killip class >1	6 (15%)
Anterior infarction	27 (67%)
Inferior infarction	13 (33%)
Peak CK-Mb mass (ng/ml)	258±173
Catheterisation	
Patients with stents	33 (82%)
IIb/IIIa inhibitors (in patients with stents)	14 (42%)
Magnetic resonance imaging at 1 week	
End-diastolic volume index (ml/m^2)	72 (23)
End-systolic volume index (ml/m^2)	39 (22)
Ejection fraction (%)	49 (13)
Number of segments per patient with	
Wall thickening at rest $\leqslant$2 mm	4 (2.8)
Wall thickness $\leqslant$5.5 mm	0.7 (1.3)
Contractile reserve $\leqslant$2 mm	3.2 (1.8)
Abnormal perfusion	2.4 (2.5)
Transmural extent of necrosis $\geqslant$50%	3 (2.4)
Magnetic resonance imaging at 6 months	
End-diastolic volume index (ml/m^2)	70 (27)
End-systolic volume index (ml/m^2)	35 (25)
Ejection fraction (%)	53 (14)
Number of segments per patient with	
Wall thickening at rest $\leqslant$2 mm	3.1 (2.7)
Wall thickness $\leqslant$5.5 mm	1.2 (1.8)
Contractile reserve $\leqslant$2 mm	2.2 (1.3)
Abnormal perfusion	1.2 (1.8)
Transmural extent of necrosis $\geqslant$50%	3.1 (2.4)

Values are mean (SD) or n (%).
CK, creatine phosphokinase; Mb, cardiac isoenzyme.

In our laboratory, intraobserver agreement on perfusion results was 94% ($\kappa = 0.86$) versus 96% ($\kappa = 0.88$) on systolic function results.[8 9]

Statistical analysis

Continuous data were expressed as the mean (SD) and comparisons between groups were made using two-tailed paired and unpaired t test. Discrete data were expressed as percentages. Comparisons between groups were made using χ^2 tests for discrete data (χ^2 test for trend was used for comparison of several percentages). Statistical significance was considered for $p<0.05$. SPSS V.11.0 was used.

RESULTS

Table 1 shows the baseline characteristics of the 40 patients.

Patients with (n = 20) and without (n = 20) TIMI grade 3 flow before revascularisation did not show significant differences ($p>0.4$ in all cases) with respect to the number of segments with abnormal wall thickening (3.7 (2.7) *v* 4.2 (2.9)), abnormal wall thickness (0.6 (1.2) *v* 0.7 (1.5)), lack of contractile reserve (3.1 (2.5) *v* 3.6 (3)), abnormal perfusion (2.1 (2.6) *v* 2.7 (2.5)) or transmural necrosis (2.7 (2.2) *v* 3.3 (2.6)) in the CMR study performed at the first week. Patients with time to thrombolysis >180 min and <180 min (median) showed a similar number of segments with abnormal perfusion (2.3 (2.4) *v* 2.4 (2.3) segments, p = 0.9).

Of the 640 segments evaluated, 482 had normal wall thickening and 158 showed systolic dysfunction at the first week. In the case of segments with normal systolic function, 478 (99%) showed normal perfusion and 78 of 158 (49%) dysfunctional segments showed abnormal perfusion. Of the 158 dysfunctional segments at the first week, 47 (30%) showed significant systolic recovery (increase in wall thickening >2 mm) from the first week to the sixth month.

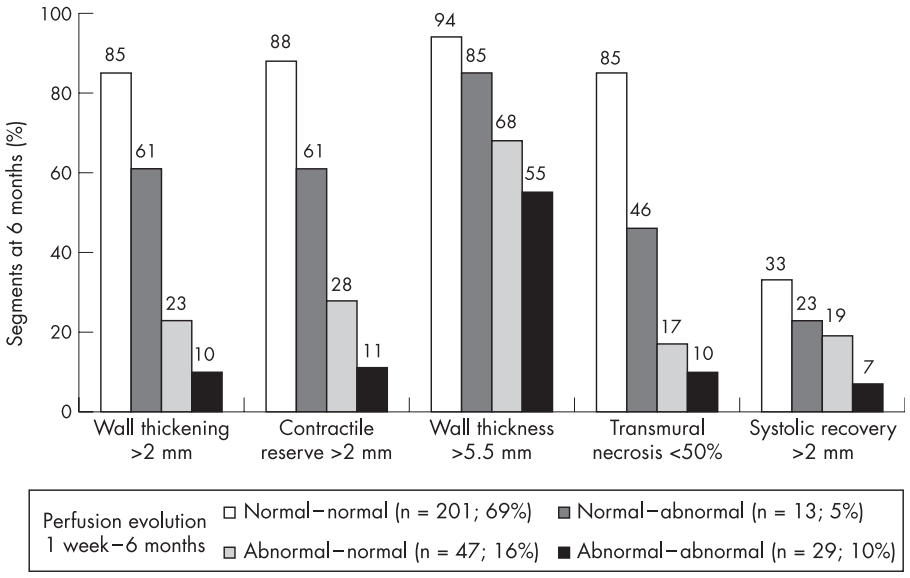

Figure 3 Percentage of segments with normal cardiovascular magnetic resonance imaging indices at the sixth month according to perfusion evolution from the first week to the sixth month; p<0.001 for the trend in all cases.

Perfusion and CMR data in the infarcted area

At the first week, of 290 segments subtended by the IRA, perfusion was normal in 214 (74%) and abnormal in 76 (26%). At the sixth month, perfusion was normal in 248 (86%) segments and abnormal in 42 (14%, p <0.0001 *v* first week).

At the first week, segments with abnormal perfusion included in the infarcted area (n = 76) showed more depressed wall thickening (0.8 (1.1) *v* 4.8 (3.4) mm, p<0.001), thinner wall thickness (8.2 (2.7) *v* 9.6 (2.7) mm, p<0.001), less contractile reserve (1 (1.4) *v* 5.7 (4) mm, p<0.001) and more extensive necrosis (80% (29%) *v* 16% (30%), p<0.001) than segments with normal perfusion (n = 214).

At the sixth month, segments with abnormal perfusion included in the infarcted area (n = 42) showed more depressed wall thickening (1.4 (1.8) *v* 4.9 (3.8) mm, p<0.001), thinner wall thickness (6.5 (2.1) *v* 8.8 (2.8), p<0.001), less contractile reserve (1.5 (2.3) *v* 5.6 (4.1) mm, p<0.001) and more extensive necrosis (77% (29%) *v* 25% (37%), p<0.001) than segments with normal perfusion (n = 248).

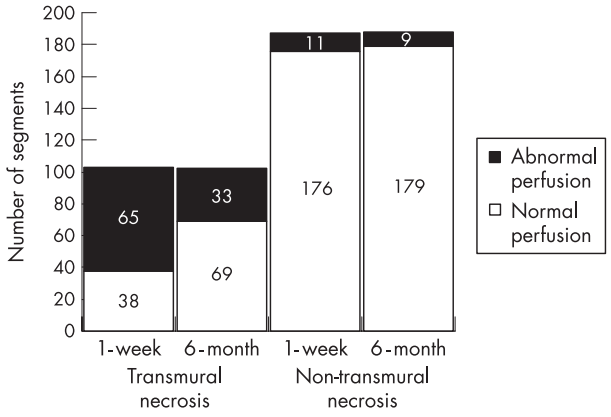

Figure 4 The percentage of segments showing transmural necrosis did not vary from the first week to the sixth month (35% *v* 35%, p = non-significant). The presence of normal perfusion did not change in segments with non-transmural necrosis (94% *v* 95%, p = non-significant), but almost doubled (37% *v* 68%, p<0.001) in those with transmural necrosis.

Implications of perfusion evolution from the first week to the sixth month

Of the 214 normally perfused segments at the first week, only 13 (5%) showed worsened perfusion at the sixth month, whereas normal microcirculation was maintained in 201 (94%) segments. Of the 76 hypoperfused segments at the first week, 47 (62%) improved perfusion at the sixth month and 29 (38%) remained abnormally perfused. Consequently, 42 (14% of the entire study group) segments showed abnormal perfusion at the sixth month.

Of the 76 hypoperfused segments at the first week, those with improved perfusion at the sixth month (n = 47) had greater wall thickness (8.8 (2.9) *v* 7.1 (2.2) mm, p = 0.003), but similar wall thickening (1 (1.4) *v* 0.8 (1) mm, p = 0.3), contractile reserve (1.4 (1.7) *v* 0.7 (1.2) mm, p = 0.08) and transmural extent of necrosis (75% (32%) *v* 81% (33%), p = 0.4) at the first week in comparison with segments that remained hypoperfused at the sixth month (n = 29).

Depending on perfusion evolution, the segments were categorised into four groups. According to this classification, a gradual decrease was observed in the percentage of segments with normal CMR indices at the sixth month (fig 3). The group of segments with chronically sustained abnormal perfusion exhibited worse CMR parameters (p<0.05 in all cases).

Perfusion and the transmural extent of necrosis in the infarcted area

The percentage of segments showing transmural necrosis did not vary from the first week (103/290, 35%) to the sixth month (102/290, 35%).

Most segments with non-transmural necrosis had normal perfusion both at the first week (176/187, 94%) and at the sixth month (179/188, 95%). However, the percentage of segments with transmural necrosis showing normal perfusion almost doubled (p<0.001) from the first week (38/103, 37%) to the sixth month (69/102, 68%; fig 4).

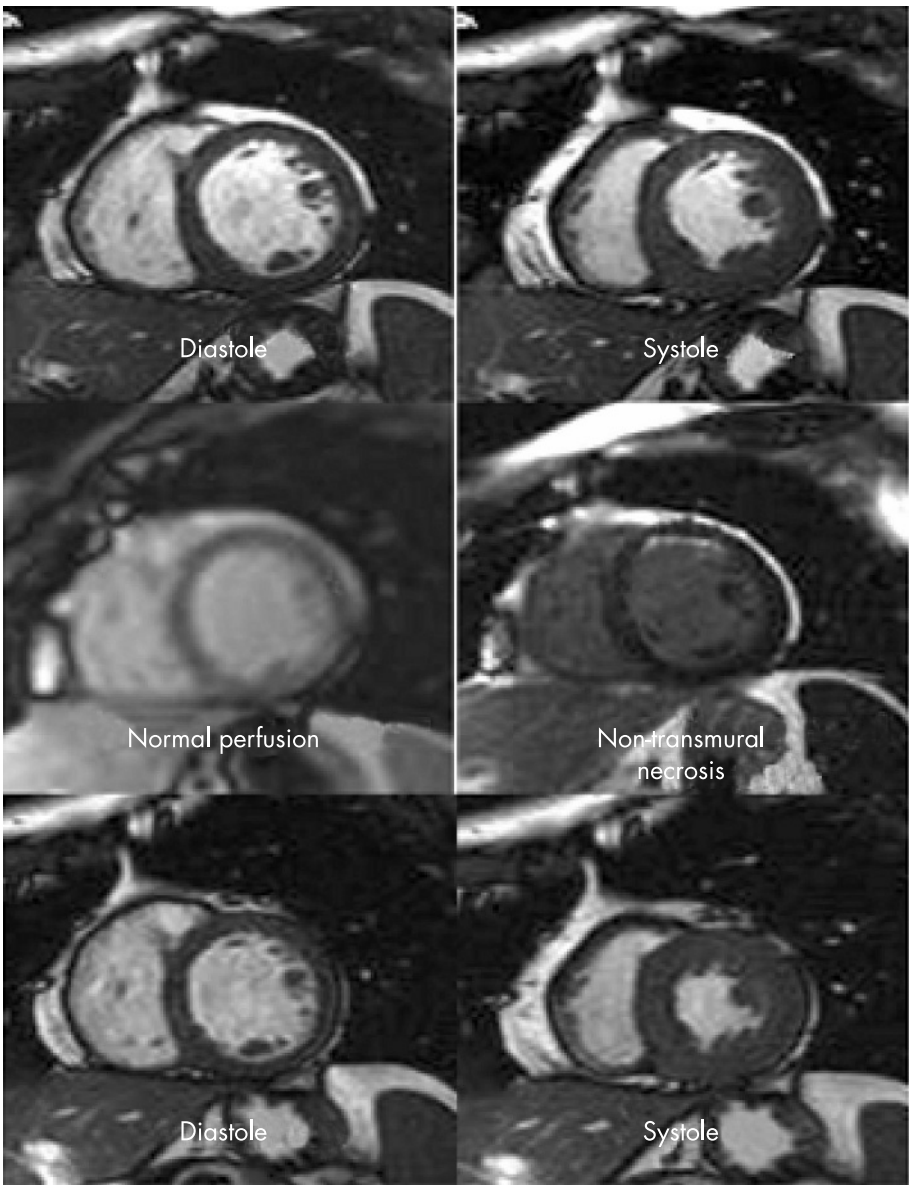

Figure 5 An anterior infarction showing abnormal systolic function (upper panels), normal perfusion and non-transmural necrosis (middle panels) at the first week. At the sixth month normal systolic function was observed (lower panels).

Quantitative assessment of perfusion

Segments with abnormal perfusion by visual assessment showed a slower upslope both at the first week (0.5 (0.2) *v* 0.8 (0.2), p<0.001) and at the sixth month (0.6 (0.2) *v* 0.8 (0.2), p = 0.04). At the first week, 90 of 290 (31%) segments subtended by the IRA showed an upslope <0.6; in these segments the upslope improved from the first week to the sixth month (0.4 (0.1) *v* 0.7 (0.2), p<0.001).

According to the 16-segment model, the percentages of segments with an upslope <0.6 in the infarcted, adjacent and remote areas at the first week were 31%, 13% and 1%, respectively. The upslope increased from the first week to the sixth month in the infarcted area (0.7 (0.2) *v* 0.8 (0.2), p = 0.001) and it did not change either in the adjacent area (0.8 (0.2) *v* 0.8 (0.2), p = 0.8) or in the remote area (0.9 (0.2) *v* 0.9 (0.2), p = 0.3).

At the sixth month, diminished upslope (<0.6) persisted in 49 segments (17%, p<0.001 *v* first week). These segments showed worse wall thickening (2.2 (3.4) *v* 5.5 (3.6) mm, p<0.001), less contractile reserve (2.2 (3) *v* 6.1 (4.2) mm, p<0.001), less wall thickness (6.8 (2.8) *v* 8.7 (2.9) mm, p<0.001) and more extensive necrosis (65% (39%) *v* 19% (34%), p<0.001) than those 241 (83%) segments with upslope ⩾0.6 at the sixth month.

DISCUSSION

To ensure reliable evaluation of perfusion after myocardial infarction, we selected a study group with a first myocardial infarction, single-vessel disease and an open IRA. In agreement with previous studies using invasive techniques,[2 5] CMR-derived normal perfusion was related to better systolic function, more extensive viable myocardium and greater systolic recovery (figs 5, 6).

Evolution of CMR-derived perfusion

Previous studies have not analysed CMR-derived data on perfusion evolution after myocardial infarction in an ideal scenario such as that represented by our study. Our findings suggest the following:

1. Spontaneous improvement of perfusion occurs after myocardial infarction.[6 13 14] More than half of the segments with abnormal microvasculature at the first week showed normal perfusion at the sixth month. Interestingly, this

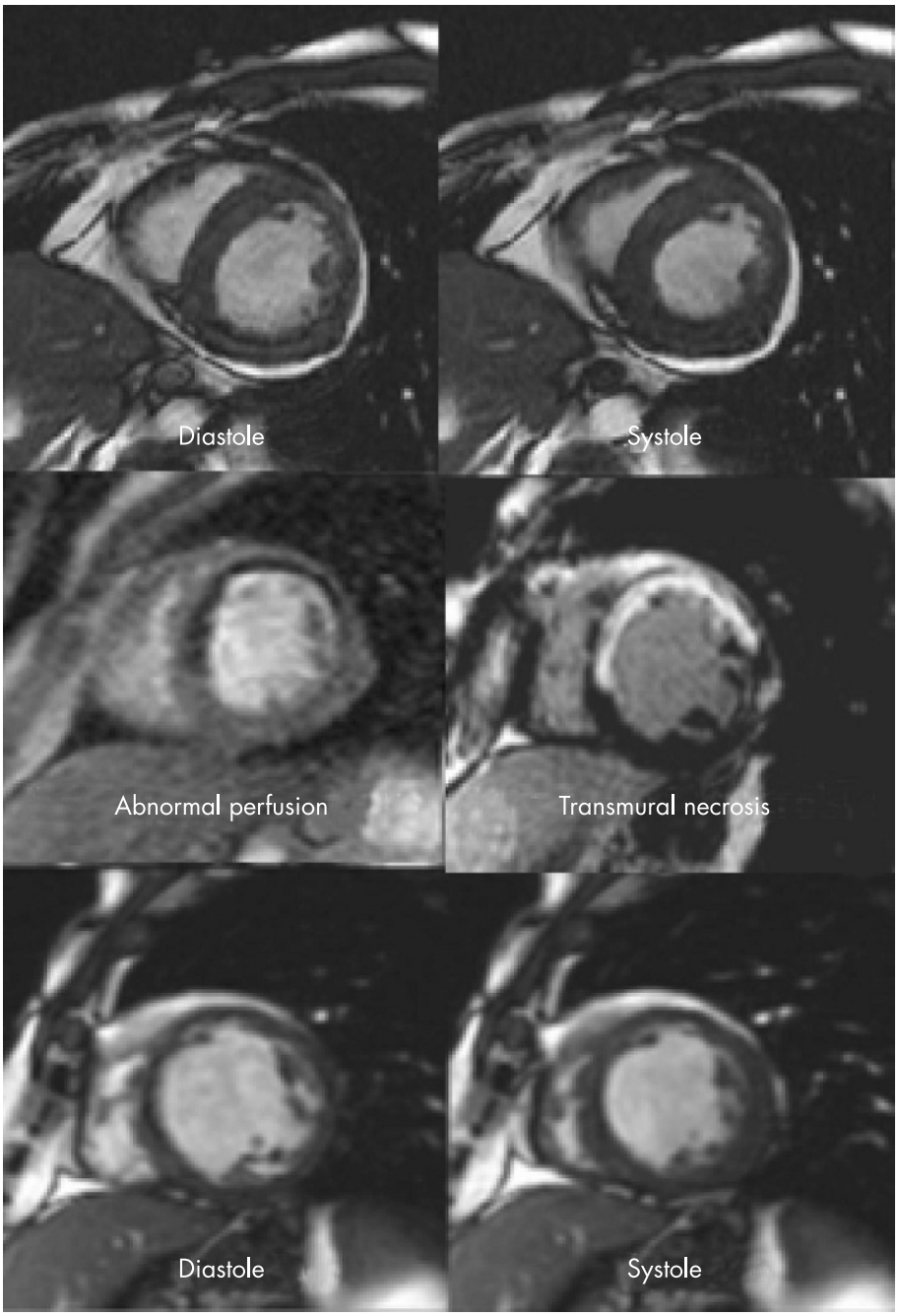

Figure 6 An anterior infarction showing abnormal systolic function (upper panels), abnormal perfusion and transmural necrosis (middle panels) at the first week. At the sixth month severe systolic dysfunction and a thin infarcted wall were observed (lower panels).

natural trend towards improved perfusion exerted beneficial effects on all parameters evaluated.

Data on the evolution of microvascular perfusion after myocardial infarction using rest first-pass perfusion CMR are scarce. Taylor *et al*[16] reported a considerable improvement in microvasculature 3 months after myocardial infarction; altered perfusion in the adjacent territory was also observed. In the present study, using a visual and quantitative assessment, we have confirmed this trend to late normalisation of perfusion; similarly, we detected hypoperfusion in as many as 13% of segments in the adjacent area.

In a different scenario of myocardial viability, hibernation, Selvanayagam *et al*[18] have reported diminished resting perfusion in dysfunctional segments with severe coronary stenosis. Improvement in perfusion after revascularisation related to systolic recovery. Similarly, in the setting of myocardial stunning, we observed that in hypoperfused segments, normalisation of perfusion related to better systolic function in the chronic phase.

2. In some cases, abnormal perfusion becomes a chronic phenomenon—this process being related to deleterious effects on all CMR parameters quantified. In fact, 14% of segments subtended by the IRA presented abnormal perfusion at the sixth month. As Hombach *et al*[14] have shown, microvascular obstruction (assessed in late-enhancement imaging) indicates a severe alteration in the microvasculature, which disappears in the months following myocardial infarction. Our results suggest that more subtle perfusion defects (detectable in rest first-pass imaging) may persist in a subset of segments. The fact that, in our study, chronically hypoperfused segments were clearly related to worse CMR indices at the sixth month is strong evidence of the existence

and the harmful effects of permanent abnormal perfusion (despite TIMI grade 3 flow).

Almost all segments with normal perfusion at the first week maintained this condition six months later. With respect to hypoperfused segments at the first CMR study, only a greater wall thickness at the first week related to normalisation of perfusion in the following months, suggesting that improvement in microvascular perfusion takes place even in segments with severe systolic dysfunction or with extensive necrosis. In fact, the absence of TIMI grade 3 flow failed to predict abnormal microvascular perfusion even in the first CMR study (carried out a few days after restoring TIMI grade 3 flow). A rapid recovery of microcirculation in the short time that elapsed between revascularisation and CMR might explain this finding. Time to thrombolysis was not related to the extent of abnormal perfusion; the wide range in time to restore TIMI grade 3 flow may explain this finding.

CMR-derived perfusion and the extent of transmural necrosis

With regard to the relationship between perfusion and the transmural extent of necrosis, our results suggest the following: (a) preserved perfusion is almost a necessary condition to prevent the presence of transmural necrosis; and (b) the rate of segments showing transmural necrosis did not vary during follow-up; however, perfusion improved (up to two thirds) even in segments with transmural necrosis.

It could be speculated that perfusion (analysed with first-pass imaging) late after myocardial infarction is in part a mirror of the transmural extent of necrosis; nevertheless, the different dynamics of both variables and the beneficial effects of late normalisation of perfusion indicate a role independent of each other and encourage further studies to clarify the clinical implications of these findings.

Limitations

The results obtained can only be generalised to patients with characteristics similar to those of our cohort.

CONCLUSIONS

In the presence of an open artery, more than half of the segments with abnormal perfusion early after infarction show normal perfusion 6 months later; this spontaneous recovery process is related to beneficial effects on systolic function, wall thickness, contractile reserve and the transmural extent of necrosis. However, in a small group of segments, abnormal perfusion remains late after infarction; these areas experience a more severe deterioration.

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Competing interests: None.

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