Incidence, diagnostic methods, and evolution of left ventricular thrombus in patients with anterior myocardial infarction and low left ventricular ejection fraction: A prospective multicenter study

Q3 Philippe Meurin, MD, a Virginie Brandao Carreira, MD, b Raphaële Dumont, MD, a Alain Shqueir, Olivier Milleron, MD, d,e Benjamin Safar, MD, d,e Sergio Perna, MD, f Charles Smadja, MD, g Marc Genest, MD, h Jérôme Garot, MD, i Bernard Carette, MD, j Laurent Payot, MD, k and Jean Yves Tabet, MD a, For the Collège National des Cardiologues des Hôpitaux Français and the Collège National des Cardiologues des Hôpitaux Français Villeneuve Saint Denis, France; Jossigny, France; Esbly, France; 10 rue du Général Leclerc, Montfermeil, France; Meaux, France; Tournan en Brie, France; Provins, France; Massy, France; Reims, France; and Montreuil sous bois, France

Background and objectives We aimed to assess the incidence and evolution of left ventricular (LV) thrombi in a high-risk population of patients with LV systolic dysfunction after anterior myocardial infarction (ant-MI). We also compared the accuracy of transthoracic echocardiography (TTE) and cardiac magnetic resonance imaging with contrast-delayed enhancement (CMR-DE) in detecting LV thrombi.

Methods We prospectively included 100 consecutive patients with LV ejection fraction (LVEF) <45% at the first TTE performed <-7 days after ant-MI. A second evaluation with TTE and CMR-DE (by blinded examiners) was performed at 30 days. A third TTE and assessment of clinical status were performed between 6 and 12 months after ant-MI.

Results Patients (males 71%; mean age 59.1 ± 12.1 years; mean LVEF 33.5% ± 6.0%) were included at a median of 5.5 days (interquartile range 25th-75th percentile 4.25-6.0 days) after ant-MI. Thrombi were detected among 26 (26%) patients at a median of 12.0 days after ant-MI (7 patients at 1-7 days after MI; 15 at 8-30 days; and 4 after day 30). Sensitivity and specificity for LV thrombi detection were 94.7% and 98.5%, respectively, for TTE as compared with CMR-DE. Most thrombi (n = 24; 92.3%) disappeared after triple antithrombotic therapy (vitamin K antagonist in addition to dual antiplatelet therapy).

Conclusion Left ventricular thrombus is a frequent complication after ant-MI with systolic dysfunction. When a search for thrombus is prespecified, the accuracy of TTE is high as compared with CMR-DE. The best antithrombotic strategy is not known.

From the aCentre de Réadaptation cardiaque de la Brie Les Grands Prés, 27 rue Sainte Christine, Villeneuve Saint Denis, France; bMarne La Vallée Hospital, 4 cours de Gondoire, Jossigny, France; cCollege National des Cardiologues Français and Cabinet Médical, Esbly, France; dLe Raincy-Montermeil Hospital, 10 rue du Général Leclerc, Montfermeil, France; eCollège National des Cardiologues des Hôpitaux Français, Meaux Hospital, 6 rue Saint France, Meaux, France; fTournois Clinic, 2 rue Jules Leffebvre, Tournan en Brie, France; gIsen Binet Hospital, route Chalautre, Provins, France; hPrivate Hospital Jacques Cartier, Centre Hospitalier Universitaire Paris Sud-ICPS, Générale de Santé, 6 Ave du Noyer Lambert, Massy, France; iCourlay Clinic, 38 rue Courlay, Reims, France; and jAndré Grégoire Hospital, 56 Boulevard de la Boissière, Montreuil sous bois, France.

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Reprint requests: Philippe Meurin, MD, Centre de Réadaptation cardiaque de la Brie Les Grands Prés, 27 rue Sainte Christine, 77174 Villeneuve Saint Denis, France.

E-mail: philippemeurin@hotmail.com

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low sensitivity for TTE, 20% to 25%.12,15 This finding is a concern because TTE is the examination regularly performed in daily practice to search for LV thrombi.

We conducted a prospective multicentric study of patients at high risk of LV thrombus, that is, those with LV ejection fraction (LVEF) <45% after ant-MI.11 We compared TTE and CMR-DE in terms of LV thrombus discovery at day 30 and assessed clinical and echocardiographic variables at 6 to 12 months of follow-up.

Methods
Study population
This was a prospective multicentric study (5 public hospitals, 1 private hospital, and 1 cardiac rehabilitation center). Between January 1, 2011, and August 31, 2013, all consecutive patients with acute anterior wall STEMI defined as follows were screened during hospitalization: characteristic symptoms of myocardial ischemia associated with persistent electrocardiographic ST elevation at the J point in 2 adjacent precordial leads (0.2 mV in leads V2 and V3 or 0.1 mV in the other precordial leads, V1 or V4-V6) and subsequent release of biomarkers of myocardial necrosis.16,17 In addition, these patients had to have LVEF <45% at the first TTE (TTE1), performed during the first 7 days after ant-MI. The only exclusion criterion was CMR contraindication based on the 1975 Declaration of Helsinki as reflected in “a priori” approval by the institution’s human research committee.

Transthoracic echocardiography
After the inclusion of TTE1, patients underwent a mandatory second assessment including TTE (TTE2) and CMR-DE at a median of 30 days (authorized range: 20-40 days) after ant-MI. The 2 examinations had to be performed as closely as possible to each other. All TTEs were performed and interpreted jointly by 2 experienced operators of the cardiac rehabilitation center, with blinded to CMR results. A predesignated third echocardiographist was consulted in case of discordance, which occurred only once. The patient’s cardiologist performed a third TTE (TTE3) at 6 to 12 months after inclusion. Moreover, to have a better understanding of the evolution of the thrombi, in patients in whom a LV thrombus had been discovered, a TTE was performed at least once a week for 4 weeks. Furthermore, intermediate TTEs were encouraged in all patients (particularly between TTE1 and TTE2) but not mandatory. All TTE2 examinations involved use of a VIVID 7-type echograph (2.5-MHz gauge, second harmonic imaging; Vingmed GE).

Left ventricular thrombi were considered all masses with echogenicity greater than that of blood, with well-defined edges distinct from the endocardium, contiguous to an akinetic or dyskinetic myocardium segment and present during the whole cardiac cycle on ≥2 different views.11,18 A thrombus was considered protruding when it projected predominantly into the LV cavity and mural when it appeared flat and parallel to the endocardial surface.11 The thrombus area was measured by planimetry on a 4-chamber apical view. Left ventricular ejection fraction was measured by the Simpson method.19 Importantly, we prespecified on the examination prescription that possible LV thrombus was part of the clinical indication for TTE.

Contrast-delayed enhancement
As mentioned above, CMR-DE was performed at a median of 30 days (authorized range: 20-40 days) after ant-MI, as closely as possible to the TTE2. Because LV thrombus is rare in patients with good global systolic function,11 we prespecified that CMR-DE would be unnecessary (and therefore unethical in a survey) for patients with LVEF ≥50% on intermediate echocardiography (between TTE1 and TTE2).

Contrast-delayed enhancement involved a 1.5 T scanner (Magnetom Espree; Siemens, Erlangen, Germany). Contiguous short-axis locations encompassing LV from the base to the apex were acquired in the cine steady-state free precession sequence. Each slice was acquired during 1 short breath hold (7-12 seconds each, depending on the heart rate). Typical imaging parameters for cine CMR were field of view, 300 to 360 mm; slice thickness, 6.0 mm; 3.1-ms repetition time; 1.6-ms echo time; flip angle, 60°; image matrix, 256 x 156; and temporal resolution, 30 to 40 ms. Delayed enhancement images were obtained 10 to 15 minutes after administration of a gadolinium-based contrast agent (Dotarem, 0.1-0.2 mmol/kg; Guerbet) by a 2-dimensional segmented inversion-recovery gradient-echo pulse sequence, with slice position identical to the cine images (field of view, 300-360 mm; slice thickness, 6.0 mm; inversion time, 200-280 ms). Left ventricular thrombi on CMR were defined as filling defects within the LV cavity, typically adherent to regions of abnormal wall motion (hypokinesis, akinesis, or dyskinesis) on cine sequences and confirmed by late DE.

Endocardial and epicardial borders were outlined manually on short-axis end-diastolic and end-systolic cine images, and LV end-systolic volume and LV end-diastolic volume were determined, and LVEF was calculated.

Evaluators were blinded to echocardiography results. We prespecified that LV thrombus detection was part of the clinical indication for CMR-DE.

Follow-up
After hospital discharge, 88 patients received care at the cardiac rehabilitation center, then patients were followed up by their cardiologists, until TTE3 was performed 6 to 12 months after the ant-MI. The other 12 patients were followed up by their cardiologist at hospital discharge, but, as detailed above, even in these 12 patients, all TTEs were performed at the cardiac rehabilitation center.
For patients with an LV thrombus, exercise training sessions were stopped, until the resolution of the thrombus and VKA therapy was advised, with an international normalized ratio target of 2 to 3 for ≥ 6 months, in addition to dual antiplatelet therapy.

Stroke was defined as new-onset focal or global neurologic deficit caused by ischemia or hemorrhage as assessed by appropriate imaging (CT or MRI) within or around the brain and lasting for > 24 hours or leading to death. According to the criteria of thrombolysis in the TIMI trial, major bleeding was defined as a decrease in hemoglobin (Hg) level > 5 g/dL, intracranial hemorrhage or > 15% absolute decrease in hematocrit level, or cardiac tamponade and minor bleeding as a decrease in Hg level of 3 g/dL or > 10% decrease in hematocrit level from an identified site; > 4 g/dL decrease in Hg level if the bleeding site was not identified; or spontaneous gross hematuria, hematemesis, or hemoptysis.

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Statistical analysis
As in the largest prospective study of the topic and comparing CMR-DE and TTE with 17 LV thrombi at baseline, we aimed to compare CMR-DE and TTE with ≥ 18 LV thrombi detected. We finally ended inclusions after comparing the 2 techniques with 19 LV thrombi (which required 100 patients) (see Figure 1).

Data are reported as mean ± SD or median (interquartile range 25th-75th percentile [IQR 25-75]) for continuous variables and n (%) for categorical variables. Unpaired Student t test and Fisher exact test were used to compare differences between continuous and categorical variables, respectively. P < .05 was considered statistically significant.

Results
Patients
Between January 1, 2011, and August 31, 2013, we included 100 consecutive patients (Figure 1). The mean age was 59.1 ± 12.1 years, and 71% were male. The mean baseline LVEF was 33.5% ± 6.0% (Table 1). All patients except 1 (99%) underwent coronary angiography; 88% underwent primary percutaneous intervention (PCI) with stenting and for 84, this involved the left anterior descending artery (LAD); for 15, the circumflex; for 2, the right coronary artery; and for 5, the left main artery. Twelve patients did not undergo PCI; 2 had a pure LAD thrombosis, which was treated with anticoagulants; 9 presented at 18 to 24 hours after onset.
Detection of LV thrombus by days after ant-MI. Days after acute anterior myocardial infarction.

Table I. Characteristics of patients with and without thrombus

<table>
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<tr>
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<th>Thrombus (n = 26)</th>
<th>No thrombus (n = 74)</th>
<th>P</th>
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<tr>
<td>Age, years, mean ± SD</td>
<td>57.4 ± 12.7</td>
<td>59.7 ± 11.9</td>
<td>.40</td>
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<tr>
<td>Male gender</td>
<td>20 (76.9)</td>
<td>32 (43.2)</td>
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<td>Current smoker</td>
<td>12 (46.2)</td>
<td>31 (41.9)</td>
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<td>Diabetes</td>
<td>3 (11.5)</td>
<td>17 (23.0)</td>
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<td>Known hypertension</td>
<td>7 (26.9)</td>
<td>26 (35.1)</td>
<td>.44</td>
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<tr>
<td>Hypercholesterolemia</td>
<td>5 (16.9)</td>
<td>3 (4.4)</td>
<td>.12</td>
</tr>
<tr>
<td>BMI, kg/m², mean ± SD</td>
<td>26.7 ± 5.1</td>
<td>26.5 ± 5.0</td>
<td>.90</td>
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<tr>
<td>Preexisting atrial fibrillation</td>
<td>1 (3.8)</td>
<td>2 (2.7)</td>
<td>.77</td>
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<tr>
<td>Coronary angiography</td>
<td>25 (96.2)</td>
<td>74 (100)</td>
<td>.10</td>
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<tr>
<td>Primary PCI</td>
<td>20 (77.0)</td>
<td>68 (92.0)</td>
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<td>LAD PCI</td>
<td>18 (69.2)</td>
<td>66 (89.2)</td>
<td>.02</td>
</tr>
<tr>
<td>Left main PCI</td>
<td>1 (3.8)</td>
<td>4 (5.5)</td>
<td>.75</td>
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<tr>
<td>PCI on several vessels</td>
<td>4 (15.3)</td>
<td>15 (20.2)</td>
<td>.57</td>
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<tr>
<td>LVEF, %, mean ± SD</td>
<td>32.3 ± 4.6</td>
<td>34.0 ± 6.4</td>
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<tr>
<td>LVEF, %, mean ± SD</td>
<td>34.1 ± 6.6</td>
<td>39.0 ± 9.2</td>
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<td>LVEF, %, mean ± SD</td>
<td>37.0 ± 10.4</td>
<td>43.6 ± 9.9</td>
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<td>Drug therapy at TTE1</td>
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<td>Aspirin</td>
<td>26 (100)</td>
<td>74 (100)</td>
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<tr>
<td>Clopidogrel</td>
<td>15 (57.7)</td>
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<td>8 (30.8)</td>
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<td>β-blocker</td>
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<td></td>
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<tr>
<td>Ibutilidine</td>
<td>1 (3.8)</td>
<td>1 (1.4)</td>
<td>.45</td>
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<td>ACE-I</td>
<td>26 (100)</td>
<td>73 (95.9)</td>
<td>.92</td>
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<td>Statin</td>
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<td>74 (100)</td>
<td>.10</td>
</tr>
<tr>
<td>MRA inhibitor</td>
<td>16 (61.5)</td>
<td>43 (58.3)</td>
<td>.77</td>
</tr>
</tbody>
</table>

Data are n (%) unless indicated. Abbreviations: BMI, body mass index; ACE-I, angiotensin-converting enzyme inhibitor; MRA, mineralocorticoid receptor antagonist.

Incidence of LV thrombus at baseline and at follow-up

A total of 26 patients (26%) had an LV thrombus that was discovered a median of 12.0 days (IQR25-75 7.2-28.7) after anti-MI: 6 at inclusion, 16 at days 8 to 30 after ant-MI, and 4 others after day 30 (Figure 2). For 24 (92.3%), the thrombus had disappeared at TTE3. On the day of discovery, the mean thrombus area was 2.1 ± 1.7 cm²; and 65% were protruding, 35% mural, and 15% mobile (Table II). Importantly, no LV thrombus was discovered in the 9 patients in whom CMR-DE had not been performed because of LVEF ≥50% on intermediate echocardiography. For patients with and without LV thrombus, the mean LVEF was 32.3% ± 4.6% and 34.0% ± 6.4% at TTE1 (P = .22), respectively; and 34.1% ± 6.6% and 39.0% ± 270 9.2% at TTE2 (P = .01) and 37.0% ± 10.4% and 43.6% ± 271 9.9% at TTE3 (P = .006) (Table I). 272

Comparison of TTE and CMR-DE in detecting LV thrombus

For all patients, TTE1 was performed at a median of 5.5 days (IQR25-75 4.25-6.0) and TTE2 at a median of 30.0 days (IQR25-75 25.2-32.0) after ant-MI (Figure 1). During follow-up, 3 patients died, and 2 underwent transplantation, so 95 273 patients underwent TTE3 at a median of 270 days (IQR25-75 210-350) after ant-MI. For 78 (78%) patients, CMR-DE was performed at a median of 30.0 days (IQR25-75 25.0-33.1) after ant-MI. For the 22 other patients, CMR-DE was unnecessary because of LVEF ≥50% measured at intermediate echocardiography (between TTE1 and TTE2) (n = 9) 283 or was not possible (patient not stable enough to undergo CMR n = 1; patient interrupting the examination because of anxiety n = 1; or pacemaker or cardioverter defibrillator implanted before that day n = 11) (Figure 1). 287

Of the 19 thrombi seen on CMR-DE, 18 were visible on TTE (Table III). For 1 patient, TTE missed a small mural apical thrombus (area 0.5 cm²), and for one other 290 patient, TTE suggested a thrombus, which could not be confirmed on CMR-DE. Therefore, the sensitivity and specificity of TTE2 compared with CMR-DE were 94.7% and 98.3%, respectively. 293

Clinical follow-up

During the median 270 days of clinical observation, 2 296 patients underwent cardiac transplantation, and 3 died (due to sudden death at day 60; a recurrent subdural hematoma at day 52 in an 80-year-old woman with an LV thrombus and not receiving a VKA; and at day 44, early after coronary artery bypass grafting). No patient was lost to follow-up. 302
One thromboembolic event occurred (arterial inferior limb and splenic embolism at day 15 after ant-MI) in a 72-year-old patient with a large protruding LV thrombus (area 9 cm²) discovered at day 6 after ant-MI, despite the introduction of low-molecular-weight heparin (enoxaparin 100 IU/kg twice a day as a bridge before reaching an international ratio ≥2) and VKA as soon as the thrombus was discovered. The evolution was favorable.

Five severe hemorrhagic events occurred: 2 (7.7%) among the 26 patients receiving triple antithrombotic therapy (1 patient 67 years old with posttraumatic intracerebral hematoma of favorable evolution and 1 with rectorradiata due to unknown colorectal cancer) and 3 (4%) among 74 others not receiving VKA (1 each with severe rectorradiata, psosas hematoma, and the recurrent lethal subdural hematoma mentioned above).

In total, 7 patients were hospitalized for decompensated heart failure during follow-up. Overall, 15 patients required implantation of an internal cardiac defibrillator and/or a cardiac resynchronization therapy; 6 others required cardiac surgery: 2 heart transplantation (mentioned above), 2 coronary artery bypass grafting (1 died), 1 mitral valvular replacement, and 1 interventricular communication correction. Finally, cancers were discovered in 3 patients (1 each of colorectal cancer, mentioned above; lung cancer; and laryngeal cancer).

### Discussion

Our results indicate that the incidence of LV thrombi among patients after major acute anterior-wall MI is high (26%) and that this complication does not seem to be prevented by potent dual antiplatelet therapy. This result should not be surprising. Indeed, our study was precisely designed to evaluate the incidence of LV thrombi in patients with the 2 main risk factors of this complication occurrence: preferential location at the apical scar of an ant-MI and low LVEF. In former studies, LV thrombi occurred in 5% to 10% of unselected cases of ant-MI, and in the ASTAMI trial, among 100 patients who survived ant-MI with moderate LV dysfunction (mean LVEF 45%), 15 LV thrombi occurred. In the present study, patients had a very low LVEF (equal to 33.5% ± 6.0% at baseline) after ant-MI. Thus, this result confirms that the lower the LVEF, the higher the thrombus incidence.

The second reason why this high incidence should not be expected is that our study involved ≥3 TTE searches for LV thrombi, so we could precisely assess the natural history of thrombus formation and resolution: most of the LV thrombi (25/26) occurred during the first 6 weeks after the MI, half (13/26) disappeared within 1 month, and nearly all (24/26) had disappeared at day 270. The last reason explaining this high thrombus incidence is that, importantly, a search for LV thrombus was prespecified on the TTE form.

The second objective of our study was to assess the accuracy of TTE as compared with CMR-DE in detecting LV thrombi. This is an important question because even if it is considered the criterion standard, CMR-DE has 2 major flaws: high cost and low accessibility. Furthermore, LV thrombus can develop at various times after ant-MI. Thus, if TTE is accurate, performing regular TTEs rather than a single CMR-DE could be useful in patients with normal echogenicity. To our knowledge, only 4 studies have tried to perform such a comparison. Two were retrospective, which does not allow for a fair comparison. Indeed, TTE is more operator dependent than is CMR-DE. This important notion is highlighted by the finding that echocardiographic performance varies by indication: if LV thrombus search is prespecified, sensitivity is multiplied by 2 (60% vs 26%) and positive predictive value by 3 (75% vs 21%) as compared with unfocused routine TTE.

In 2 prospective studies, 41 LV thrombi were detected by CMR-DE; TTE had surprisingly low sensitivity, 24% and 42%, respectively, but good specificity, 95% and 86%. However, in our study, TTE accuracy was excellent, with sensitivity, 94.7% and specificity, 98.5%. The authors of the smaller study (12 LV thrombi) emphasized that echocardiographic nonvisualized thrombi were usually mural and small, which agrees with our results. Indeed, the only LV thrombus that we missed at TTE was mural and was 0.5 cm². Unfortunately, at the second prospective study, of 29 LV thrombi (17 at baseline, 12 at 4 months later), the thrombi characteristics (size, mobility, and protruding character) were not described. The very low sensitivity of TTE in this study could be due to the CMR-DE detection of small mural thrombi of less clinical interest because of low embolic risk or to methodological issues: indeed, because
this study was a substudy of the HEBE trial, in which CMR and TTE were primarily performed to detect change in regional myocardial function and not to compare their accuracy in LV thrombus detection, the low TTE sensitivity in LV thrombus detection could be due to the lack of prespecifying a thrombus search on the examination prescription. Moreover, in this study, 2% of the patients were excluded because of poor-quality CMR-DE.

In the present study, LV thrombus occurred in 22.7% of patients who underwent primary PCI versus 50% of the others (P = 0.04). Furthermore, patients with LV thrombus showed worse recovery of systolic function as shown by the LVEF evolution between TTE1 and TTE3. These 2 points could suggest worse viability in the LV apex of patients developing an LV thrombus, but this aspect was beyond the scope of our study.

Regarding LV thrombus treatment, no prospective randomized study has ever shown the efficacy of VKA; however, a meta-analysis of 7 observational studies found an association of anticoagulation therapy for 6 months and reduced rate of embolization (odds ratio 0.14, 95% CI 0.04-0.52). Therefore, despite the lack of strong evidence, it is usually recommended that patients with an LV thrombus after MI receive a VKA, without stopping dual antiplatelet therapy. However, many questions remain. First, the optimal duration of this triple therapy is not known. Repeated imaging of the left ventricle after 3 months of therapy may allow for VKA discontinuation earlier than 6 months if evidence of thrombus is no longer present, particularly with recovery of apical wall motion. Second, in the WOEST study, the use of clopidogrel without aspirin for patients already under oral anticoagulant therapy and undergoing PCI was associated with reduced risk of bleeding without increased thrombotic risk. Even if these mostly stable patients differed from the patients included in our study, these results could lead to a wider use of this new antithrombotic strategy.

Our results suggest that this triple antithrombotic therapy strategy is feasible, but, obviously, we cannot conclude whether other antithrombotic strategies would have had better or worse results.

We observed only 1 thromboembolic event (ie, 3.8% of patients with an LV thrombus), which agrees with recent findings with identical treatment. However, the rate of severe hemorrhagic complications (7.7%, n = 2) remained high.

Our study has several limitations; first, even if CMR-DE is considered a criterion standard, validation of CMR-DE findings was not histopathologically confirmed. Second, we did not use contrast echo because our objective was to test TTE accuracy in daily practice. Third, whether management of LV thrombus requires VKA therapy in addition to dual antiplatelet therapy cannot be definitive-ly concluded from this observational study and requires confirmation in a randomized trial of patients with LV thrombus after acute MI. Finally, with the study calendar, even if unlikely, LV thrombi occurring after day 30 and disappearing spontaneously before TTE3 could have been missed.

In conclusion, we show that the frequency of LV thrombus is high after acute ant-MI with LV systolic dysfunction, mostly in the first 6 weeks. As well, when LV thrombus detection is specified in the examination prescription, TTE has excellent performance as compared with CMR-DE. Therefore, in this high-risk population, regular echocardiographic follow-up is required, at least in the first 6 weeks, whereas CMR-DE should be reserved for patients in whom the LV apex is not well observed during echocardiography.

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