Case Report

Benign Disappearance of Ventricular Thrombus After Embolic Stroke

A Case Report

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The source for a patient's middle cerebral artery territory embolic stroke was found by echocardiography to be a left ventricular cardiac thrombus. The apical mass was large, pedunculated, and moved with systole into the ventricular cavity. The absence of ventricular dyskinesia was thought to favor a tumor, and surgery was considered before repeat echocardiography showed disappearance of the mass, making thrombus the likely diagnosis. No further embolic events occurred during or since the disappearance of the thrombus while on anticoagulation therapy. Serial echocardiography for change in or disappearance of a ventricular mass may be critical in distinguishing thrombus from tumor. (Stroke 1988;19:393-396)

Identification of an inobvious cardiac embolic source is often difficult in patients with otherwise typical clinical evidence of cerebral embolism. A recognized subgroup of stroke, so-called "unknown source embolism," implies that extensive cardiac and cerebrovascular investigations have been negative in providing a cause for stroke that occurs suddenly and without warning in a recognized vascular distribution, such as the middle cerebral artery (MCA) territory, susceptible to embolism. The yield has been low in identifying a potential cardiac embolic condition in such patients despite monitoring for arrhythmia, efforts to detect silent myocardial infarction or valvular disease, or other echocardiographic abnormalities. Rarer still is the finding of a ventricular thrombus by echocardiography as the source for MCA embolism.

The present case is unusual in that a large, left ventricular mass without detectable ventricular wall motion abnormalities was initially thought to represent tumor, but its disappearance during anticoagulation treatment, and without any further clinical embolic events, made the diagnosis of thrombus probable.

Case Report

An 80-year-old, previously healthy, right-handed woman was walking down the hallway on her way out to shop and suddenly, without warning, fell to the floor unable to get up. She had no history of transient monocular blindness, transient hemispheral attacks, hypertension, or cardiac disease.

On initial examination she had a blood pressure of 130/70 mm Hg and a regular heart rate of 90 beats/min; cardiac examination was normal. There were no carotid, subclavian, or ocular bruits. She was aware that her right upper extremity was weak and that her speech was not normal. Her speech was garbled, and she had right lower facial weakness and a flaccid right arm. Within an hour, however, she had improved. Her speech was mildly abnormal with occasional paraphasic errors and impaired comprehension for complicated verbal commands; she was able to lift her right arm against gravity and open and close her right fist slowly. Walking was normal.

Computed tomography (CT scan) without contrast was normal on admission. CT scan 4 days later showed a low-density area in the left temporal parietal region consistent with infarction. Electrocardiography was normal except for nonspecific ST-T wave changes. Serial cardiac enzyme studies were all normal, ruling out a recent myocardial infarction.

Two-dimensional echocardiography (2DE) performed on the fourth day demonstrated a 2 x 2.5 cm pedunculated filling defect in the inferior apex of the left ventricle (Figure 1). The mass moved with heart contraction. There were echolucent cystic areas within the mass. No ventricular wall motion abnormalities, either regional or global, were noted. The cardiologic impression was that the mass probably represented tumor rather than thrombus, and surgery was considered. It was believed that the most likely tumor in this location would be metastatic, although screening studies revealed no evidence of malignancy outside the heart. Meanwhile, however, the patient was anticoagulated with heparin and continued to improve in
speech and right arm-hand functions. Twelve days after 2DE cardiac isotope angiography revealed no intracardiac masses, but a small area of left ventricular wall hypokinesis was noted. 2DE was then repeated, and the intraventricular mass had disappeared (Figure 2). No new symptoms occurred during this time. She has remained asymptomatic on warfarin anticoagulation for 2 years.

Discussion

The present case is instructive for several reasons: 1) a cardiac embolic cause was discovered for a presumed embolic stroke in which the initial presentation provided no clue of cardiac disease, 2) the left ventricular mass was misinterpreted as tumor on initial echocardiography because no associated ventricular wall dyskinesia was apparent until the mass had disappeared, and 3) the disappearance of the left ventricular thrombus (LVT) during anticoagulation was not associated with further embolism.

In stroke patients without preexisting heart disease, the indications for further cardiac evaluation are unclear*5 and the yield of finding a lesion of embolic potential by 2DE in some series has been low./6-' In three separate reports totalling 132 patients, none had a detectable cardiac source for emboli except for one patient found to have mitral valve prolapse.7'9 Even in patients with existing heart disease in these series, the yield with 2DE was low.6'8 Others have found a much higher yield of unsuspected cardiac-source lesions by 2DE.1 2DE has a sensitivity of 77-95%10-12 and a specificity of 86--88% in detecting LVT. The absence of LVT by 2DE does not eliminate a cardiac source for cerebral emboli since the material may be too small to detect or may have already dislodged from the heart with no trace. In the absence of detectable LVT, left ventricular wall motion abnormalities may be sought as a marker for spent thrombus. Radionuclide angiography is a simple, noninvasive means of detecting LVT13,14 and may be another means of identifying areas of hypokinesis. The apparent absence of a hypokinetic ventricular segment in our patient initially led to the erroneous conclusion that the ventricular mass was probably tumor and not thrombus since LVT is regularly associated with wall motion abnormalities.12 In our patient the disappearance of the ventricular mass allowed visualization of the previously obscured hypokinetic area and suggests that serial echocardiographic evaluations may be critical in the interpretation of left ventricular masses.

The reported incidence of symptomatic embolic events in patients with LVT detected by 2DE varies from 7% to 39%.15-17 Thrombus configuration and attachment may influence embolic propensity; flat thrombus within an aneurysm is less likely to embolize than thrombus protruding into the ventricular cavity or with free intracavitary motion.17 Haugland et al17 found the pretest likelihood of embolization given the presence of a left ventricular thrombus to be 27%; the presence of intracavitary motion increased the posttest likelihood of embolization to 60%. Nineteen percent were recurrent. Our patient had movement of the ventricular mass with heart contraction but showed no evidence of recurrent embolism.

Our patient was anticoagulated (initially heparin, then warfarin) during the time that the thrombus resolved and no further embolic events occurred. Whether thrombus resolution was effected by the body's own thrombolytic mechanisms or anticoagulation therapy cannot be settled by this single case. The
Disappearance of a ventricular mass detected by 2DE or angiocardiography has been documented on rare occasions. In one report, anticoagulation therapy was begun after LVT was detected by 2DE after anterior myocardial infarction, but 3 days later the patient suffered a MCA-distribution stroke. Repeat 2DE showed disappearance of the original LVT. Weinreich et al studied 261 patients with 2DE after transmural myocardial infarction. Eighteen percent were found to have mural thrombi; 34% had anterior myocardial infarctions, and 1.5% had inferior wall myocardial infarctions. An apical wall abnormality was noted in all patients with thrombus. Twenty-five of 43 patients followed with serial 2DE were treated with anticoagulation, and none had embolic events. Seven patients of the remaining 18 not receiving anticoagulation therapy suffered an embolic event within 4 months. These authors also noted that three of 43 patients with LVT had freely mobile thrombi attached to the endocardium by a thin stalk; two of these patients on anticoagulation had no embolic event, with resolution of LVT on follow-up 2DE. The third patient, not on anticoagulation, had a cerebral embolic event and continued visualization of the peduncular thrombus on repeat 2DE. Autopsy confirmed intracardiac thrombus and intracerebral embolism.

Based on our experience and those reported above, if a ventricular mass is discovered on echocardiography, further follow-up testing with echocardiography might be appropriate. In addition, anticoagulation therapy may be reasonable in patients with cerebral embolism and an echocardiography-demonstrated ventricular mass suggesting thrombus or tumor, at least until more extensive, controlled scientific studies bear on the issue.

References
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