Case Report

Giant Atrial Thrombus in a Patient with Mitral Stenosis: A Case Report

Trombo Atrial Gigante em Paciente com Estenose Mitral. Relato de Caso

Julia Garcia Leal Elias[1], Maurício Marson Lopes[1], Fernanda Betanho Mori[1], Luana Monferdini, Maria Ligia Ciscon[1], Guilherme Viana Barbosa[1]

[1] Hospital da Pontifícia Universidade Católica de Campinas, Campinas, SP, Brazil.

Introduction

intracardiac thrombi can occur in any cardiac chamber. They are either formed in the heart or are formed in other locations, and then, migrate to the heart. Mitral stenosis (MS) and atrial fibrillation (AF) are important risk factors for the formation of intracardiac thrombi. The incidence of thrombi in MS patients is up to 17% but its incidence increases two-fold in the presence of AF.[1,2] Identification of intracardiac thrombus in patients with AF-associated MS is essential for selection of an appropriate therapeutic approach and surgical planning in view of the high morbidity and mortality associated with the embolic events that occur during disease progression and intraoperatively.[3,4]

Case report

a 67-year-old Caucasian female patient born in Aracajú, Sergipe, Brazil, and living in Itatiba, São Paulo, Brazil, presented with progressive dyspnea with minimal exertion for six months associated with orthopnea, paroxysmal nocturnal dyspnea, palpitations, and lipothymia, and sought the echocardiography sector of the Hospital of the Pontifical Catholic University of Campinas, São Paulo, on April 17, 2019, to undergo transthoracic echocardiography (TTE) as requested by the attending physician for clinical assessment. The patient had a history of systemic arterial hypertension, type 2 diabetes mellitus, chronic AF without anticoagulation, and rheumatic fever during childhood. She was receiving daily doses of valsartan (320 mg), atenolol (50 mg), hydrochlorothiazide (25 mg), metformin (1,700 mg), acetylsalicylic acid (100 mg), and sertraline (50 mg). She did not smoke or consume alcohol or illicit drugs. The results of physical examination were normal. The patient was flushed, hydrated, afebrile, normotensive, and normocardial, with an oxygen saturation of 94% in ambient air. Lung examination showed a reduction in vesicular murmur at right base and crackles on both sides. Cardiovascular examination showed arrhythmic sounds, diastolic murmur (2+/6+) in mitral focus, and symmetrical edema (1+/4+) in the lower limbs.

Keywords

Mitral stenosis; Left Atrial Thrombus; Atrial fibrillation.

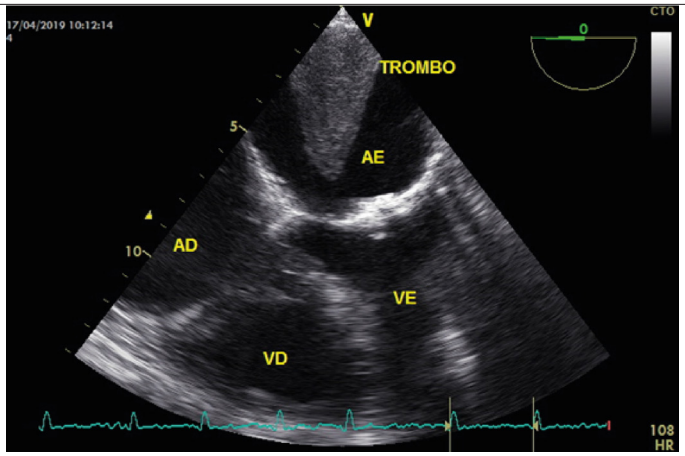
Mailing Address: Julia Garcia Leal Elias •
E-mail: julialeal@hotmail.com
Article received 11/4/2019; revised 11/24/2019; accepted 1/20/2020

DOI: 10.5935/2318-8219.20200034

TTE showed increased left atrial (LA) volume associated with an echogenic mass measuring 2.5 × 4.2 cm in apical four-chamber view and occupying the entire upper and middle portion of the LA. In addition, there was marked calcification of the mitral valve ring, left ventricle with normal global systolic function (ejection fraction of 55.45% using Teicholz's formula), no segmental change, right ventricle with diffuse hypokinesia and mild systolic dysfunction (CAF 23%), and marked pulmonary hypertension (pulmonary artery systolic pressure of 99 mmHg). Transesophageal echocardiography (TEE) was performed and a thrombus was detected in all views, especially at 0° and 120° (Figures 1 and 2), occupying the entire LA cavity, except the interatrial septum and the atrial face of the mitral valve. Pronounced calcification of the mitral valve annulus with commissural fusion, valvar area of 0.7 cm^2 (as revealed by two-dimensional planimetry), mean diastolic gradient of 9 mmHg (underestimated), and absence of mitral reflux was detected. The LA appendage was poorly developed, with a slow flow, and no evidence of an organized thrombus. Owing to the impairment of the LA systolic volume and decompensated heart failure compatible with hemodynamic profile B, hospital admission was requested for clinical compensation and assessment of need for the surgical approach.

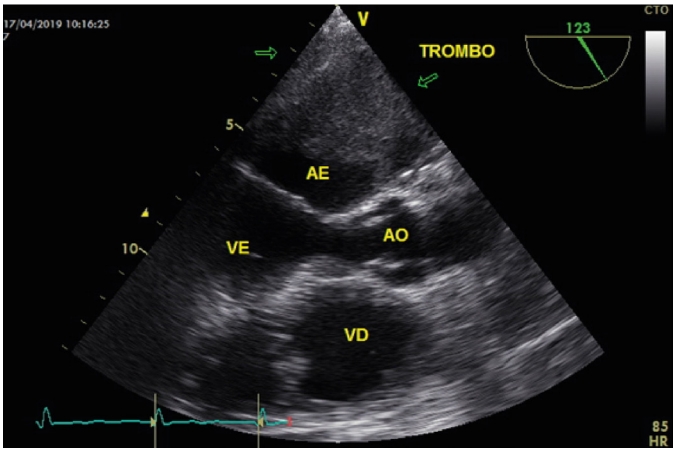
Laboratory tests were requested to assess the presence of anemia. Renal function and electrolytes, organ dysfunction, and infectious foci were within the normal range. Therapeutic optimization was performed and furosemide was intravenously administered due to decompensated heart failure. Full anticoagulant therapy with enoxaparin was used for the LA thrombus, and there was an indication for surgical intervention for mitral valve replacement, which was performed on April 22, 2019.

During the intraoperative period, organized and calcified thrombus was observed to be infiltrating the LA wall and pulmonary veins (Figure 3). The thrombus was successfully resected (Figure 3). The mitral valve was excised, and a biological prosthesis number 31 was implanted. The patient was referred to the coronary unit for postoperative care. On day 2 after the surgery, the patient presented fluctuations in the level of consciousness, and a computed tomography scan of the skull was performed, which showed no signs of hemorrhage, ischemia, or other changes. On day 12 after the surgery, the patient developed sepsis with pulmonary focus and acute respiratory failure and underwent orotracheal intubation and intravenous antibiotic therapy. The neurological level did not improve despite adequate treatment, and cranial tomography showed temporal cortico-subcortical hypodensity in the right middle cerebral artery



V, ventricle; RA, right atrium; LA, left atrium; LV, left ventricle; RV, right ventricle.

Figure 1 – *Transesophageal echocardiogram view at 0° showing a large thrombus in the left atrium, sparing the interatrial septum and the atrial face of the mitral valve, which shows marked calcification of its ring.*



V, ventricle; RA, right atrium; LA, left atrium; LV, left ventricle; RV, right ventricle.

Figure 2 – *Transesophageal echocardiogram view at 120° showing large echogenic mass (thrombus) in the left atrium.*

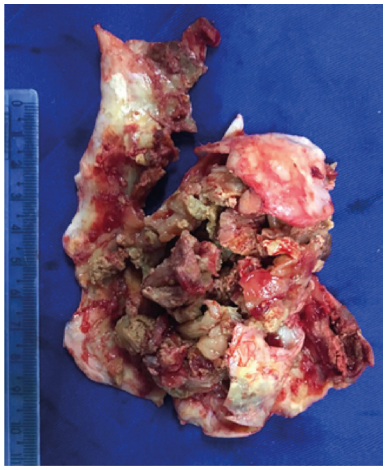


Figure 3 – *Material resected from the left atrium. Note the organized and infiltrative characteristic of the thrombus, in addition to important calcification.*

territory that was compatible with subacute ischemia, and lacunar hypodensity in the internal capsule and left portion of the thalamus that was compatible with lacunar infarction. Electroencephalogram showed marked diffuse depression of brain's electrical activity. On May 21, 2019 (day 29 after the surgery), the patient died from hemodynamic instability refractory to the clinical treatment.

Discussion

mS and AF are critical risk factors for the formation of intracardiac thrombi, whose incidence is approximately 17% in patients with MS, and the presence of AF increases this incidence two-fold. The incidence of an intracardiac thrombus in patients with only AF is unknown because previous studies have focused on symptomatic embolic events. Most thrombi are located in the LA appendage and may extend to the LA cavity in 2% of cases, and occupy the entire atrial extension in only a few cases.[1,2]

MS causes obstruction of the LA flow, and thus, increases atrial volume and local blood stasis. Structural, inflammatory, and fibrotic changes associated with valvular heart disease contribute to the development of AF, which further increases atrial volume and blood stasis. Patients with MS also exhibit decreased antithrombin III activity and an increase in factor VIII and platelet adherence and aggregation, which increases the thrombogenic potential.[2,5]

Echocardiogram is the most commonly used imaging technique for diagnosing this complication. Transthoracic echocardiography is recommended for detecting ventricular thrombus, and transesophageal echocardiography is useful for detecting atrial thrombus.[1,6] Anticoagulation therapy is the preferred treatment approach to prevent thrombus development and progression. Percutaneous or surgical removal may be useful in patients with high risk of embolization; however, surgical intervention in patients with large LA thrombi is associated with high mortality because of intraoperative embolization.[1,4,6]

Author contributions

Elias JGL conceived and designed the study; Lopes MM collected the data; Mori FB analyzed and interpreted the data; Mori FB, Monferdini L, Ciscon ML, and Barbosa GV wrote the manuscript; Elias JGL critically reviewed the manuscript for intellectual content.

Conflict of interest

The authors have declared that they have no conflict of interest.

References

1. Egolum UO, Stover DG, Anthony R, Wasserman AM, Lenihan D, Damp JB. Intracardiac thrombus: diagnosis, complications and management. Am J Med Sci. 2013;345(5):391-5.
2. Rost C, Daniel WG, Schmid M. Giant left atrial thrombus in moderate mitral stenosis. Eur J Echocardiogr. 2009;10(2):358-9.
3. Dawn B, Varma J, Singh P, Longaker RA, Stoddard MF. Cardiovascular death in patients with atrial fibrillation is better predicted by left atrial thrombus and spontaneous echocardiographic contrast as compared with clinical parameters. J Am Soc Echocardiogr. 2005;18(3):199-205.
4. Gallo JI, Ruiz B, Duran CM.. A safe Technique for removal of massive left atrial thrombus. Ann Thorac Surg. 1981;31(3):283-4.
5. Davison G, Greenland P. Predictors of left atrial thrombus in mitral valve disease. J Gen Intern Med. 1991;6(2):108-12.
6. Lin AN, Lin S, Lin K, Gokhroo R. A giant left atrial thrombus. BMJ Case Rep. 2017;2017. pii: bcr-2017-219792.