Editorial Comment

\textbf{111In Platelet Scintigraphy and Risk Stratification for Embolization From Chronic Left Ventricular Thrombi}

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Arterial thromboembolization is a potentially catastrophic complication of acute anterior myocardial infarction, left ventricular aneurysm, and diffuse cardiomyopathy. Management of patients at risk of thromboembolization is directed at determining the potential benefit of anticoagulation in preventing embolization compared with the risk of bleeding complications from anticoagulation. In the absence of prospective, randomized trials evaluating the efficacy of anticoagulation in reducing the rate of embolization, recommendations\(^1\)\(^2\) are largely based on clinical, surgical, and autopsy studies that have demonstrated: 1) an association of anticoagulation with reduced rate of embolization, 2) an association of thrombus prevention or resolution with anticoagulant therapy, or 3) an association of morphologic features according to echocardiography with increased risk of embolization.

In the case of acute anterior myocardial infarction, clinical studies have demonstrated decreased embolization rates in patients receiving anticoagulation from the time of presentation with acute myocardial infarction\(^3\)^4 or after echocardiographic demonstration of thrombus.\(^5\)^6 In addition, echocardiographic studies have shown thrombus reduction or resolution,\(^5\)^7\(^8\) as well as prevention,\(^9\) with anticoagulation. Finally, several studies demonstrate increased embolic risk in patients with mobile, protruding thrombi demonstrated by echocardiography.\(^10\)^\(^13\)

In the case of chronic left ventricular aneurysms, the incidence of a clinical embolic event is only 0.35 per 100 patient-years.\(^14\) Such a low incidence makes it difficult to assess the efficacy of anticoagulant therapy in the prevention of systemic embolization. In patients with chronic left ventricular thrombi, primarily associated with remote myocardial infarction, warfarin therapy is associated with a higher incidence of thrombus resolution compared with no therapy (59\% vs. 29\%, respectively) as assessed by echocardiography.\(^15\) Surgical series have demonstrated no apparent effect of anticoagulation on the prevalence of mural thrombi, identified at the time of aneurysmectomy.\(^16\)^\(^17\) On the other hand, the presence of thrombus has been shown to correlate inversely with duration of therapy.\(^16\) That is, a longer period of anticoagulation appears to be associated with a lower incidence of thrombus. Thrombus protrusion and mobility are related to increased embolic risk\(^15\) as are thrombi associated with recent myocardial infarction. Although some studies have demonstrated that spontaneous changes in thrombus morphology (mobile or protruding vs. sessile) may occur after recent myocardial infarction,\(^18\) these changes occur only rarely when thrombi are associated with remote myocardial infarction or cardiomyopathy.\(^15\)

In the case of diffuse cardiomyopathy, a retrospective study\(^19\) demonstrated embolization in 18\% of patients not on anticoagulants (approximately 4 embolic events per 100 patient-years). However, none of the patients who received anticoagulation had emboli. In addition, the presence of thrombus may not be related to the rate of embolization. Gottdiener et al\(^20\) demonstrated that systemic emboli occurred in 11\% of 96 patients with dilated cardiomyopathy but that emboli were not more frequent in patients with cardiomyopathy who had left ventricular thrombus.

There is general agreement on the need for anticoagulation in patients with acute anterior myocardial infarction and in patients with diffuse cardiomyopathy, especially when thrombus is demonstrated. Anticoagulation in patients with chronic left ventricular thrombi in association with an aneurysm remains controversial. In this issue of Circulation, Stratton and Ritchie\(^21\) provide provocative evidence that indium 111 platelet scintigraphy may be of use in identifying patients with chronic left ventricular thrombi at increased risk of embolization. In their study, embolic rates in 30 patients with positive echocardiograms and platelet scintigrams for thrombus were compared with embolic rates in 28 patients.
having positive echocardiograms but negative scintigrams. The groups were well matched in terms of incidence of prior myocardial infarction (93% vs. 82%) and interval from prior myocardial infarction (24±17 vs. 30±34 months). The two groups also had similar echocardiographic features of thrombus protrusion and mobility. Embolization during the follow-up period occurred in 23% of patients with a positive echocardiogram and platelet scintigram compared with only 4% of patients with a positive echocardiogram and a negative platelet scintigram. Furthermore, the incidence of a positive platelet scintigram was 88% in the eight patients with a positive echocardiogram and embolization compared with 46% in the 50 patients with a positive echocardiogram but no embolization.

Straton and Ritchie show that 111 In platelet scintigraphy has potential as a screening test in patients with suspected left ventricular thrombus related to either prior anterior myocardial infarction or cardiomyopathy. In patients with positive platelet scintigrams, embolization occurred in 21% of patients compared with only 3% of patients with a negative platelet scintigram during a mean follow-up of 38±30 months.

Despite the inherent limitations of a retrospective study, platelet scintigraphy appears to provide "functional" information that is supplementary to the "structural" identification of thrombus by echocardiography. 111 In-labeled platelet scintigraphy has a sensitivity of 71% and a specificity of 100% for detecting left ventricular thrombi, which compares favorably with 77% and 92%, respectively, with echocardiography.22 Also, scintigraphic data acquisition and interpretation appear reproducible.23,24 However, there are technical factors in platelet imaging, which have limited widespread clinical availability. First, the 111 In platelet labeling, when properly performed, does not cause significant degradation of platelet function25; however, there are steps in the procedure that may alter platelet reactivity26 or labeling efficiency.27 Furthermore, the platelet-labeling procedure requires about 90 minutes.28 Second, platelet imaging requires a delay of 3–4 days after platelet injection to achieve optimal ratios of thrombus to background radioactivity for detecting chronic thrombi.24 Third, scintigrams may be difficult to interpret in the presence of an elevated left hemidiaphragm and with increased blood pool activity in ventricular aneurysm.24

In summary, optimal care of patients with chronic left ventricular thrombi includes the identification of those patients who may benefit from chronic anticoagulation. Although the clinical use of platelet scintigraphy should be considered tentative, the increased rate of embolization of chronic left ventricular thrombi with positive platelet scintigraphy should stimulate interest in additional prospective clinical trials of anticoagulation in subsets of patients at high risk for embolization.

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