CASE REPORTS

Two-dimensional Echocardiographic Demonstration of Left Atrial Thrombi in Patients with Prosthetic Mitral Valves

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SUMMARY Although M-mode echocardiography (MME) is not a reliable method for detecting left atrial thrombi, recent reports suggest that two-dimensional echo (2DE) may be more effective than MME in identifying intracardiac thrombi.

In three patients with prosthetic mitral valves who presented with either arterial embolization or prosthetic valvular dysfunction, 2DE demonstrated left atrial masses consistent with thrombi, while MME was either negative (two patients) or suspicious (one patient) for left atrial thrombus. Thrombi were documented by surgical or postmortem examination in all cases. Clear delineation of the atrial cavity and the margins of the masses, visualization on multiple echocardiographic views and comparison of serial examinations were helpful in identifying these masses as thrombi. In addition, the masses visualized had certain patterns of motion which seem unique and may allow characterization of atrial masses as thrombi.

LEFT ATRIAL THROMBUS formation in patients after prosthetic mitral valve placement may lead to systemic embolization, prosthetic malfunction or mitral orifice occlusion with severe hemodynamic consequences. The diagnosis of left atrial thrombus is difficult, however, and contrast opacification of the atrium using levophase pulmonary or transseptal atrial angiography is usually required for non-operative diagnosis.\(^1,2\) The latter approach is associated with significant risk to the patient.\(^3\) While M-mode echocardiography has proved to be a reliable, noninvasive method for identifying certain intra-atrial masses such as myxomas or vegetations in patients with bacterial endocarditis,\(^4,5\) intra-atrial thrombi have been difficult to image using this technique.\(^6\) Even in highly suspicious cases the distinction between clot and normal left atrial posterior wall echoes may be difficult.\(^7\) Two-dimensional echocardiography has been successfully used to identify thrombi in the left ventricle\(^8,11\) and in the left atrium of patients with mitral stenosis.\(^10\) These reports indicate that two-dimensional echocardiography may be more efficacious in identifying intracardiac thrombus formation than standard M-mode echocardiography.\(^5,11\)

We have recently used two-dimensional echocardiography to demonstrate left atrial thrombi in three patients with mitral valve replacement who presented clinically with evidence of peripheral embolization or prosthetic valve dysfunction.\(^11\) In all three patients atrial thrombus formation was considered as a possible cause of the clinical presentation before the two-dimensional study. M-mode echocardiograms were either not helpful or nonspecific. In each case cineangiographic and surgical or postmortem examinations confirmed the presence of atrial thrombus.

Two-dimensional echograms were obtained using a Varian V-3000 ultrasonicoscope. Multiple acoustical windows with various sector orientations were used in a systematic approach\(^11\) to provide complete imaging of the left atrial and mitral valve anatomy. The anterior precordial or parasternal, apical and subxyphoid windows were used to obtain the images. In the anterior precordial window the sector plane was initially oriented parallel to the long axis of the left ventricle to obtain a long-axis view and subsequently rotated 90° to obtain the anterior precordial short-axis view. In the apical window the transducer was placed at the point of maximal impulse with the sector plane perpendicular to the interventricular septum to obtain a four-chamber view. The transducer was then rotated approximately 90° for the apical two-chamber view. From the subxyphoid window the sector plane was positioned to obtain a long-axis view of the heart and then rotated 90° for the subxyphoid short-axis view.

Case Reports

Case I

A 52-year-old man with dyspnea and orthopnea was admitted. He had a history of acute rheumatic fever.
Dyspnea on exertion and atrial fibrillation had developed 9 years before admission and 1 month before admission he developed acute pulmonary edema. Physical examination revealed mitral stenosis and insufficiency. An M-mode echocardiogram confirmed the diagnosis of mitral stenosis. The left atrial dimension was 62 mm. At cardiac catheterization moderately severe mitral stenosis and moderate mitral insufficiency were demonstrated, and the patient underwent mitral valve replacement with a #29 Edwards porcine heterograft. At surgery there was no evidence of left atrial thrombus. The postoperative course was uncomplicated until the third day, when he developed transient right hemiparesis. Anticoagulation with coumadin was initiated. He had no further complications and was discharged 16 days postoperatively. Three days later he was readmitted after he developed sudden onset of orthopnea and paroxysmal nocturnal dyspnea. He had no chills, fever, constitutional symptoms or chest pain.

On physical examination he had a blood pressure of 140/80 mm Hg, an irregular pulse at 120 beats/min and tachypnea. The lungs had bibasilar rales. There was elevated jugular venous pressure and a positive hepatojugular reflux. A grade II/VI mid-diastolic rumble and a grade II/VI systolic ejection murmur were heard at the apex. Heart sounds were normal in quality and intensity with a prominent diastolic sound present at the apex. Neurologic examination was normal and there were no mucocutaneous signs of systemic emboli. An ECG demonstrated atrial fibrillation and was unchanged from preoperative tracings. Mild cardiomegaly with engorgement of the pulmonary veins and bilateral Kerley B lines and pleural effusions were seen on chest x-ray.

M-mode echocardiography was performed. The left atrial dimension was 65 mm, and there was a layer of echoes noted above the posterior atrial wall (fig. 1). This pattern was not present on the preoperative M-mode echo and, although considered a nonspecific finding, was suggestive of left atrial thrombus. Two-dimensional echocardiography demonstrated a large, slightly mobile mass in the left atrium (figs. 2 and 3). On viewing this mass from different acoustical win-
dows, including the anterior precordial long- and short-axis, apical two- and four-chamber, and subxyphoid long-axis views, it appeared contiguous with the interatrial septum and sewing ring of the mitral valve. The valve leaflets, stents, and motion of the sewing ring appeared normal. Cardiac catheterization and cineangiography were performed immediately after two-dimensional echocardiography. A levophase pulmonary arteriogram confirmed the presence of a large mass in the left atrium.

We recommended urgent surgical exploration. However, while the patient was being prepared for operation approximately 14 hours after angiography, he suddenly developed electromechanical dissociation and could not be resuscitated. Postmortem examination revealed a large thrombus that nearly filled the left atrium and occluded the mitral orifice (fig. 4). The thrombus was attached to the interatrial septum and extended to the sewing ring. The valve leaflets and stents were not attached to the thrombus and the leaflets were mobile and coapted well.

Case 2

A 60-year-old man presented with increasing dyspnea on exertion, orthopnea, paroxysmal nocturnal dyspnea and pedal edema. Two years before admission mitral valve disease and complete atrioventricular block were diagnosed during an admission for minor trauma, and a permanent pacemaker was implanted. Symptoms of heart failure developed 4 months before hospitalization and progressed despite treatment with digoxin and diuretics. The patient was admitted for further evaluation.

Physical examination revealed a blood pressure of 140/80 mm Hg with a regular rhythm. The neck veins were distended with intermittent cannon A waves and a positive hepatojugular reflex. The apical impulse was diffuse, extending to the left midaxillary line in the fifth intercostal space. A grade III/VI holosystolic apical murmur and a I/VI systolic ejection murmur at the left sternal border were noted. Carotid pulsations were normal. There was 2+ pedal and sacral edema. The ECG demonstrated a paced rhythm. A chest x-ray revealed cardiomegaly, pulmonary venous congestion and bilateral pleural effusions. An M-mode echocardiogram demonstrated left ventricular cavity dilatation and holosystolic prolapse of both mitral valve leaflets. The left atrium was enlarged (45 mm) but was otherwise normal. Two-dimensional echocardiography demonstrated mitral valve prolapse, but revealed no vegetations or masses on the mitral valve or in the left atrial cavity. After treatment, cardiac catheterization was performed. No evidence of mitral stenosis or aortic valve disease was found. On left ventriculography there was 3-4+ mitral regurgitation with mitral valve prolapse and a diffusely hypokinetic left ventricle. Coronary arteriography demonstrated significant stenotic lesions in the left anterior descending and right coronary arteries.

At surgery a #33 Edwards porcine heterograft was
placed in the mitral position and saphenous vein grafts were inserted into the left anterior descending and right coronary arteries. There was no evidence of left atrial clot. Two days after surgery he developed transient left hemiparesis and impaired speech. No etiology for these problems was evident on physical examination or M-mode echo. A two-dimensional echocardiogram was performed. On several sector orientations from the anterior precordial long-axis and subxyphoid long-axis views, a poorly defined group of mobile echoes in the superior portion of the left atrium was imaged. Although left atrial thrombus was suspected, the lack of clear definition of this group of echoes did not allow a firm diagnosis. The neurologic symptoms rapidly cleared and were completely resolved in 48 hours. Nine days postoperatively, symptoms of heart failure developed without physical findings suggestive of prosthetic dysfunction. M-mode echocardiography showed left atrial enlargement (52 mm) with no other atrial abnormalities. Another two-dimensional echo was performed, and the anterior precordial long- and short-axis, apical two- and four-chamber and subxyphoid long-axis views demonstrated a large echo-dense mass in the superior-posterior portion of the left atrium (fig. 5). The superior aspect of the mass of echoes was mobile and the larger basal portion was contiguous with the left atrial posterior wall. This atrial mass had not been present on the preoperative two-dimensional echo (fig. 6) and was larger and had more distinct margins than at the earlier postoperative study. A levophase pulmonary angiogram was performed but was not diagnostic of an atrial mass. Anticoagulation was initiated. Nineteen days postoperatively the patient developed acute pulmonary edema but
responded to diuretics and oxygen. He improved over the next few days, but then developed acute abdominal pain, fever and hypotension, and exploratory laparotomy was performed. Multiple infarcts in the abdominal viscera and bowel were found but attempted embolectomy was not successful. He died soon after this surgery.

Postmortem examination revealed extensive recent embolization to the superior mesenteric and celiac arteries. In the left atrium two separate but contiguous thrombi, partially endothelialized, were attached to the atrial wall (fig. 7). The first was slightly pedunculated and attached to the posterior atrial wall and measured 1.5 × 1.0 × 0.5 cm. One centimeter superior to this a second, larger thrombus was noted with a large portion free within the atrial cavity. The estimated age of these thrombi was 2–3 weeks. The mitral porcine prosthesis was well seated and was not obstructed by the thrombi. The position of these thrombi and their size correlated well with the atrial mass identified by two-dimensional echo.

Case 3

A 53-year-old man was admitted with a 5-day history of orthopnea and increasing dyspnea on exertion. Six years earlier he had undergone mitral valve replacement with a Starr-Edwards ball valve prosthesis for progressive congestive heart failure due to presumed traumatic rupture of chordae tendineae. Two years later he developed periprosthetic regurgitation, and the Starr-Edwards ball valve was replaced with a Björling-Shiley prosthesis. One month before admission he developed fever, chills, and night sweats. Physical examination revealed crisp prosthetic valve sounds and no murmurs. The chest x-ray demonstrated cardiomegaly. Multiple blood cultures were negative. M-mode echocardiography revealed a normal excursion of the disc prosthesis and the expected postoperative abnormal interventricular septal motion. The left atrium measured 50 mm. Two-dimensional echocardiography showed no evidence of vegetation on the prosthesis and prosthetic motion appeared normal, as did the left atrial cavity. His symptoms improved and fever disappeared with a short course of antibiotic therapy. He was discharged, although a definitive diagnosis was never made.

At the time of admission 1 month later, physical examination revealed a blood pressure of 100/50 mm Hg and a regular pulse of 60 beats/min. His temperature was normal and there were no peripheral signs of embolic phenomena. Tachypnea and bibasilar rales were noted. Cardiovascular examination revealed jugular venous distension and a positive hepatojugular reflex. The point of maximal impulse was hyperdynamic and displaced laterally. Prosthetic valve sounds were crisp and a new grade III/IV holosystolic murmur was present at the apex, as was a third heart sound. Chest film showed cardiomegaly with increased pulmonary venous markings and a right pleural effusion. The prothrombin time was 22.8 seconds, with a control of 11.7 seconds. Blood cultures were negative.

Prosthetic dysfunction with mitral regurgitation was clinically diagnosed; however, it was unclear if the mitral insufficiency was secondary to perivalvular leak or central regurgitation from interference with disc motion. M-mode echocardiography when compared with the previous study showed continued normal excursion of the mitral prosthetic disc, with no evidence of vegetation; however, septal motion had normalized, suggesting the development of significant regurgitant leak. The left atrial dimension was 50 mm and there was no evidence of left atrial thrombus. Two-dimensional echocardiography was performed and on the anterior precordial long-axis and subxiphoid long-axis views a large area of increased echoes was identified in the left atrium just above the sewing ring, suggesting thrombus adherent to the prosthesis (fig. 8). In addition, the prosthetic valve showed an abnormal rocking motion of the sewing ring between systole and diastole (22°). There was loss of continuity between the sewing ring and the adjacent tissue surfaces of the annulus medially, indicating partial dehiscence of the prosthetic valve.

Figure 7. Postmortem view of the left atrial thrombi in patient 2. A pedunculated area is contiguous with a basal portion of thrombus (small arrows). The annulus of the mitral valve is indicated by the large arrow.
The patient was taken directly to the catheterization laboratory and left ventricular angiography was performed using standard techniques. This study confirmed severe periprosthetic leak with dehiscence of the valve ring. Opacification of the left atrium with regurgitant contrast revealed a radiolucent mass attached to the atrial edge at the sewing ring that was felt to be an adherent thrombus. Surgery was performed the next day, and on direct inspection a spherical thrombus approximately 1.5 cm in diameter was present on the atrial aspect of the prosthetic sewing ring (fig. 9). The valve sewing ring was detached along 60% of its circumference on the posterior and medial surfaces. The detached valve was replaced with a new prosthesis and the patient recovered uneventfully and was discharged home.

Discussion

Left atrial thrombus formation is generally considered to be secondary to abnormalities of atrial size, rhythm, or wall surface properties that occur alone or in combination with abnormalities of the mitral valve. Formation of left atrial thrombi is a recognized postoperative complication of mitral valve prosthetic placement and may recur in patients with thrombus formation in the preoperative period, despite surgical removal of the clot, or may appear de novo after mitral valve replacement. Diagnosis may be difficult on clinical grounds and contrast opacification of the atrium using levophase pulmonary arterial or transseptal atrial angiography is usually required for nonoperative diagnosis. However, left atrial opacification by levophase pulmonary arteriography may be relatively insensitive for demonstrating left atrial clots, with false-negative studies occurring as illustrated by our second patient. Transseptal catheterization of the atrium with angiography involves a potentially serious risk for the patient with intra-atrial thrombus. M-mode echocardiography is not reliable for identifying atrial thrombi. In our first patient the appearance of a dense pattern of echoes in the posterior portion of the left atrium on the postoperative M-mode echo was suggestive of a left atrial clot (fig. 1). However, this type of echo pattern may be nonspecific and did not allow a firm diagnosis, so that further confirmation was felt necessary. M-mode echocardiography failed to demonstrate abnormal echoes or serial changes in the left atria of the other two patients.

Two-dimensional echocardiography, on the other hand, has been used to identify intracardiac thrombus formation when M-mode techniques have failed. The tomographic display provided by two-dimensional echocardiography increases the imaged area and displays a more familiar anatomic format than does the single-crystal examination. Each cardiac chamber can be imaged in its entirety and spatial relationships more accurately assessed.

This report illustrates the ability of two-dimensional echocardiography to detect left atrial
thrombi of various sizes, ages, and morphologic characteristics in patients with prosthetic mitral valves. Several criteria were useful in identifying the presence of left atrial masses by two-dimensional echo in these patients. The echo-producing masses were confined to a clearly defined left atrial cavity and their presence was noted on multiple echocardiographic views. Thrombi were identified on the anterior precordial long- and short-axis, apical two- and four-chamber and subxyphoid long-axis views in the first two patients. In the third patient the thrombus was demonstrated on the anterior precordial long-axis and subxyphoid long-axis views only. Inability to image the thrombus in this patient on the apical views probably relates to the proximity of the clot to the prosthetic sewing ring. Since the echo beam on this view must pass through the prosthesis before encountering the atrial cavity, masses closely adherent to the atrial side of the prosthesis are more likely to be obscured by reverberations than masses more distal in the atrial cavity, as in the first two patients. We did not find any single view to be better in visualizing the masses, although the short-axis views from both the anterior precordial and subxyphoid windows seemed least useful. It is important to visualize the masses on multiple views, as this reduces the possibility of generating false-positive studies from artifacts that might be present on a single view. In all three of our patients the borders of the masses were well defined. In our second case a two-dimensional echo on the second postoperative day demonstrated a group of amorphous echoes without distinct margins along the superior-posterior border of the left atrium. One week later these echoes had increased in size and distinct margins were noted (fig. 5), raising the possibility that some degree of organization of thrombi is needed to allow acoustical differentiation of thrombi from blood or cardiac structures.

Several other features of these cases allowed characterization of the masses as thrombi. Serial two-dimensional echograms had been obtained in each of our cases. Comparison of serial studies not only allowed clear identification of a left atrial mass, but also suggested thrombus as the most plausible cause of the masses when the time course and clinical setting were considered. Certain characteristic motion patterns of these masses on the two-dimensional echo may also suggest thrombus. Since left atrial clots have variable age, size, morphologic characteristics and attachments to the intra-atrial surfaces, it is not surprising that the overall motion of clots in different patients would vary. In fact, the thrombi in each of our patients had somewhat different patterns of intracavitary motion. In patient 3, the small size of the thrombus and its attachment to the sewing ring of the mitral prosthesis caused it to move closely with the dehisced prosthesis, showing little intracavitary motion. In patients 1 and 2 the thrombi displayed some independent intracavitary motion, moving toward the mitral valve orifice in diastole. The thrombi of these two patients also showed a second, low-amplitude undulating or jiggling motion during the cardiac cycle. This second type of motion may be characteristic of thrombi that have some free intracavity movement, but may be less likely with mural thrombi that adhere to intra-atrial surfaces. This undulation or jiggling may be an expression of the consistency of the thrombus or the lack of firm attachment of certain portions of the thrombus and thus may be useful in identifying an intra-atrial mass as thrombus in cases where the lack of serial echoes or the clinical history make the diagnosis less clear than in our patients.

These cases do not define the sensitivity or specificity of two-dimensional echocardiography for detecting left atrial thrombi, and several limitations should be mentioned. Flat mural thrombi that adhere closely to the contour of the atrial wall may not be adequately detected by two-dimensional echocardiography, a shortcoming of cineangiographic techniques as well. Also, thrombus formation confined to the area of the atrial appendage or clots that are smaller than those reported here may be more difficult to identify than large or mobile atrial thrombi that involve the main body of the left atrium. The age of the clot and its degree of organization may also limit detection by two-dimensional echocardiography, but the experience with patient 2 suggests that thrombi may be imaged within days of their formation.

Awareness of the potential for false-positive images and techniques for avoiding this problem are important. Gain variation aids in identifying the atrial wall and margins of thrombi. Artifact arising from various cardiac structures such as the tricuspid annulus, an abnormal aortic valve or aortic valve annulus, the interatrial septum or pulmonary veins may appear in the left atrial cavity secondary to reverberation, beam width or beam angulation. Motion of left atrial echoes duplicating that of an identified cardiac structure such as the tricuspid annulus identifies such echoes as reverberations or secondary to beam width. In contrast, thrombi in patients 1 and 2 demonstrated a unique form of motion that was clearly different in real time from identified cardiac structures. Clear identification of a suspected mass on several echocardiographic views also aids in distinguishing thrombi from artifact, because reverberations from a given structure may vary in position with changes in transducer position, while thrombi remain in a specific area of the atrial cavity.

The three cases presented demonstrate the ability of two-dimensional echocardiography to identify thrombus formation in the left atrium and raise the possibility that this echocardiographic method may supplant the need for angiographic techniques. Although further experience with this technique is required, initial reports of detection of other intracardiac thrombi and the cases presented here indicate that two-dimensional echocardiography is useful for detecting intracardiac thrombi.

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