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Left Atrial Appendage Thrombus Despite Anticoagulation

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Abstract

The American College of Cardiology Foundation/American Heart Association task force on practice guidelines recommend therapeutic anticoagulation for at least 3 weeks prior to cardioversion in patients with atrial fibrillation of 48-hour duration or longer, or when the duration of atrial fibrillation is unknown. This case report demonstrates the presence of thrombi in the left atrial appendage despite adequate anticoagulation, challenging the current guidelines. Therapeutic anticoagulation for at least 3 weeks followed by transesophageal echocardiography in search of thrombus may enhance thromboembolic safety of elective cardioversion. Atrial fibrillation (AF) and heart failure (HF) have emerged as major cardiovascular epidemics in developed nations over the past decade. They share similar risk factors, seem to mutually accelerate progression and are associated with increased morbidity and mortality. Their relationship involves complex hemodynamic, neuro-hormonal, inflammatory and electrophysiologic mechanisms, which go beyond just mutual risk factors. This review focuses on updates in AF and HF with a hope of better understanding this relationship and the management of this complex duo.

Introduction

A 50-year-old man with a history of atrial fibrillation presented to the hospital with exertional dyspnea, lower extremity edema, and in atrial fibrillation with rapid ventricular response. His medical history included non-ischemic cardiomyopathy for which an implantable cardiovertor defibrillator (ICD) was implanted for primary prevention of sudden cardiac death, type II diabetes mellitus, dyslipidemia, and a prior transient ischemic attack so his $CHADS_2$ (Cardiac failure, Hypertension, Age ≥75, Diabetes, and Stroke [Doubled]) score was 4. He was diagnosed with atrial flutter 5 months prior to this presentation when he underwent successful electrical cardioversion and was started on anticoagulation with a vitamin K antagonist for a goal International Normalized Ratio (INR) between 2.0 to 3.0. Two months after cardioversion, the patient returned with recurrent atrial fibrillation. A pre-cardioversion transesophageal echocardiogram(TEE) showed no thrombus in the left atrium or left atrial appendage(LAA) (Image 1); however, multiple masses associated with atrial and ventricular defibrillator leads were observed. Thus, cardioversion was deferred. When the patient presented for the index admission, he had been on anticoagulation with an INR between 2.0 to 3.0 for 28 days. However, repeat TEE on this admission revealed new large lobulated thrombi in the left atrial appendage (Images 2 and 3). No cardioversion was performed and the patient was treated medically with increase in the goal INR to 2.5 to 3.5. A TEE a year later performed prior to pulmonary vein isolation and atrial fibrillation ablation showed LAA without thrombus.

Randomized studies of antithrombotic therapy are lacking for patients undergoing cardioversion of atrial fibrillation or atrial flutter. In case-control series, the risk of thromboembolism was between 1% and 5%.[1, 2] The risk was near the low end of this spectrum when anticoagulation (INR 2.0 to 3.0) was given for 3 to 4 weeks before and after conversion.[3-5] Transesophageal echocardiography has been suggested as a useful screen to identify patients without left atrial thrombus as an alternative to anticoagulation prior to cardioversion,[6-11] but a subsequent investigation[12] and meta-analysis found this approach to be unreliable.[13] In TEE-positive patients, left atrial thrombus can still be present in 11–50% of patients after a standard course of coumadin.[2] Cardioversion of these patients may or may not be associated with an increased embolic risk. Patients undergoing cardioversion using this approach still require post-conversion anticoagulation for at least 4 weeks.

The American College of Cardiology Foundation/American Heart Association task force on practice guidelines[14] recommend anticoagulation (INR 2.0 to 3.0) for at least 3 weeks prior to cardioversion in patients with atrial fibrillation of 48-hour duration or longer, or when the duration of atrial fibrillation is unknown (Class I Recommendation, Level of Evidence: B). As an alternative to anticoagulation prior to cardioversion, it is reasonable to perform

Disclosures:
None.

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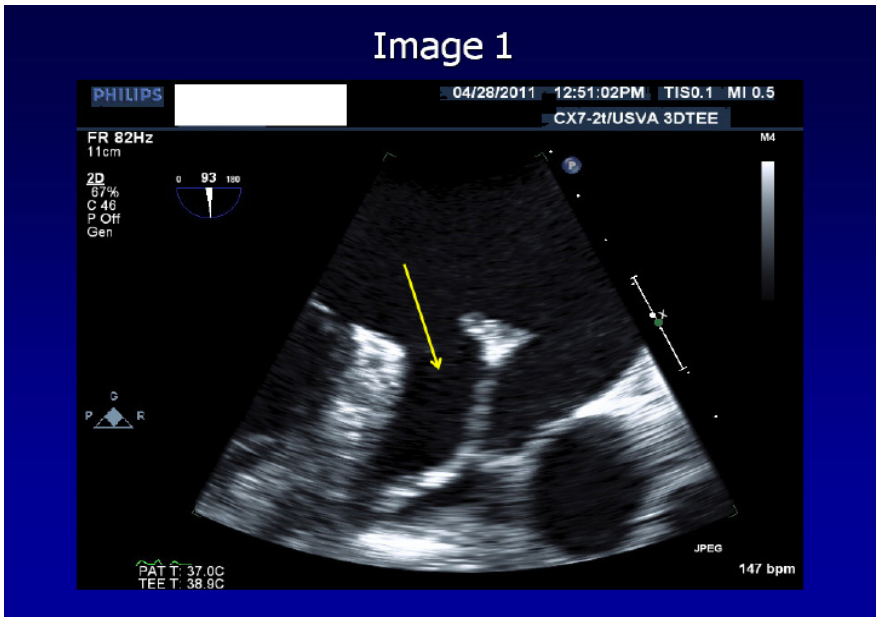

Figure 1: Still frame of 2-D transesophageal echocardiogram showing left atrial appendage without any thrombus (arrow)

TEE in search of thrombus in the left atrium or left atrial appendage. (Class IIa Recommendation, Level of Evidence: B).[14] Our case demonstrates the presence of thrombi in the left atrial appendage despite adequate anticoagulation for 4 weeks. Review of all patients referred for TEE prior to atrial fibrillation ablation at our hospital identified 3 more patients in the past 4 years who were found to have thrombi in the left atrial appendage despite therapeutic anticoagulation for at least 3 weeks.

At least one case of persistent thrombus in the left atrial appendage despite sufficient anticoagulation was described in the literature.[15] These findings challenge the current guidelines on prevention of thromboembolism in patients with atrial fibrillation undergoing cardioversion.[14] The combination of the two approaches, namely, anticoagulation (INR 2.0 to 3.0) for at least 3 weeks followed by TEE in search of thrombus in the left atrium or left atrial appendage should probably be recommended to enhance thromboembolic safety of elective cardioversion.

Conclusions:

AF and HF share common mechanisms and treatment strategies; consequently, therapies directed toward HF may protect the heart against the occurrence of AF. Although restoration of sinus rhythm in patients with HF may offer hemodynamic and clinical benefits, recent clinical trials have failed to demonstrate the clinical advantage of sinus rhythm over optimal rate control. The deleterious effects of currently available antiarrhythmic drugs, coupled with their low efficacy, may blunt the potential benefit of sinus restoration. A variety of therapies, including drugs, devices, and ablation procedures, are available to aid in the management of symptomatic and asymptomatic patients with both AF and HF. Recent advances in catheter-based

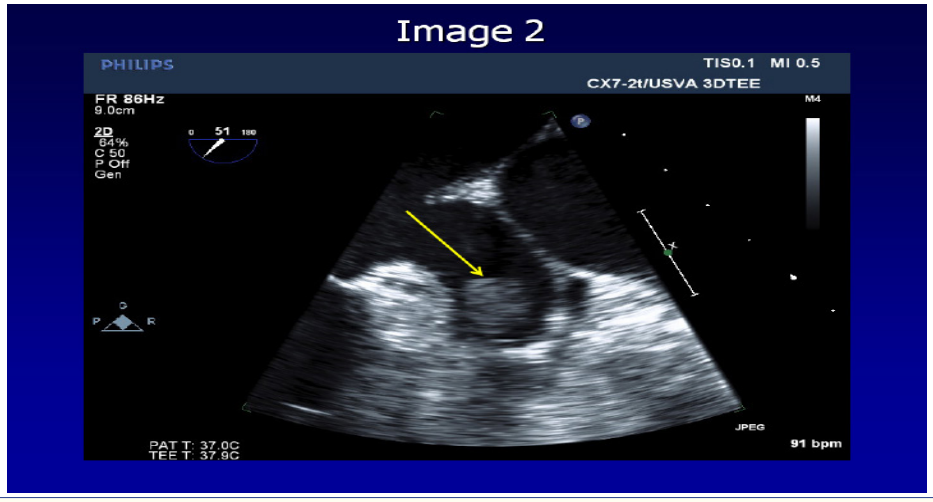

Figure 2: Still frame of 2-D transesophageal echocardiogram showing left atrial appendage with a thrombus (arrow)

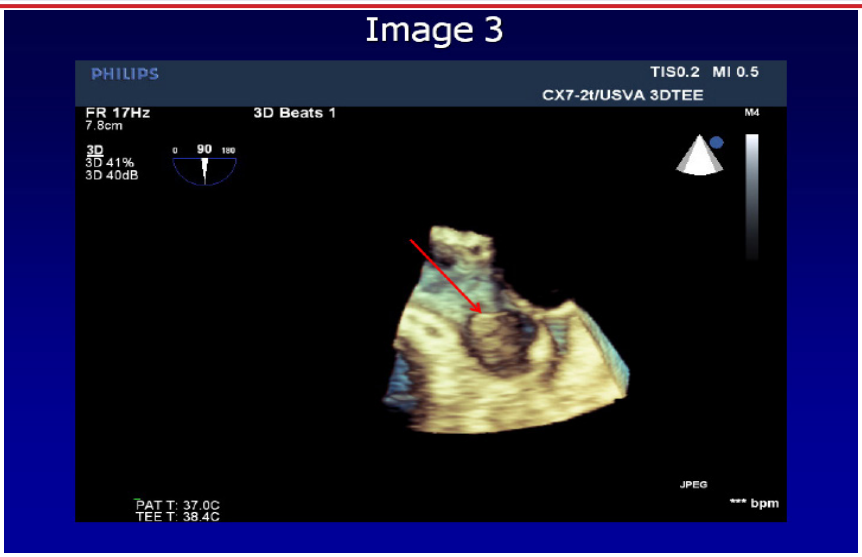

Figure :3 **Still frame of 3-D transesophageal echocardiogram showing left atrial appendage with a thrombus (arrow)**

ablative therapies for AF have been demonstrated to be effective in well-selected patients with HF, resulting in significant improvements in cardiac function, symptoms, and quality of life.

Along with further advances in pharmacotherapy and catheter-based ablative therapies, more trials comparing ablation with medical therapy in patients with AF and HF are needed before a standardized therapy for patients with AF and HF can be recommended. Based on the available therapies outlined above, it is fair to say that an individualized approach is a better strategy and might help improve symptoms and prognosis for patients with AF and HF.

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