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Case Report Open Access

Left Atrial Slow Flow and Its Potential Complication

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Abstract

It has been estimated that 2.2 million people in United States have paroxysmal or persistent atrial fibrillation. The incidence has increased 13% over the past 20 years and it seems to be on the rise. The most feared complication of atrial fibrillation is systemic thromboembolic events. Mitral stenosis and atrial fibrillation is risk factors for cardiac thrombus formation. We report an interesting case in which a 69-year-old male with rheumatic mitral stenosis, atrial fibrillation and congestive heart failure developed rapidly worsening dyspnea on anticoagulation. Subsequent investigation demonstrated impaired left ventricular systolic function, severe mitral valve stenosis and a large left atrial thrombus.

Since the patient's symptoms had worsened rapidly during a relatively short period of time, we hypothesize that the thrombus was obstructing blood flow through the left atrium and was causing the patient's symptoms. Mitral valve stenosis, itself, is a risk factor for thromboembolic events. Combined with atrial fibrillation and left ventricular systolic dysfunction, slow flow in the left atrium may contribute to thrombus formation.

Even though, our patient was anticoagulated, the stability of oral anticoagulation therapy is essential. We want to highlight one particular group of patients, classified as high risk, in which close clinical follow-up and more intense anticoagulation might be of benefit. Also, the possibility of thrombus formation in the left atrium should be considered when there is a sudden change in symptoms.

Introduction

It had been estimated that 2.2 million people in United States have paroxysmal or persistent atrial fibrillation (AF). The incidence has increased 13% over past 20 years and it seems to be on the rise [1,2]. Severe mitral valve stenosis results in left atrial dilatation and is frequently associated with AF. Mitral stenosis and AF are risk factors for cardiac thrombus formation. This risk increases when the problems are combined. Recent studies show that the prevalence of left atrial thrombus in the patient with mitral valve stenosis in normal sinus rhythm is 6.6% compared with 38% in patient with AF [3,4].

The most feared complication of left atrial thrombus is systemic embolization, which occurs in 10-45% of patients with mitral stenosis [5]. The presence of left atrial thrombus also has important therapeutic implications, not only in terms of anticoagulation, but also the possible need for valve replacement.

We present an interesting case in which a male with mitral stenosis and AF developed rapidly worsening dyspnea most likely due to the increase in size of thrombus in the left atrium while the patient was anticoagulated.

Case Presentation

A 69 year-old Hispanic male with diabetes, hypertension, stroke, coronary artery disease, cardiomyopathy, rheumatic mitral valve stenosis and chronic AF presented with rapidly increasing shortness of breath. The patient reported rapidly declining functional capacity during the 4 weeks period prior to evaluation. The patient complained of lack of energy and the development of paroxysmal nocturnal dyspnea. Physical examination demonstrated an elderly male in no distress, normal vital signs, irregularly irregular rhythm with grade 1 diastolic rumbling murmur without radiation.

The patient has a known history of rheumatic mitral valve disease. He had balloon valvuloplasty in 1999. He then developed AF with cerebrovascular accident in 2001, and anticoagulation therapy was initiated.

Transesophageal (TEE) echocardiography demonstrated decreased left ventricular systolic function with ejection fraction of 35-39%. It also revealed severe mitral valve stenosis with effective orifice area of 0.8 cm^2 and the presence of a large echogenic mass in the left atrial appendage extending into the left atrium. Subsequent coronary angiogram showed no significant coronary artery disease (Figure 1). Mitral valve replacement and thrombus removal were performed (Figures 2 and 3). A left atrial mass was confirmed to be thrombus with histopathology.

Discussion

This case is interesting in that it demonstrates both 2-Dimensional and 3-Dimensional echocardiographic images of both the left atrial thrombus and the mitral valve stenosis (Figure 4-6). Despite the large size of the left atrial thrombus, this thrombus was visualized on TEE images, but was not seen on transthoracic images. Since the patient's symptoms had worsened rapidly during a relatively short period of time, we hypothesize that the thrombus was obstructing blood flow through the left atrium and was contributing to the patient's symptoms. It is reasonable to assume that the patient's dyspnea was a result of both his mitral stenosis and the thrombus in combination, limiting forward blood flow.

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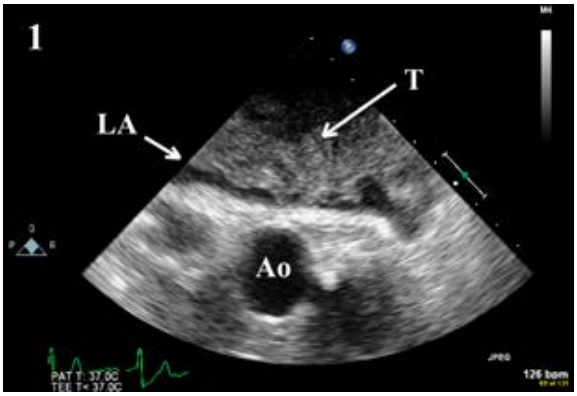


Figure 1: 2-Dimensional Transesophageal Echocardiography (TEE) demonstrated huge left atrial echogenic mass in the left atrial appendage extending into the left atrium. (LA – Left Atrium, T – Thrombus, Ao – Aorta**).**

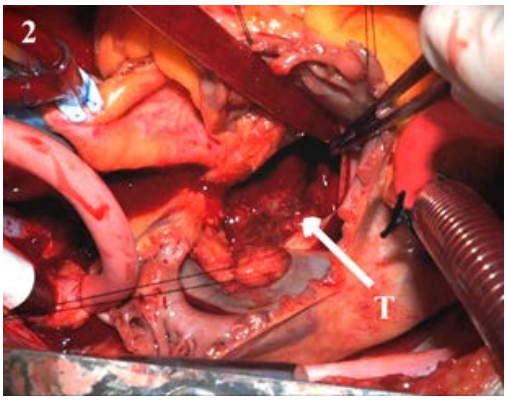


Figure 2: Intraoperative image of a giant thrombus in the left atrium.

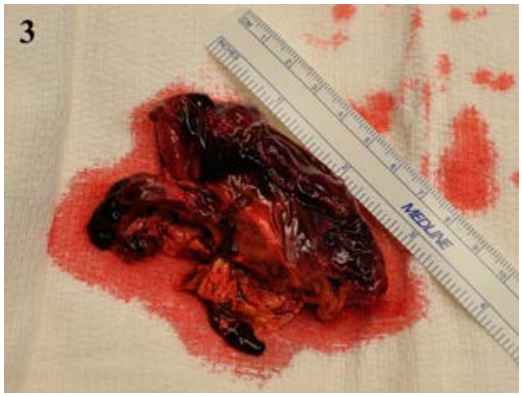


Figure 3: A Giant thrombus (8 × 4 × 2 cm) was removed from the left atrium.

It is interesting that such a large left atrial thrombus could develop in a patient who was receiving anticoagulation. Estimating the risk of stroke for individual AF patients is crucial in making the decision to provide anticoagulant therapy. The threshold risk which warrants anticoagulation is controversial. Recent guidelines suggest that patients with a stroke risk of 2% per year or less (classified as low risk) do not benefit substantially from oral anticoagulation [6]. For high-risk AF patients with stroke rates of ≥ 6% per, the comparable number needed-to-treat is 25 or fewer, strongly favoring anticoagulation.

Recent studies also include systolic heart failure (HF) with decreased ejection fraction as a "moderate-risk factor" as it is thought to cause increased risk for stroke and thromboembolic complications. Some reports demonstrate that patients with HF are hypercoaguable because of increased platelet activation and elevated coagulation markers such as D-dimer, thromboglobulin, and thrombin-antithrombin III complexes which could lead to thrombus formation [6].

Our patient presented with worsening shortness of breath which initially was thought to be secondary to progression of mitral valve

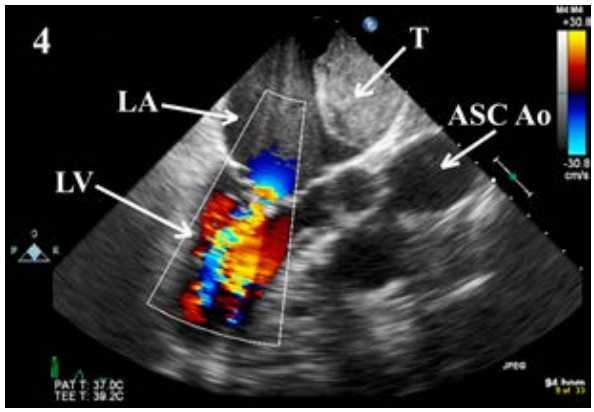
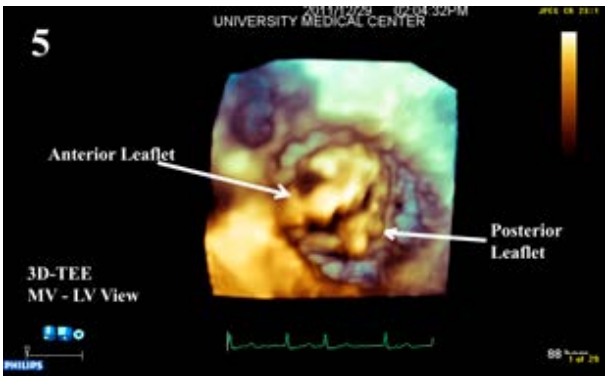
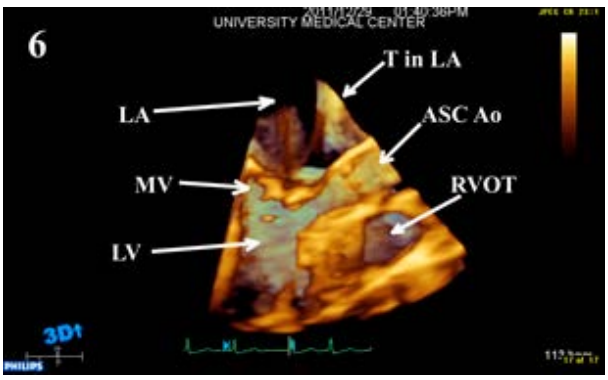


Figure 4: 2-D TEE (Color Doppler Mitral Valve inflow) demonstrated severe mitral valve stenosis and large left atrial thrombus. (LA – Left Atrium, LV – Left Ventricle, T – Thrombus, ASC Ao – Ascending Aorta).





Figures 5 and 6: 3-D TEE (Left ventricular view) demonstrated severe mitral valve stenosis and large left atrial thrombus. (LA – Left Atrium, LV – Left Ventricle, MV – Mitral Valve, T – Thrombus, ASC Ao – Ascending Aorta, RVOT – Right Ventricular Outflow Tract, T in LA – thrombus in Left Atrium).

disease. Subsequent investigation demonstrated a giant left atrial thrombus. Regarding risk stratification, our patient was classified as having 'high' risk due to prior stroke and rheumatic mitral stenosis. He also had multiple moderate-risk factors including diabetes, hypertension, and systolic heart failure. Even though our patient had already been treated with anticoagulant, stability of oral anticoagulation therapy is essential to avoid thromboembolic as well as bleeding complications. For the majority of these patients, a target INR (international normalized ratio) of 2.5 is optimal [7]. Recent guidelines also recommend that in patients with AF who have ischemic stroke or systemic embolism during treatment with low-intensity anticoagulation (INR 2.0 to 3.0); it may be reasonable to raise the intensity of anticoagulation to a maximum target INR of 3.0 to 3.5.

Summary

We want to emphasize that AF, mitral valve stenosis and left ventricular systolic dysfunction in combination cause slow flow in the left atrium, causing a high risk for thrombus formation. The practitioner should be aware that in high risk patients, even though patients are treated with anticoagulant, the possibility of thrombus formation in the left atrium may cause a sudden change in symptoms. In this group of high risk patients, close clinical follow-up and supra-therapeutic INR may be of benefit.

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