Left Atrial Cavity Thrombus and Fatal Systemic Embolization in a Stroke Patient with Nonvalvular Atrial Fibrillation: A Caveat against Left Atrial Appendage Closure for Stroke Prevention

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Abstract

An 88-year-old male with nonvalvular atrial fibrillation (NVAF) and severe congestive heart failure (HF), was admitted to the Neurological Intensive Care Unit because of the acute onset of aphasia and left hemiplegia. Transthoracic echocardiography revealed a left atrial (LA) cavity thrombus. Its “fatal” distal embolization to abdominal aorta occurred in a few days. These observations should lead to a cautious approach in proposing a percutaneous closure of LA appendage in older NVAF patients, with HF and/or left ventricular dysfunction and larger LA volumes, who are not adequately anticoagulated.

Keywords: Left atrial appendage occlusion, left atrial cavity thrombus, nonvalvular atrial fibrillation

INTRODUCTION

It is generally accepted that in nonvalvular atrial fibrillation (NVAF) patients, left atrial appendage (LAA) is the most common source of thrombus resulting in a stroke. However, the location of atrial thrombus in NVAF patients could also be extra appendage. Here, we illustrate a case of a left atrial (LA) cavity thrombus detected in a nonanticoagulated NVAF patient with severe heart failure (HF). In the present case, a preventive LAA occlusion would not have prevented its “fatal” distal embolization to the abdominal aorta.

CLINICAL COURSE

An 88-year-old male, body surface area 2.04 m², with a long history of hypertension, suffering from polidistreccual vasculopathy and coronary artery disease (previous anteroapical myocardial infarction), in dual antiplatelet therapy, was admitted to the Neurological Intensive Care Unit because of the acute onset of aphasia and left hemiplegia. Electrocardiogram showed normofrequent atrial fibrillation (AF), heart rate 70 bpm, with old anterior wall myocardial infarction. Blood tests documented mild anemia (hemoglobin 12.5 g/dl) and severe kidney failure (estimated glomerular filtration rate <30 ml/min/1.73 m²). Chest X-ray showed cardiomegaly, pulmonary congestion, and mild bilateral pleural effusion. Multiple cortical and subcortical right hemisphere embolic lesions, involving temporal and occipital lobes and the right neocapsular area, were detected on the brain magnetic resonance imaging. A transthoracic echocardiography, performed on the 2nd day of hospitalization, revealed severe left ventricular (LV) dilatation (LV volume indexed of 95 ml/m³ and dysfunction (LV ejection fraction of 28%), severe LA enlargement (LA volume indexed of 70 ml/m³), moderate mitral and tricuspid regurgitation, systemic venous congestion, and severe pulmonary hypertension (systolic pulmonary artery pressure of 70 mmHg). Notably, an echogenic large ball-shaped floating mass (dimensions 3 cm × 2.5 cm) was observed within the LA cavity (LAC);
the mass was attached to the atrial roof (likely through a thin stalk), in proximity of the right upper pulmonary vein ostium. [Figure 1 and Video 1] This finding was attributed to a LAC thrombotic mass. Left atrial appendage (LAA) was not investigated by transesophageal echocardiography, due to the patient’s critical condition. A repeat echocardiogram, 2 days later, documented the complete disappearance of the mass within the LAC, [Figure 2] confirming its thrombotic nature and suggesting its systemic embolization. In the present case, AF was not previously detected, and the patient was treated with aspirin 100 mg/die, clopidogrel 75 mg/die, subcutaneous enoxaparin sodium 4000 IU/b.i.d. (adjusted dosage according to the estimated degree of renal function), bisoprolol 2.5 mg/die, amlodipine 5 mg/die, and finally furosemide intravenous 60 mg/die. On the 4th day of hospitalization, the patient showed a sudden abdominal pain spread to both inferior limbs and progressive signs of hypoperfusion secondary to acute critical ischemia. A contrast-enhanced computed tomography was performed on the same day, and a near total occlusive saddle embolus of the aortic bifurcation was demonstrated. [Figure 3] The next day, the patient’s condition quickly worsened for the occurrence of ongoing congestive symptoms and signs. The patient’s exitus was ascertained on the 5th day after the hospital admission.

**Discussion**

AF is an important cause of disabling stroke and systemic embolism and is increasingly prevalent with advancing age.\(^1\) Therefore, elderly patients with AF are often candidates for long-term anticoagulation. However, the frequent coexistence of several comorbidities, frailty, polypharmacy, chronic kidney disease, and dementia strengthens the perception that risk–benefit ratio of anticoagulant therapy could be unfavorable and explains why such treatment is underused in the elderly.\(^2\) Furthermore, there are several contraindications to oral anticoagulation therapy (OAC), such as active bleeding, intracranial hemorrhage, or neurosurgical procedure with high risk for bleeding within the past 30 days, an intracranial neoplasm or vascular abnormality with high risk of bleeding, recurrent life-threatening gastrointestinal bleeding events, severe thrombocytopenia, severe liver disease, and finally patients with a poor prognosis approaching the end of life.\(^3\)

It is generally accepted that in NVAF patients, LAA is the most common source of thrombus resulting in a stroke. This evidence has led to the development of the possible indication of LAA closure as an alternative treatment to prevent strokes in high-risk patients with NVAF, who are not candidates for OAC or in whom OAC therapy has failed.\(^4\)–\(^6\)

However, the location of atrial thrombus in NVAF patients could also be extra-appendage, that is, attached to the LA free wall or atrial roof or interatrial septum.\(^7\) If not detected and properly treated, a LA thrombus may cause an embolism, syncope, congestive HF, and sudden death.\(^8\)–\(^9\)

The meta-analysis of Mahajan et al.\(^10\) reported that 11% (95% confidence interval 6%–15%) of atrial thrombi, detected by tranesophageal and transthoracic echocardiography in NVAF patients, were located outside the LAA; this percentage increased till 22% in studies enrolling high-risk older and nonanticoagulated NVAF patients. The main factors associated with LAC thrombus include severe LV dysfunction, larger atrial volume, recent stroke, and anticoagulation status.

In a recent study,\(^11\) LAC thrombi were detected in only 0.07% of AF patients, electively candidate to external electrical cardioversion; however, population was mainly composed of anti-coagulated patients.

The present case highlights that in old and nonanticoagulated NVAF patients with severe LV dysfunction and cardioembolic
stroke, the thrombus formation was within the LA chamber, near the atrial roof and in the proximity of the right superior pulmonary vein ostium. The thrombus detachment from the atrial wall and its “fatal” distal embolization to abdominal aorta occurred in a few days.

These observations should lead to a cautious approach in proposing a percutaneous closure of LAA in older NVAF patients, with HF and/or LV dysfunction, larger LA volumes, who are not adequately anticoagulated given that thrombus could be in the atrial chamber. LAA closure alone will not eliminate the chances of thromboembolism in this cohort of patients.\[12\]

In conclusion, these data should be carefully considered when an LAA closure, and the consequent interruption of anticoagulant therapy, is planned in NVAF patients who are at high risk for LAC thrombosis.

Declarations of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form, the patient has given his consent for his images and other clinical information to be reported in the journal. The patient understands that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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