Differential Diagnosis of Left Ventricular Mural Thrombi by Myocardial Contrast Echocardiography
—— A Preliminary Study ——

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Two-dimensional echocardiography has become the procedure of choice to diagnose left ventricular mural thrombi. However, small or flat thrombi may be difficult to distinguish from myocardium. The authors presumed that the absence of arterial supply to a fresh thrombus may allow MCE to distinguish between thrombus and myocardium. In the 2 cases presented here, MCE was performed with the same technique as that used for the purpose of visualization of myocardial perfusion; as a result, an apical mural thrombus, indistinct from myocardium before MCE, was visualized as a contrast defect during imaging. Conversely, myocardium that mimicked a thrombus was imaged by MCE as a contrast-opacified area. These findings suggest that MCE after reperfusion therapy is useful to distinguish mural thrombi from myocardium. (Jpn Circ J 1999; 63: 50–52)

Key Words: Echocardiography; Mural thrombus; Myocardial contrast; Myocardial infarction

The spatial distribution of the ventricular myocardial blood flow can be imaged with myocardial contrast echocardiography (MCE). In patients with reperfused acute myocardial infarction (AMI), MCE is a promising method to estimate the extent of myocardial microvascular damage.1–3 The technique is particularly accurate in the detection of microvascular damage associated with intramyocardial hemorrhages approximately 1 month after reperfusion therapy.3

A common complication of large myocardial infarctions is the development of left ventricular mural thrombi. These thrombi are usually adjacent to a dyskinetic or akinetic area, which is often aneurysmal. Two-dimensional (2-D) echocardiography has become the procedure of choice to diagnose mural thrombi.5 However, small or flat thrombi may be difficult to distinguish from myocardium. We presumed that the absence of arterial supply to a fresh thrombus may allow the distinction between thrombus and myocardium when using MCE. The following 2 cases illustrate the contribution of MCE after reperfusion therapy in the differential diagnosis of left ventricular mural thrombi.

Case Reports

Patient 1
A 68-year-old man was admitted to hospital because of severe anterior chest pain due to an anterior AMI. Coronary angiography revealed the presence of a complete occlusion of the proximal left anterior descending artery. TIMI grade 3 flow was reestablished by primary percutaneous transluminal coronary angioplasty. The time from onset of infarction to myocardial reperfusion was 5 h and serum creatinine kinase (CK) peaked at 4,480 IU/L. Despite successful reperfusion therapy and no further ischemic events, 2-D echocardiography in the apical view 1 month after reperfusion showed akinesis of the anterior wall and apex. An increase in the apical wall thickness raised the suspicion of an apical mural thrombus, although the lack of typical findings precluded its definitive diagnosis (Fig 1A). Coronary angiography was repeated, confirming the patency of the infarct-related artery. The left ventriculogram showed no obvious filling defect within the akinetic area. After the completion of follow-up coronary angiography, MCE was performed with a commercially available phased-array system (EUB-555, Hitachi) and a 3.5-MHz fundamental transducer. Images were recorded on 1.25-cm videotape with an S-VHS, AG-7355 Panasonic recorder. Two milliliters of sonicated albumin containing microbubbles was injected into each coronary artery. MCE showed decreased, but distinct, contrast opacification of the akinetic apical myocardium, and a contrast defect in the area of suspected mural thrombus (Fig 1B). Treatment with warfarin potassium was begun. However, the patient was noncompliant and, 2 month later, 2-D echocardiography revealed the presence of a thrombus protruding into the left ventricular cavity (Fig 2).

Patient 2
A 59-year-old woman was admitted to hospital because of chest pain. Coronary angiography revealed complete occlusion of the proximal left anterior descending artery. Primary coronary angioplasty was performed successfully and TIMI grade 3 flow reestablished. The time from onset of infarction to reperfusion was 11 h and serum CK peaked...
at 3,266 IU/L. Two-dimensional echocardiography 1 month after reperfusion showed nearly identical images as in patient 1. However, MCE revealed the presence of a distinct contrast opacification of the area where a mural thrombus has been suspected (Fig 3). The patient was discharged from the hospital without anticoagulation therapy and subsequent follow-up 2-D echocardiograms have remained unchanged.
Discussion

Although 2-D echocardiography is capable of imaging left ventricular mural thrombi, the technique has limitations. Whereas large, bright, mobile thrombi are relatively easy to identify, sessile mural thrombi may be difficult to diagnose echocardiographically. The clot may be subtle and must be differentiated from nonthrombogenic echoes. We have previously reported that, in patients with reperfused AMI, MCE with intracoronary injection of sonicated albumin during the chronic phase of myocardial infarction allows the accurate detection of post-reperfusion microvascular damage associated with intramyocardial hemorrhage. In the cases presented here, MCE was performed with the same technique as that used for the purpose of visualization of myocardial perfusion; as a result, an apical mural thrombus, indistinct from myocardium before MCE, was visualized as a contrast defect during imaging. Conversely, myocardium that mimicked a thrombus was imaged as a contrast-opacified area by MCE. These findings suggest that MCE after reperfusion therapy is useful to distinguish mural thrombi from myocardium, when the all following conditions are met: (1) intracoronary injection of contrast agent after successful reperfusion; (2) optimal imaging and enhancement in the infarct area; and (3) early formation of thrombi after the onset. The early detection of mural thrombi is helpful with respect to the timely initiation of chronic anticoagulation and prevention of thromboembolic events. On the other hand, unnecessary anticoagulation can be avoided in patients whose myocardial images mimic thrombi.

Left heart opacification by the intravenous injection of air-containing microbubbles improves the delineation of left ventricular endocardial borders. Such contrast, however, is inadequate to visualize myocardial perfusion. Therefore, in this study, MCE was performed during intracoronary injection of sonicated albumin. Further studies will be needed to determine whether new contrast agents injected intravenously, such as perfluorocarbon containing microbubbles, are equally capable of detecting the presence of mural thrombi.

It has been reported that coronary arteriography in the presence of mural thrombi may show neovascularity to the region of the thrombi, contrast blush within the thrombi, and fistulous communication with the cardiac chamber in which the thrombi adheres. These angiographic abnormalities most likely represent an intermediate stage in the evolution of the thrombi. In such cases, it may be difficult to differentiate thrombi from myocardium by MCE.

The distinction of mural thrombi from subendocardial infarction is diagnostically important. In the presence of a mural thrombus, an increase in the thickness of the akinetic wall is observed, as illustrated in Fig 1. In contrast, the thickness of the akinetic wall is usually not increased in the case of subendocardial infarction. In cases of failure of reperfusion, or the ‘no reflow’ phenomenon, clots cannot be distinguished from myocardium. Previous studies have found that patients with ‘no reflow’ on MCE have poor left ventricular functional recovery and a progressive increase in left ventricular end-diastolic volume in the convalescent phase of myocardial infarction. In these cases, chronic anticoagulation therapy seems desirable if a thrombus is suspected. It has been recently reported that a thrombus-targeting contrast agent can improve the visualization of left atrial appendage thrombi in dogs. If such an agent became clinically available, it could be used to detect the presence of mural thrombi in cases of ‘no reflow’.

References