From Swirling to a Mobile, Pedunculated Mass—The Evolution of Left Ventricular Thrombus Despite Full Anticoagulation*

Echocardiographic Demonstration
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We describe the unusual evolution of a left ventricular thrombus following acute anterior myocardial infarction despite adequate anticoagulation. Serial echocardiographic examinations demonstrated the evolution from swirling in the left ventricle through a solid apical mass gradually dislodging into a mobile, pedunculated mass that was removed surgically to prevent embolization. This report emphasizes the need to follow echocardiographically left ventricular thrombi during treatment with anticoagulants, and to identify morphologic changes that may predict embolization. This case suggests that left ventricular thrombectomy should be considered in selected patients in whom a very high-risk thrombus morphology is detected.

(Chest 1993; 103:281-83)

Left ventricular thrombus formation is a well-known complication of anterior myocardial infarction (MI), which carries a variable risk of systemic embolization.1,2 This risk may become substantial if protrusion into the lumen of the ventricle, mobility, or pedunculated appearance are detected by two-dimensional echocardiography.3,4 Although there may be a role for anticoagulants in reducing thrombus formation and embolic complications during acute MI,5 very little is known about the morphologic evolution of established thrombi under anticoagulant treatment.

We present sequential echocardiographic studies of left ventricular thrombus evolution under full anticoagulation, from swirling of blood in the left ventricle through the development of a solid mural thrombus, followed by gradual dislocation of the thrombus, resulting in the formation of a large, mobile, pedunculated mass that was then removed surgically.

CASE REPORT

A 62-year-old man was admitted to the hospital with a clinical and electrocardiographic diagnosis of extensive anterior MI. He had had an antero septal MI two years prior to his present hospital admission that was successfully treated at that time with streptokinase. He had been asymptomatic until his present hospital admission. Noninvasive studies revealed mild impairment of left ventricular function.

Four hours following the onset of chest pain, the patient was admitted to the coronary care unit, and treatment with intravenous streptokinase was started, followed by clinical, electrocardiographic, and biochemical signs of reperfusion. Treatment with heparin, routinely administered following thrombolytic treatment, had to be stopped due to upper gastrointestinal bleeding on the second day of hospitalization. Despite clinical evidence for reperfusion, echocardiographic study carried out on the second day revealed a large anteroapical akinetic segment with markedly reduced left ventricular function.

A second echocardiographic study performed on the fourth hospital day revealed again a large akinetic anterior segment. Marked swirling of blood within the apical part of the ventricle ("spontaneous contrast") was present. Three days later, a 4 x 4 cm solid mural thrombus in the left ventricular apex was observed (Fig 1a). Heparin therapy was started again, and the partial thromboplastin time was meticulously kept at the therapeutic range from this time on. One day later, warfarin treatment was initiated as well. Echocardiogram performed on the eighth hospital day demonstrated the transition of the thrombus into a more rounded,

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FIGURE 1. Evolution of left ventricular thrombus during hospitalization. First demonstrated on the seventh hospital day, it appeared as a 4 x 4 cm apical mural thrombus (1a, top) that later developed on the eighth hospital day into a mobile rounded protruding mass (1b, center) and later (on the 11th hospital day) into a freely moving pedunculated globular mass (1c, bottom).
partially mobile mass protruding into the left ventricular lumen (Fig 1b). Three days later, while still receiving anticoagulation, the thrombus had turned into a 3 x 3.5-cm pedunculated globular mass connected to the endocardium by a very narrow stalk, moving freely in a "wavy" motion within the left ventricular lumen (Fig 1c).

Since the risk for embolization in the presence of a thrombus with such a configuration had been reported to approach 60 to 80 percent, the patient was referred to surgery on his 11th postinfarction day. The operative procedure was performed as described previously. A 3 x 4-cm thrombus was successfully removed. The postoperative course was uneventful and the patient was discharged from the hospital on the tenth postoperative day.

DISCUSSION

Left ventricular thrombus is a common complication of anterior MI and may occur in up to 40 percent of these cases. An early postinfarction thrombus carries a significant risk of systemic embolization, which is about 20 percent of all diagnosed thrombi. The higher tendency of fresh thrombi to embolize is related to their increased friability and protrusion into the left ventricular lumen, as opposed to well-organized, laminated thrombi formed within aneurysms. Certain morphologic features, characterized by twodimensional echocardiography, may predict a higher likelihood for embolization. Such features include protrusion into the left ventricle, mobility, and pedunculated appearance. It should be noted, however, that the information concerning the association between morphology and embolization is derived mainly from retrospective analysis of cases with established embolism. Anticoagulant treatment has an established role in reducing the rate of thrombus formation and embolic phenomena during acute MI. However, the role of anticoagulant treatment initiated after an established thrombus has already been detected is yet unclear.

Little information is available concerning the effect of anticoagulants on the morphologic features of left ventricular thrombus. Although reduction in thrombus size, as well as transformation from a protruding into a mural thrombus have been documented, the opposite may also happen. Spontaneous changes in the morphologic features of left ventricular thrombi without any anticoagulation have been documented previously as well.

Our case clearly documented that anticoagulant treatment did not prevent the malignant change in thrombus configuration. One may even argue that it might have even contributed to the change by enhancing loss of thrombus tissue in the area of connection to the endocardium. Whether this is the underlying mechanism of embolization described following the initiation of anticoagulant therapy or thrombolytic agents remains to be established. This observation is consistent with a recently published series that suggested that in patients who had received thrombolytic therapy, anticoagulation with heparin was associated with a higher incidence of initial mural morphology as compared with protruding morphologic features and a higher tendency of the former to change later into protruding morphologic features during treatment with heparin.

It seems advisable therefore to closely follow thrombus morphology by repeated echocardiographic studies when anticoagulants are being administered. In selected cases, in which "malignant" change in morphologic features occurs, surgical thrombectomy should be considered. This approach is based on others' as well as on our favorable surgical results in these cases. However, due to the small number of patients previously treated with this approach, as well as the lack of prospective studies on the risk of embolism as a function of thrombus morphology, this approach needs further confirmation.

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Fungal endocarditis is a rare complication of permanent pacemaker implantation. In all reports we have identified, this infection has been fatal, diagnosed postmortem. We present a patient in whom early echocardiographic diagnosis resulted in curative surgical and antimicrobial therapy. Fungal endocarditis is an unusual, but treatable complication of permanent pacemakers. (Chest 1993; 103:283-84)

Endocarditis is an uncommon complication of pacemaker placement. This problem is reported in only 1 percent to 7 percent of patients, and is usually caused by staphylococcal infection. Pacemaker endocarditis due to fungi is distinctively rare. We have identified reports of only four such cases, and all were diagnosed postmortem. We present a patient in whom fungal pacemaker endocarditis was identified ante mortem. Early diagnosis was facilitated by echocardiography and led to curative surgical and medical treatment.

CASE REPORT

A 56-year-old man was referred with fever, nonproductive cough, dyspnea, and pleurisy of five weeks’ duration. He had undergone uncomplicated placement of a dual chamber (DDD) pacemaker five years earlier for second degree Mobitz II heart block, but otherwise was generally well. An initial chest roentgenogram was clear and he was given a course of erythromycin. He returned with persistent symptoms and wheezing and was placed on a regimen of cefaclor and a tapering dose of prednisone. Ten days prior to referral, he developed worsening fever, chills, and cough. Laboratory findings included a white blood cell count of 13,500/cu mm, PaO₂ of 66 mm Hg, and an unremarkable chest roentgenogram. The patient was hospitalized and treated with empiric broad-spectrum antibiotics. A 99mTc MAA perfusion lung scan showed absent perfusion of the left lung, suggesting massive pulmonary embolism, and he was heparinized. Follow-up chest roentgenogram showed left lower lobe consolidation.

On transfer to our medical center, the patient had a low-grade fever with diminished breath sounds and left basilar crackles. Laboratory studies showed a white blood cell count of 16,000/cu mm with no left shift. Chest roentgenogram demonstrated infiltrates in the left upper and lower lobes. Heparin therapy was continued, but a lower extremity Doppler failed to demonstrate deep vein thrombosis. Pulmonary arteriography was performed (Fig 1). This study demonstrated subtotal occlusion at the origin of the left pulmonary artery, suggesting an obstructive mass rather than pulmonary embolism. Fiberoptic bronchoscopy showed no endobronchial lesion.

The patient’s condition deteriorated with worsening hypoxemia. A two-dimensional echocardiogram revealed multiple large right atrial masses prolapsing into the right ventricle and with possible adherence to the right atrial wall and pacemaker wire. Blood cultures from the referring hospital were then reported to be growing Candida albicans, and amphotericin B therapy was initiated. The following day the patient underwent right atriotomy and pulmonary arteriotomy with removal of the pacemaker leads. Intraoperative transesophageal echocardiography (Fig 2) confirmed operative findings, and surgery demonstrated several friable soft-tissue masses encasing the atrial pacer wire (Fig 3). Although the tricuspid valve appeared uninvolved, a large fungus ball

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Candida Endocarditis*
A Treatable Form of Pacemaker Infection


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FIGURE 1. Pulmonary angiogram revealing complete occlusion of the left pulmonary artery with normal right pulmonary artery.

FIGURE 2. Intraoperative transesophageal echocardiogram showing right atrial masses prolapsing into the right ventricle. RA = right atrium; RV = right ventricle; M = mass.

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