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.

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.
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...
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.

References