Prognostic significance and natural history of left ventricular thrombi in patients with acute anterior myocardial infarction: a two-dimensional echocardiographic study

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ABSTRACT  Fifty-eight patients with transmural anterior myocardial infarction were prospectively studied with serial two-dimensional echocardiography to determine the clinical implications and prognostic significance of detection of left ventricular thrombus during acute myocardial infarction, the incidence of systemic embolization, and the possible occurrence of spontaneous regression of left ventricular thrombi. Patients were not treated with anticoagulants or platelet inhibitors during the acute phase of infarction or during follow-up. Two-dimensional echocardiograms were obtained within 24 hr of myocardial infarction, every 24 hr until day 5, every 48 hr until day 15, and every month for a follow-up of 2 to 11 months (mean 7), in the surviving patients; a total of 774 echocardiograms were obtained. Left ventricular thrombi were identified in 24 (41%) of the 58 study patients, and developed within 48 hr of infarction in 11 of these patients. Ten (91%) of the 11 patients with early thrombus formation died during hospitalization or during follow-up, while only two (15%) of the 13 who developed a thrombus after 48 hr of infarction died (p < .005). Incidence of Killip class III or IV, total lactic dehydrogenase values, and extent of wall motion abnormalities were significantly higher in patients who developed a thrombus within 48 hr of infarction than in patients without thrombus. On the other hand, in patients who developed a thrombus after 48 hr of infarction, these parameters were not significantly different from those in patients who did not develop a thrombus. Spontaneous regression of thrombi was documented in three (20%) of the 15 patients who survived the acute phase of myocardial infarction. The incidence of clinically evident systemic embolic events was low: one of the 24 patients with left ventricular thrombus experienced transient ischemic attacks, and no embolic events were detected in patients without thrombus. We conclude that development of left ventricular thrombi within 2 days of acute anterior myocardial infarction occurs in patients with the most extensive infarcts and is predictive of high mortality. Our data also demonstrate that spontaneous regression of thrombi may occur. Finally, in our study patients, none of whom were treated with anticoagulants or platelet inhibitors, the incidence of clinically evident systemic embolic events was low.

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TWO-DIMENSIONAL ECHOCARDIOGRAPHY has recently demonstrated that left ventricular thrombi are common in patients with acute anterior myocardial infarction.1–5 However, the clinical implications and prognostic significance of detection of thrombi during acute myocardial infarction, the incidence of systemic embolization, and the possible occurrence of spontaneous regression of left ventricular thrombi have not yet been investigated in prospective studies conducted in a large population of patients not treated with anticoagulants. Therefore, it is not known if the detection of left ventricular thrombi during acute anterior myocardial infarction should alter the course of treatment. To address these questions, we obtained serial two-dimensional echocardiograms, at short intervals, in the acute phase and during the months after anterior myocardial infarction in a consecutive series of patients who were not treated with anticoagulants or platelet inhibitors.

Methods

Selection and characterization of patients. Fifty-nine consecutive patients admitted to the coronary care unit of the Ente Ospedaliero Ospedali Galliera within 24 hr of transmural anteri-
or myocardial infarction and who had no evidence of prior anterior myocardial infarction entered the study. A satisfactory visualization of the left ventricular cavity in the echocardiographic apical views could not be obtained in one patient, who was excluded from further study. The remaining 58 patients comprise our study population.

The patients ranged in age from 35 to 87 years (mean 63); 44 (76%) were men. The diagnosis of acute anterior myocardial infarction was documented in each patient by a typical history of chest pain, serial electrocardiographic changes, and serial elevation of serum enzymes. The electrocardiographic criterion for diagnosis of transmural infarction was the appearance of Q waves of greater than 0.04 sec in the anterior precordial leads. Total creatine kinase (CK) and CK-MB activity was measured on admission, at 12 hr after the onset of symptoms, and then every 4 hr for the next 16 hr. Total lactic dehydrogenase (LDH) activity was measured on admission, and daily for the first 3 hospital days. By clinical criteria, each study patient was assigned to the highest (most severe) Killip class6 be reached during hospitalization. In patients who developed left ventricular thrombi, Killip class was also assessed at the time of the first echocardiographic detection of thrombus. Four patients had echocardiographic documentation of a prior inferior infarction in addition to the acute anterior infarct. By design, no study patient was treated with anticoagulants or platelet inhibitors in the acute phase of infarction or during the follow-up period.

Two-dimensional echocardiography. An Advanced Technology Laboratory (ATL) Mark 300 mechanical sector scanner with a 3 MHz transducer was used to perform the echocardiographic studies. Two-dimensional echocardiographic images were recorded on a Panasonic NW-8200 videotape. A Sony U-matic videotape VO-5800 PS was used to review the studies in slow-motion and real-time modes. In each patient, two-dimensional echocardiograms were obtained within 24 hr of acute infarction, every 24 hr until day 5, every 48 hr until day 15, and every month for a follow-up of 2 to 11 months (mean 7) in the surviving patients. A total of 774 two-dimensional echocardiograms were obtained in the 58 study patients.

Images were obtained in the parasternal long- and short-axis and apical two- and four-chamber views with the use of standard transducer positions. In most studies, images were also obtained from the apical short-axis view and from sector orientations intermediate between the two- and four-chamber views. Routinely, while recording from the apical views, depth was adjusted to examine the left ventricular apex more thoroughly. Multiple gain and reject settings were also used to improve definition of the epicardium, endocardium, and margins of the thrombus. Left ventricular thrombus. Total lactic dehydrogenase level was used to diagnose when an echodense mass with defined margins was visualized contiguous to the endocardial surface, and it was clearly identifiable throughout the cardiac cycle.1,7,8 Multiple sector orientations from the apical windows were used to differentiate left ventricular thrombi from muscle trabeculations, chordal structures, and false masses that may result from oblique angulation of the ultrasound beam through the left ventricular wall.7 Agreement of two observers who were blinded to each other’s interpretation was required before an echocardiogram was considered positive for left ventricular thrombus. To minimize false-positive diagnoses, studies in which there was observer disagreement that persisted after joint review of the study were considered negative for thrombus.9 However, our protocol provided for serial two-dimensional echocardiograms obtained at short intervals in each patient, and the comparison of sequential echocardiograms was helpful in reducing the number of equivocal studies. Echocardiograms were considered equivocal, and therefore negative for left ventricular thrombus in only two of the 58 study patients.

The serial echocardiograms obtained in each patient were also analyzed with regard to shape of the left ventricular thrombus, and thrombi were classified either as mural, when they were flat and parallel to the endocardial surface, or protruding, when they were spherical and projected into the left ventricular cavity.7

Left ventricular wall motion analysis. Left ventricular wall motion was assessed from the combined analysis of the echocardiographic apical two- and four-chamber views. In these two-dimensional planes, the left ventricle was divided into nine segments that identified the left ventricular apex, the basal and midventricular regions of the interventricular septum, and the posterolateral, inferior, and lateral free walls.10,11 Wall motion in each segment was classified as normal, hypokinetic, akinetik, dyskinetic, or hyperkinetic on the basis of a visual assessment of left ventricular wall movement during systole. A wall motion index was calculated according to the method of Gibson et al.,11 in which a numerical score is assigned to each segment on the basis of the severity of the wall motion abnormalities as follows: normal, 0; hypokinetic, +1; akinetik, +2; dyskinetic, +3; and hyperkinetic, −1. A left ventricular wall motion index was then calculated by adding the scores from each segment and dividing by the number of segments analyzed. In each patient, left ventricular wall motion was assessed twice, from the echocardiogram recorded on day 1 of infarction and from the echocardiogram obtained 30 days after infarction. In patients who died within 30 days of suffering myocardial infarction, the second assessment of wall motion was made from the last echocardiogram recorded before death. In the 58 study patients, a total of 116 echocardiograms were analyzed to calculate wall motion index. Of the 1044 possible left ventricular segments, 960 (92%) were considered adequate for analysis.

Statistical analysis. Continuous data were expressed as mean ± SD. Differences between two means were analyzed by the unpaired or paired Student’s t test, as appropriate. Differences between three means were tested by the one-way analysis of variance. Differences between proportions were analyzed by the chi-square test.

Results

Incidence of left ventricular thrombi and prognosis. Left ventricular thrombi were identified in 24 (41%) of the 58 study patients, and were either confined to the left ventricular apex, or extended from the apex over the interventricular septum. The thrombus was protruding in 14 (58%) of the patients, and mural in 10 (42%). In none of the patients did the thrombus show vigorous mobility within the left ventricular cavity during the cardiac cycle. In each patient, left ventricular thrombus was associated with adjacent wall motion abnormalities. Incidence of Killip class III or IV, peak total LDH values, extent of wall motion abnormalities, and mortality were significantly higher in patients who developed thrombi than in patients who did not (table 1).

However, during the course of the study a relationship between time of development of thrombus and clinical outcome. Ten (91%) of the 11 patients who developed thrombi within 48 hr of acute infarction died during hospitalization (eight patients), or during fol-
TABLE 1
Clinical data and wall motion index in patients with and without left ventricular thrombus (LVT) after acute myocardial infarction

<table>
<thead>
<tr>
<th></th>
<th>Age (years)</th>
<th>Sex (No. of men)</th>
<th>CK (IU/l)</th>
<th>MB (IU/l)</th>
<th>LDH (IU/l)</th>
<th>Killip class III or IV (No. of patients)</th>
<th>Wall motion index±</th>
<th>Mortality (No. of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patients with LVT (n = 24)</td>
<td>63 ± 12</td>
<td>21 (88%)</td>
<td>2410 ± 832</td>
<td>275 ± 91</td>
<td>1902 ± 639</td>
<td>10 (42%)</td>
<td>0.46 ± 0.23</td>
<td>0.67 ± 0.34</td>
</tr>
<tr>
<td>Patients without LVT (n = 34)</td>
<td>64 ± 13</td>
<td>23 (68%)</td>
<td>1935 ± 886</td>
<td>232 ± 146</td>
<td>1440 ± 582D</td>
<td>4 (12%)</td>
<td>0.31 ± 0.26B</td>
<td>0.35 ± 0.26D</td>
</tr>
</tbody>
</table>

Wall motion index was calculated from the echocardiograms obtained on day 1 and on day 30 of myocardial infarction (or from the last echocardiogram recorded before death in patients who died within 30 days of infarction).

*p < .05 compared with patients with LVT; 2p < .025 compared with patients with LVT; 3p < .01 compared with patients with LVT.

low-up (two patients), while only two (15%) of the 13 who developed thrombi after 48 hr of acute infarction died (p < .005). We therefore decided to further analyze our clinical and echocardiographic findings in these two subgroups of patients (the group with early and that with late development of thrombus). Data from these two patient subgroups were also compared with those obtained in patients who did not develop left ventricular thrombi.

Peak CK and CK-MB values did not differ significantly among the three study groups. Peak total LDH values were significantly higher in patients who developed a thrombus early during myocardial infarction than in patients who did not develop a thrombus, while in patients with late-developing thrombus this variable did not differ significantly from that in the other two groups (table 2). Incidence of Killip classes III or IV and mortality were also significantly higher in patients with early-developing thrombi than in patients without thrombi, while in patients who developed thrombi late, these variables did not differ significantly from those in patients without thrombi (table 2; figures 1 and 2).

Wall motion indexes calculated from the echocardiograms obtained on day 1 and day 30 of infarction (or from the last echocardiogram obtained before death in patients who died within 30 days of infarction) were significantly higher (indicating more extensive wall motion abnormalities) in patients who developed a thrombus early than in patients without thrombus (table 3). In patients with late thrombus formation, wall motion index obtained on day 1 of infarction was not significantly different from that in the other two groups; wall motion index obtained on day 30 was significantly lower than that in patients with early-developing thrombi, but higher than that in patients without thrombi.

In addition, wall motion indexes obtained at these two times during the course of infarction were compared within each group of patients. In patients who developed an early thrombus, left ventricular wall motion was significantly more compromised at the time of its second assessment than at the first assessment, while in patients who developed a thrombus late and in patients without thrombi, wall motion indexes did not differ significantly from those at the first and second assessments (table 3).

We also analyzed the relationship between patient clinical condition (Killip class) at the time when a thrombus was first detected and subsequent patient clinical course. Of the 11 patients who developed

TABLE 2
Clinical data on patients with left ventricular thrombus (LVT) within 48 hr of acute myocardial infarction (AMI), with LVT after 48 hr of AMI, and without LVT

<table>
<thead>
<tr>
<th></th>
<th>Age (years)</th>
<th>Sex (No. of men)</th>
<th>Killip class III or IV (No. of patients)</th>
<th>CK (IU/l)</th>
<th>MB (IU/l)</th>
<th>LDH (IU/l)</th>
<th>Mortality (No. of patients)</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVT within 48 hr of AMI (n = 11)</td>
<td>66 ± 8</td>
<td>9 (82%)</td>
<td>7 (64%)</td>
<td>2448 ± 920</td>
<td>281 ± 114</td>
<td>2087 ± 667</td>
<td>10 (91%)</td>
</tr>
<tr>
<td>LVT after 48 hr of AMI (n = 13)</td>
<td>59 ± 13</td>
<td>12 (92%)</td>
<td>3 (23%)</td>
<td>2368 ± 772</td>
<td>268 ± 64</td>
<td>1695 ± 555</td>
<td>2 (15%)</td>
</tr>
<tr>
<td>No LVT (n = 34)</td>
<td>64 ± 13</td>
<td>23 (68%)</td>
<td>4 (12%)</td>
<td>1935 ± 886</td>
<td>232 ± 146</td>
<td>1440 ± 582B</td>
<td>4 (12%)</td>
</tr>
</tbody>
</table>

*p < .005 compared with patients with LVT within 48 hr of AMI; 2p < .001 compared with patients with LVT within 48 hr of AMI.
FIGURE 1. Patients who developed left ventricular thrombus (LVT) within 48 hr of acute myocardial infarction (AMI), those who developed LVT after 48 hr of AMI, and patients without LVT are compared with regard to incidence of Killip class III or IV.

thrombi early after infarction, four were in Killip class I or II at the time of first detection of the thrombus. Three of these patients died suddenly and unexpectedly during hospitalization. Autopsy demonstrated cardiac rupture and extensive myocardial necrosis in each patient. The remaining seven patients with early-developing thrombi were in Killip class III or IV when the thrombus was first identified. These patients died during hospitalization (five patients) or during follow-up (two patients). Each of the 13 patients who developed thrombus late were in Killip class I or II at the time of first detection. Three of these patients progressed to class III or IV during hospitalization, and two of these three patients died (one of cardiogenic shock on day 5, the other of reinfarction 3 months after the initial infarction).

Causes of death. Sixteen study patients died and the causes of death are summarized in table 4. In the group of patients who developed a thrombus within 48 hr of infarction, cardiogenic shock (five patients) and cardiac rupture (three patients) were the most common causes of death. Reinfarction during follow-up was the cause of death in three of the six patients who died among the patients who either developed a thrombus late, or did not develop one at all. The remaining three

FIGURE 2. Patients who developed left ventricular thrombus (LVT) within 48 hr of acute myocardial infarction (AMI), those who developed LVT after 48 hr of AMI, and patients without LVT are compared with regard to mortality.

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TABLE 3
Wall motion index on day 1 and day 30 of myocardial infarction (MI) in patients with early left ventricular thrombus (LVT), in patients with late-developing LVT, and in patients without LVT

<table>
<thead>
<tr>
<th></th>
<th>Wall motion index on day 1 of MI</th>
<th>Wall motion index on day 30 of MI</th>
</tr>
</thead>
<tbody>
<tr>
<td>LVT within 48 hr of MI (n = 11)</td>
<td>0.50±0.21</td>
<td>0.82±0.25</td>
</tr>
<tr>
<td>LVT after 48 hr of MI (n = 13)</td>
<td>0.42±0.25</td>
<td>0.55±0.14</td>
</tr>
<tr>
<td>No LVT (n = 34)</td>
<td>0.31±0.26</td>
<td>0.35±0.26</td>
</tr>
</tbody>
</table>

*In patients who died within 30 days of MI, wall motion index was calculated from the last echocardiogram recorded before death.

*p < .04 compared with patients with early LVT; †p < .001 compared with patients with early LVT; ‡p < .05 compared with patients with late LVT.

TABLE 4
Causes of death in 16 study patients

<table>
<thead>
<tr>
<th></th>
<th>LVT within 48 hr of AMI</th>
<th>LVT after 48 hr of AMI</th>
<th>No LVT</th>
</tr>
</thead>
<tbody>
<tr>
<td>(No. of patients)</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Cardiogenic shock</td>
<td>5</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Reinfarction</td>
<td>1</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
<td>Cardiac rupture</td>
<td>3</td>
<td>0</td>
<td></td>
</tr>
<tr>
<td>Unexplained sudden death*</td>
<td>1</td>
<td>0</td>
<td>0</td>
</tr>
</tbody>
</table>

AMI = acute myocardial infarction; LVT = ventricular thrombus.

*This patient had recurrent nonsustained ventricular tachycardia, was treated with amiodarone, and died during sleep at home 3 months after myocardial infarction.

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patients in this group died of cardiogenic shock (two died within 24 hr of acute infarction and the third died on day 5).

**Autopsy.** Autopsies were performed in 13 of the 16 study patients who died. In 12 of the 13 patients, autopsy findings confirmed the echocardiographic interpretation (positive for left ventricular thrombus in nine patients and negative in three). In one patient, autopsy identified a thrombus that had not been detected by echocardiography.

In each patient, the location of the thrombus within the left ventricular cavity as verified at autopsy was in agreement with that predicted by echocardiography.

**Spontaneous regression of left ventricular thrombi.** Spontaneous regression of thrombus could be demonstrated in three (20%) of the 15 patients who survived the acute phase of myocardial infarction (figure 3). In these three patients, the regression was documented on the echocardiograms obtained on days 12, 26, and 257 after the thrombus was first identified. The thrombus was located at the left ventricular apex in the first two patients, and extended from the apex over the interventricular septum in the third patient (figure 3, B). The left ventricular wall adjacent to the thrombus was akinetic or dyskinetic in each of the three patients and remained so after the thrombus resolved. Of note is the fact that, in the patient in whom regression of thrombus was documented 257 days after its first detection, a large left ventricular aneurysm developed early after infarction, but it did not prevent the subsequent spontaneous regression of thrombus (figure 3, C).

**Systemic embolic events.** Clinically apparent systemic embolic events occurred in only one patient with a left ventricular thrombus who had two transient ischemic attacks on days 9 and 11 after infarction. No changes in size of thrombus were detected on the echocardiograms obtained during the days after transient ischemic attacks. The patient died of reinfarction 3 months after his first infarction. No systemic embolic events occurred in the group of patients without thrombus. Therefore, the total incidence of embolic events in our study population was 2%.

**Discussion**

**Incidence and prognostic significance of detection of thrombi.** Previous two-dimensional echocardiographic studies have shown that the incidence of left ventricular thrombus is high in patients with acute anterior myocardial infarction (30% to 43% of the patients). In the present investigation, thrombi were detected in about 40% of the study patients. Wall motion abnormalities were more extensive, and peak total

LDH, incidence of Killip class III or IV, and mortality were significantly higher in patients who developed compared with patients who did not develop thrombi. However, the high number of serial echocardiograms obtained in our study patients early during infarction has allowed the identification of a relationship between time of development of thrombus and clinical outcome. More than 90% of the patients who developed thrombi within 2 days of acute anterior infarction died during hospitalization or follow-up. Cardiogenic shock and cardiac rupture, both indicative of extensive myocardial necrosis, were the most common causes of death in these patients. On the other hand, only 15% of the patients who developed thrombi more

![FIGURE 3. Stop-frame images of two-dimensional echocardiograms obtained at end-diastole in the same patient during hospitalization (A) and during follow-up (B and C). Each stop frame is accompanied by a schematic illustration. A, Sixteen days after myocardial infarction, a relatively small, lobulated thrombus is visualized at the left ventricular apex. B, Thirty days after infarction, the thrombus is greatly increased in size, and it now extends from the apex over the interventricular septum. C, Nine months (257 days) after infarction, no thrombus is visualized, and a large left ventricular aneurysm is present. LA = left atrium; LV = left ventricle; VS = interventricular septum; RA = right atrium; RV = right ventricle; T = thrombus.](image-url)
than 2 days after acute infarction died, and their mortality rate was not different from that of patients who did not develop thrombi. Furthermore, the analysis of clinical variables and left ventricular contractility in these patient subgroups substantiated the conclusion that the time of development of thrombus after acute myocardial infarction identifies subsets of patients with different clinical profiles and outcome. For example, in the group of patients who developed a thrombus early, incidence of Killip class III or IV, peak total LDH, and extent of wall motion abnormalities were significantly higher compared with those in patients who did not develop a thrombus. On the other hand, patients who developed a thrombus late did not differ significantly from patients without thrombi with regard to Killip class, enzyme values, and left ventricular wall motion (assessed in the acute phase of infarction). These findings demonstrate that development of left ventricular thrombus within 48 hr of myocardial infarction occurs in patients with the most extensive infarcts and is associated with particularly high mortality.

Although the majority of patients with acute myocardial infarction and large infarcts usually present with or progress to Killip class III or IV, about 15% of patients with extensive myocardial damage and high pulmonary pressures are not identified on the basis of clinical evaluation, but only by means of invasive hemodynamic measurements. Four of our study patients who developed left ventricular thrombus within 48 hr of myocardial infarction had been assigned by clinical criteria to Killip class I or II at the time of admission. Of note is the fact that three of these four patients died suddenly and unexpectedly during hospitalization. Autopsy demonstrated cardiac rupture and extensive myocardial necrosis in each of these three patients. This finding would suggest that echocardiographic detection of early formation of thrombus during acute anterior infarction may identify a subset of patients whose poor prognosis would not be predicted on the basis of their clinical presentation.

Spontaneous regression of thrombus. Spontaneous regression of left ventricular thrombus in patients with myocardial infarction who are not treated with anticoagulants has been previously reported in three patients. In our study, thrombus resolved in three of the 15 patients (20%) who survived the acute phase of infarction. The period of time between formation of the thrombus and its spontaneous regression was greatly variable in these three patients, ranging from a minimum of 12 days to a maximum of 9 months. The left ventricular segments contiguous to the thrombus were akinetic or dyskinetic in each patient and remained so after the thrombus resolved. Regression of thrombus was not associated with clinically evident embolic events in any of the three patients. During follow-up, one of these patients developed a large left ventricular aneurysm associated with poor global left ventricular contractility and, interestingly, the presence of this large aneurysm did not prevent subsequent spontaneous regression of thrombus (figure 3).

Incidence of systemic embolic events. The incidence of clinically apparent systemic embolic events in patients with myocardial infarction and echocardiographic documentation of left ventricular thrombus is controversial, and the available data are from small numbers of patients, the majority of whom were treated with anticoagulants. Aisinger et al. reported no evidence of systemic embolization during a mean follow-up of 9 months in nine patients with anterior myocardial infarction and left ventricular thrombus, seven of whom were treated with anticoagulants. In a preliminary study, Kothari et al. reported one embolic event in 11 patients who received anticoagulants and one embolic event in eight patients not treated with anticoagulants; no data on follow-up were provided. In contrast, Keating et al. reported embolic events in six of seven patients who were not treated with anticoagulants (mean follow-up 1 month) and no embolic events in nine patients on anticoagulants (mean follow-up 11 months). More recently, Weinreich et al. reported systemic embolization in seven of 18 patients who did not receive anticoagulants and in none of 25 patients treated with anticoagulants (minimum follow-up 6 months).

None of our 58 study patients was treated with anticoagulants or platelet inhibitors during the acute phase of infarction or follow-up. The incidence of clinically apparent systemic embolic events was low, with only one patient with left ventricular thrombus having transient ischemic attacks. No embolic events occurred in the remaining patients with or without thrombus during a mean follow-up of 7 months. Therefore, in our study population the total incidence of clinically apparent systemic embolic events was 2%. While this incidence is in agreement with the 0.6% to 6.4% reported in several large series of patients with myocardial infarction, it contrasts with the substantially higher incidence of systemic embolization identified by Keating et al., and Weinreich et al. in study populations similar to ours. We do not have a definite explanation for these conflicting findings. Our study patients were a consecutive series, and no patient was treated with either anticoagulants or platelet inhibitors; therefore,
our results should not be importantly affected by selection factors. Several investigators have described the vigorous mobility of left ventricular thrombi within the left ventricular cavity as an echocardiographic feature associated with embolization. \(^{21-24}\) None of the thrombi identified in our study patients showed vigorous mobility during the cardiac cycle. This morphologic feature could explain, in part, the relatively low incidence of embolic events in our population. However, larger prospective studies are needed to define the clinical and echocardiographic profile of patients with myocardial infarction and left ventricular thrombus who are potentially at higher risk of systemic embolization and who may benefit from treatment with anticoagulants.

In conclusion, our findings indicate that development of left ventricular thrombus within 2 days of acute anterior myocardial infarction occurs in patients with the most extensive infarcts and is predictive of high mortality. Our study also demonstrates that spontaneous regression of thrombus may occur in patients with myocardial infarction, despite the persistence of severe wall motion abnormalities. Finally, in our study patients with left ventricular thrombi, none of whom were treated with anticoagulants or platelet inhibitors, the incidence of clinically apparent systemic embolic events was low.

References


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