Editorial Comment

Acute Infarction, Left Ventricular Thrombus and Systemic Embolization: An Approach to Management*

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Role of echocardiography and platelet scintigraphy in the diagnosis of ventricular thrombi. It has long been recognized that the left ventricle is an important source of systemic embolization; however, it was not until recently that the direct recognition of left ventricular thrombi was possible. As Visser and his colleagues (1) in this issue of the Journal indicate echocardiography is a valuable tool for the diagnosis of left ventricular thrombi in the setting of acute infarction. It is a noninvasive method and may be performed at the bedside. Initial studies using M-mode echocardiography were disappointing because this technique does not routinely examine the cardiac apex, where the overwhelming majority of left ventricular thrombi occur. Early studies (2–4) using two-dimensional echocardiography, however, demonstrated that it affords a major advantage over the M-mode technique because the cardiac apex may be viewed from a number of transducer positions. Echocardiography identifies a mass lesion and does not provide a direct index of thrombus activity. For this reason, a second technique using indium-111–labeled platelets was developed.

Indium-111 has physical characteristics suitable for imaging. The St. Louis group (6) were the first to demonstrate that when 8-OH quinoline was chelated with indium-111, a lipid-soluble complex is formed that is internalized within the platelet cytoplasm and permanently labels human platelets without altering physiologic function. This technique depends entirely on the active exchange of platelets at the blood thrombus interface and, therefore, not only identifies the thrombus but also reflects its activity (6). Several studies (7–9) have been performed to define the accuracy of both echocardiography and platelet scintigraphy. These have been prospective studies and have used findings at aneurysmectomy or autopsy as reference standards. In one echocardiographic study (8), platelet scintigraphy, because of its high specificity, was used as the reference standard in those cases where tissue confirmation could not be obtained.

Advantages and disadvantages. For echocardiography, sensitivity varies from 92 to 77% and specificity from 94 to 84% (7–9). It is estimated that up to 25% of studies may be technically inadequate (9). Thus, echographic quality provides the major limitation of this technique. Platelet scintigraphy has a specificity that approaches 100% (9,10). In patients with a chronic left ventricular aneurysm, scintigraphy has a lower sensitivity (71%) (9). This finding relates directly to the activity of the thrombus. For thrombi developing during acute infarction, there is indirect evidence that platelet scintigraphy has a higher sensitivity because the incidence of thrombi by scintigraphy is close to that found at autopsy (10). The major disadvantage of platelet scintigraphy is that 90 minutes is required for platelet labeling and the diagnosis is reliable only after 48 to 96 hours after injection of the platelet suspension. This is mainly due to the high background activity derived from the circulating platelets that preclude target to background ratios suitable for earlier thrombus detection. To obviate this difficulty, background subtraction (11) and tomographic imaging techniques (12) have been employed. These, however, are cumbersome, especially when used in acutely ill patients. The use of platelet-specific antibodies and the potential for manipulating the background activity holds the greatest hope for the early recognition of ventricular thrombi and promises broader clinical application of this technique (13,14).

In summary, echocardiography should be the first technique used to identify a left ventricular thrombus in acute infarction. Examination should be performed by day 3 and repeated on about day 10 if the initial study is negative. If the examination is technically inadequate or inconclusive, platelet scintigraphy may then be employed. Since the halflife of the isotope and injected platelets allows imaging for 5 to 7 days, a single injection of the labeled platelet is adequate. Insofar as monitoring efficacy of therapy is concerned, platelet scintigraphy may be used investigatively but does not, as yet, have a proven clinical role.

Clinical relevance of ventricular thrombi. These studies raise important questions concerning the clinical relevance of identifying a left ventricular thrombus. It has become clear that a ventricular thrombus is rare in patients with inferior infarction and in patients with subendocardial infarction; therefore, questions of therapy should be limited to those patients who have sustained an acute transmural anterior myocardial infarction (9,15,17). As Visser et al. (1) state, the relation of left ventricular thrombus formation

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to embolization, and the effect of various forms of anticoagulant therapy can only be truly addressed in a randomized prospective study. A trial of this type should involve both echocardiography and platelet scintigraphy, performed in several centers to achieve an adequate sample size and take into consideration the current enthusiasm for employing thrombolytic therapy during the acute phase of myocardial infarction. Thrombolytic agents may influence the development of ventricular thrombi by a direct effect on the thrombus and also by salvaging myocardium, thereby causing less regional left ventricular dysfunction and, thus, less local stasis. This latter benefit may also be seen with pharmacologic and surgical therapy. Therefore, recommendations concerning therapy must be tempered by the fact that the definitive study in the context of today’s medicine has not been performed. Most authors agree that emboli tend to occur early (18), within the first 6 months, and most trials (19,20) support treatment with anticoagulants when embolism is the end point. Successful treatment of thrombosis may be more difficult to assess because the effect may be seen by scintigraphy and not by echocardiography (21); in addition, thrombi tend to be spontaneously evenescence. Echocardiography may, as Visser et al. have shown, pinpoint protruberant thrombi that are more likely to embolize.

**Recommendations.** Until a prospective randomized study is completed, it is reasonable to conclude that patients with anterior myocardial infarction and a ventricular thrombus (particularly if the thrombus is protruberant), unless important contraindications to anticoagulant therapy exist, should receive intravenous heparin on presentation followed by 6 months of Coumadin therapy. This obviously implies careful and regular monitoring of anticoagulant therapy. One can argue, based on the older literature, that all patients with transmural anterior myocardial infarction in whom a thrombus cannot be reasonably excluded should be anticoagulated. In these patients, the probability of embolism should be carefully weighed against the danger of bleeding. If the probability of a major complication is low, it is not unreasonable to advise anticoagulation therapy.

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**References**


