Visualization of Ventricular Thrombi With Contrast-Enhanced Magnetic Resonance Imaging in Patients With Ischemic Heart Disease

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Background—Ventricular thrombus formation is a frequent and potentially dangerous complication in patients with ischemic heart disease. Although transthoracic echocardiography (TTE) is generally used as diagnostic technique, we explored the role of contrast-enhanced (CE)-MRI to detect ventricular thrombi.

Methods and Results—In 57 patients with acute myocardial infarction, chronic myocardial infarction, or ischemic cardiomyopathy, MRI was performed to evaluate ventricular function (CINE-MRI) and to depict presence of myocardial necrosis and/or scarring and no-reflow areas (CE-MRI). All studies were analyzed for concomitant ventricular thrombi. CE-MRI depicted 12 mural thrombi (3.1±2.9 cm$^3$), located in left ventricular (LV) apex or adherent to anteroseptum, presenting as black, well-defined structures surrounded by bright contrast-enhanced blood. Thrombus formation on CE-MRI was related to larger end-diastolic volumes; lower ejection fractions; the region of delayed enhancement and lowest wall motion score, especially in left anterior descending coronary artery territory; and LV aneurysm formation. On CINE-MRI, thrombi were found in 6 patients. Nonvisualized thrombi were usually small (mean size 1.2±0.7 cm$^3$).

Conclusions—CE-MRI is not only an excellent technique to depict myocardial necrosis and scar tissue in patients with ischemic heart disease, but this study also suggests a better identification of LV thrombi than with presently used clinical imaging modalities, such as TTE. (Circulation. 2002;106:2873-2876.)

Key Words: magnetic resonance imaging ■ thrombus ■ echocardiography ■ heart disease ■ myocardial infarction

Left ventricular (LV) thrombus formation is a frequent complication in patients with ischemic heart disease. Although its prevalence after myocardial infarction (MI) has decreased with improved treatment strategies, recognition and appropriate treatment remain important because of risk of systemic embolization, occurring in $\approx$13% of patients. When LV thrombus is present, oral anticoagulation significantly reduces risk of embolization. Risk factors for developing LV thrombus are infarct location (ie, anterior MI), infarct size and extent, and impairment in global and regional LV function.

In clinical practice LV thrombi are diagnosed by transthoracic echocardiography (TTE). Recent studies with contrast-enhanced (CE)-MRI have shown promising results in defining presence and extent of myocardial necrosis and scarring. Because injection of paramagnetic contrast material also enhances the ventricular blood pool, the purpose of the present study was to evaluate the role of CE-MRI in detecting ventricular thrombi in patients with ischemic heart disease.

Methods

Study Population

During an 8-month period, all consecutive patients with acute myocardial infarction (<7 days’ duration), chronic myocardial infarction (>7 days’ duration), or ischemic cardiomyopathy referred for MRI to assess myocardial viability were enrolled in the present study. All studies were performed according to the guidelines of the hospital committee on medical ethics and clinical investigation. Informed consent was obtained from all patients.

MRI Protocol

Studies were performed on a 1.5-T Intera system (Philips Medical Systems). CINE-MRI, with the use of breath-hold–balanced fast-field-echo (b-FFE) technique, was performed in cardiac short-axis, vertical, and horizontal long-axis plane. CE-MRI was performed...
after intravenous injection of gadolinium-DTPA (0.2 mmol/kg body weight) with 3D-T$_1$-weighted turbo-field-echo (TFE) technique in similar imaging planes. The inversion pulse was adjusted to optimally suppress normal myocardial signal. Images were obtained every 3 to 5 minutes during 20 minutes after injection.

**MRI Analysis**

MRI studies were analyzed blinded to echocardiography results and patient identity. LV function was quantified on CINE-MRI. CE and CINE-MRI were regionally analyzed with coronary artery (left anterior descending coronary artery, left circumflex coronary artery, right coronary artery) perfusion areas, thereby dividing the LV into 17 segments.

Regional wall motion was described as normal (score: 5), mild hypokinesia (score: 4), severe hypokinesia (score: 3), akinesia (score: 2), or dyskinesia (score: 1), taking into account the segment with the lowest score in each perfusion territory. CE-MRI studies were similarly analyzed for abnormal increases in myocardial signal intensity and presence of no-reflow zones. The latter were defined as nonenhancing zones in areas with delayed enhancement. MRI studies were analyzed to assess LV thrombi. Thrombi were identified as intracavitary masses, distinguishable from papillary muscles, muscular trabeculations, and chordae. If present, signal intensity, size, and location were defined. Criteria to differentiate mural thrombi from no-reflow zones included: (a) differences in location (intracavitary versus intramyocardial); (b) differences in contrast fill-in on consecutive CE-MRI acquisitions; and (c) differences in appearance (well defined with sharp peripheral borders versus patchy, inhomogeneous, or well-defined subendocardial zones gradually thinning toward the periphery).

**Transthoracic Echocardiography**

Patients received 2D-TTE within one day of MRI study, with a Vingmed System Five (General Electric), Vingmed Vivid7, or an Agilent Sonos5500. Studies were independently analyzed from MRI findings. Thrombus was defined as an echodense mass within LV cavity with margins distinct from LV wall and distinguishable from technical artifacts and papillary muscles in at least two different echocardiographic views.

**Statistical Analysis**

Results are shown as mean±SD. A probability value of <0.05 was considered as statistically significant. Unpaired Student’s $t$ test was used to compare patients with and without thrombus on CE-MRI. An ANOVA test was used to compare presence or absence of thrombus in relation with regional wall motion.

**Results**

In 42 of 57 consecutive patients no thrombus was detected. In 3 of 57 patients, TTE suggested an apical thrombus, which could not be confirmed on MRI. On CE-MRI, in 12 of 57 patients a mural thrombus (size ranging 0.5 to 8.6 cm$^3$) was found (apex: 10, anteroseptal wall: 2), appearing as a dark intracavitary mass clearly distinguishable from the contrast-enhanced blood (Table 1, Figure 1/Movie I). Of thrombi visible on CE-MRI, 6 and 5 of them were visible on CINE-MRI and TTE, respectively. Thrombi on CINE-MRI presented a similar signal as the adjacent myocardium. Size of nonvisualized thrombi on CINE-MRI ranged from 0.5 to 2.5 cm$^3$ (mean 1.2±0.7 cm$^3$). Size of nonvisualized thrombi on TTE ranged from 0.8 to 8.0 cm$^3$ (mean 3.0±3.2 cm$^3$), and 6 of them were located in LV apex.

Presence of regional dyskinesia or akinesia was associated with a significantly higher number of thrombi than mildly hypokinetic ($P=0.0075$ versus dyskinesia and $P=0.023$ versus akinesia) or normally contracting regions ($P=0.0015$ versus dyskinesia and $P=0.0043$ versus akinesia) but not compared with severe hypokinetic regions ($P=0.074$ versus dyskinesia and $P=0.23$ versus akinesia). Presence of myocardial enhancement was associated with a significantly higher number of adjacent thrombi than regions without enhancement ($P=0.0024$). As shown in Table 2, thrombus formation on CE-MRI was related to larger end-diastolic volumes, lower ejection fractions, delayed myocardial enhancement, low wall motion scores in left anterior descending coronary artery territory, and presence of apical aneurysm. In 11 of 12 patients, oral anticoagulation was subsequently started, and in 9 patients follow-up was available (Figure 2).
Discussion
Ventricular thrombi are not only a frequent finding on CE-MRI (21%), but this technique visualizes a substantially higher number of thrombi than CINE-MRI and TTE. Because of the homogeneous, strong enhancement of the LV cavity after gadolinium-DTPA administration, abnormal intraventricular structures will have a dark appearance. This allows the visualization of small thrombi (<1 cm³) invisible on CINE-MRI and TTE (eg, when trapped within trabeculations, such as in Figure 1C). On TTE, large (usually apical) thrombi are often difficult to image, which is likely related to insufficient image quality and problems assessing the LV apex, especially when aneurysmatic (near-field probe). Non-

| TABLE 2. Comparison of Patient Characteristics Between Positive and Negative Group |
|---------------------------------|---------------------------------|-----------------|---|
| Patient Characteristic           | Patients With Thrombus Assessed | Patients Without Thrombus Assessed | P  |
| Age/Sex                         | 59.7±12.5 (10 M, 2F)            | 62.2±9.9 (36 M, 9 F)               | 0.40 |
| End-diastolic volume, mL         | 209±67                          | 170±60                        | 0.010 |
| Ejection fraction, %             | 34±14                           | 46±16                         | 0.039 |
| Ischemic heart disease type      |                                 |                               |     |
|      Acute myocardial infarction  | 1                               | 10                            | ... |
|      Chronic myocardial infarction| 4                               | 7                             | ... |
|      Ischemic cardiomyopathy     | 7                               | 28                            | ... |
| Areas of delayed enhancement, n (%) |                                 |                               |     |
|      LAD                         | 11 (92)                         | 23 (51)                       | 0.014 |
|      LCx                         | 4 (33)                          | 10 (22)                       | 0.51 |
|      RCA                        | 6 (50)                          | 26 (58)                       | 0.81 |
| Areas of no-reflow, n (%)        |                                 |                               |     |
|      LAD                         | 4 (33)                          | 4 (9)                         | 0.081 |
|      LCx                         | 0                               | 1 (2)                         | 0.63 |
|      RCA                        | 0                               | 2 (4)                         | 0.47 |
| Wall motion score               |                                 |                               |     |
|      LAD                         | 2.0±0.8                         | 3.3±1.5                      | 0.013 |
|      LCx                         | 3.7±1.2                         | 4.3±0.9                      | 0.14 |
|      RCA                        | 3.5±1.5                         | 3.3±1.3                      | 0.72 |
| Ventricular aneurysm, n (%)     | 4 (33)                          | 2 (4)                         | 0.023 |

LAD indicates left anterior descending coronary artery; LCx, left circumflex coronary artery; and RCA, right coronary artery.

Figure 1. A, Typical presentation of large apical thrombus on CE-MRI (patient 7), short-axis view (left), vertical long-axis view (right). Thrombus visible as dark, intracavitary structure (dark arrow). Presence of an almost completely transmural scar in LV apex (white arrow). Thrombus was visible on CINE-MRI, not on TTE. B, Small apical thrombus not visible on CINE-MRI or TTE (patient 10). Horizontal long-axis view (above), vertical long-axis view (below). CINE-MRI (Movie I, II) shows apical dyskinesia, but no thrombus. On CE-MRI, small apical thrombus is visible (black arrow). Presence of transmural scar in LV apex (white arrow). C, Small thrombus adjacent to anteroseptal wall (patient 2). Small, black thrombus trapped in myocardial trabeculations is well visible on CE-MRI (arrow), but not visible on short-axis and vertical long-axis CINE-MRI (Movie III) or on TTE.

Figure 2. Small septal thrombus (arrow) visible on CE-MRI (patient 3, above). After 2 weeks of oral anticoagulation, no residual thrombus is visible (below).
visualized thrombi on CINE-MRI are much smaller than on TTE. Presence of slow and turbulent flow patterns in dysfunctional wall segments and lack of contrast between mural thrombus and adjacent myocardium, even with the new steady-state CINE-MRI techniques, explain why small thrombi can be invisible on CINE-MRI. Because false-positive findings are not infrequent on TTE, MRI might be advantageous to rule out thrombi in those patients.

Because of the lack of histopathological validation of CE-MRI findings, we compared patients with and without thrombus formation on CE-MRI. Presence of thrombi was significantly related to the region of most severe functional impairment and/or the region with myocardial enhancement (ie, infarction or scarring). It should be mentioned that the criteria to differentiate no-reflow zones from mural thrombi are not definite, and thus differentiation may not always be straightforward. Because fill-in of no-reflow zones might take an hour or more, this criterion is not always useful for general clinical studies with a limited imaging time. As found by Paydarfar et al, who used T1-weighted MRI before and after contrast administration, organized thrombi may show peripheral, inhomogeneous enhancement. Because CE-MRI uses an additional inversional pulse to suppress the signal of normal myocardium, the relaxation of thrombotic tissue will also be altered. Further research and histopathological correlation is needed to evaluate the role of CE-MRI to differentiate subacute from organized clots.

In conclusion, the present study results stress the unique position of CE-MRI to assess patients with ischemic heart disease. Not only can the impact of the ischemic event on myocardial morphology and function be precisely assessed, but a frequent complication such as thrombus formation can also be diagnosed on CE-MRI. Moreover, in combination with CINE-MRI, perfusion-MRI, and possibly in the near future, MRI of the coronary arteries, a more complete evaluation can be made with this single noninvasive technique.

References

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