

Case reports

Silent embolism of a large pedunculated left atrial thrombus

Moreno Cecconi, Marcello Manfrin, Alessandra Moraca, Veronica Cacitti*, Epifanio Molini, Luigi Ferrini**, Gian Piero Perna

*Division of Cardiology, G.M. Lancisi Hospital, *Department of Pathology, University of Ancona, **Division of Cardiac Surgery, G.M. Lancisi Hospital, Ancona, Italy*

Key words:
Atrial mass; Embolism;
Thromboembolism.

Patients with a left atrial thrombus are considered as being at high risk for thromboembolic events. Although embolization may be asymptomatic, embolic events related to large thrombi are usually clinically manifest and tend to be associated with a worse prognosis.

We describe the very unusual case of a patient with atrial fibrillation in whom two-dimensional echocardiography revealed a large, pedunculated, highly mobile left atrial thrombus attached by a thin stalk to the interatrial septum. Because of the high risk of embolism the patient was submitted to urgent surgery. At surgery, only the stalk was found. No mass was visualized at intraoperative transesophageal echocardiography. The patient had an uneventful postoperative course without signs of embolism. Computed tomography of the brain did not reveal any cerebral infarction.

(Ital Heart J 2002; 3 (3): 199-201)

© 2002 CEPI Srl

Received September 24, 2001; revision received January 7, 2002; accepted February 4, 2002.

Address:

Dr. Moreno Cecconi

Via Guazzatore, 66
60027 Osimo (AN)
E-mail: morecconi@
tiscalinet.it

Introduction

In patients with atrial fibrillation the presence of a left atrial thrombus is associated with a high risk of thromboembolism¹⁻³. Previous studies have estimated that three fourths of strokes in patients with atrial fibrillation are due to embolism from left atrial thrombi^{1,2}. Although embolism may be asymptomatic^{4,7}, embolic events related to large thrombi are usually clinically manifest and tend to be associated with a worse prognosis^{8,9}. We report a case of a large, pedunculated, left atrial thrombus which dislodged from the interatrial septum without any sign of embolism.

Case report

A 70-year-old woman in whom a left atrial mass was detected at transthoracic echocardiography was referred to our hospital. Her medical history included systemic arterial hypertension and the development of atrial fibrillation 1 month before admission. Anticoagulant therapy with warfarin was started 24 hours after the onset of atrial fibrillation; the therapeutic value of the INR (> 2) was reached 4 days later. At the time of admission, the INR was 2.7. The results of the other routine blood

laboratory analyses were normal; in particular, the fibrinogen level was 335 mg/dl and the platelet count 197 000/mm³. The patient was not assessed for the presence of anticardiolipin antibodies.

Physical examination was unremarkable except for an irregular pulse and a grade II/VI systolic ejection murmur best heard at the upper right parasternal area. An ECG showed atrial fibrillation with a normal ventricular rate, signs of left ventricular hypertrophy and nonspecific ST- and T-wave changes.

Transthoracic echocardiography was suggestive of a mildly hypertrophied left ventricle with a normal systolic function, a mildly enlarged left atrium (antero-posterior diameter 4.5 cm; latero-lateral diameter 4.9 cm; supero-inferior diameter 5.5 cm) and a left atrial mobile mass attached to the interatrial septum. In order to better evaluate the mass, the patient was also submitted to multiplane transesophageal echocardiography. This showed spontaneous echo contrast in the left atrial cavity and left atrial appendage, low flow velocities in the left atrial appendage (peak filling and emptying velocities < 25 cm/s) and a pedunculated, highly mobile left atrial mass, approximately 2 cm long and 1.5 cm wide, attached by a thin stalk to the fossa ovalis of the interatrial septum (Fig. 1). Careful in-

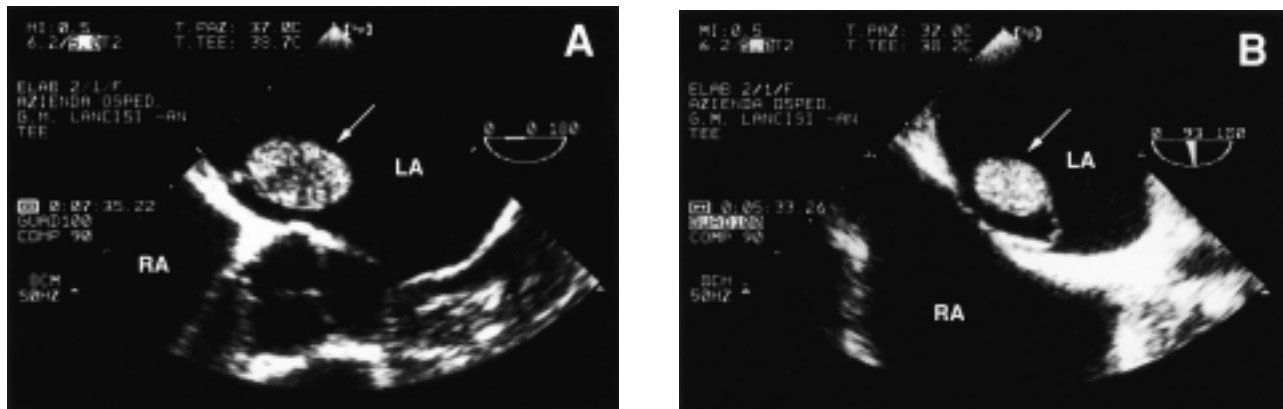


Figure 1. A: transesophageal echocardiographic view of the atria and interatrial septum (transverse plane) showing the presence of a left atrial mass (arrow) attached to the interatrial septum by a thin stalk. B: transesophageal echocardiographic view of the atria and interatrial septum (longitudinal plane 93°); the image demonstrates that the mass (arrow) is attached to the fossa ovalis. LA = left atrium; RA = right atrium.

spection of the remaining cardiac structures, in particular the left atrial appendage, revealed no additional masses. The systolic and diastolic forward flow velocities in the left upper pulmonary vein were 38 and 55 cm/s respectively. There was no evidence of patent foramen ovale. It was not possible to characterize the mass as a myxoma or thrombus on the basis of the echocardiographic characteristics alone. Because of the high risk of embolism the patient was submitted to urgent surgery. Heparin (3 mg/kg) was administered intravenously before the start of cardiopulmonary bypass.

At surgery, only the stalk was found; it was easily removed without excision, suggesting the diagnosis of thrombus. No mass was visualized at intraoperative transesophageal echocardiography. The patient had an uneventful postoperative course without signs of embolism. Computed tomography of the brain did not reveal any cerebral infarction. Histological examination of the stalk confirmed the diagnosis of thrombus (Fig. 2).

The patient underwent successful cardioversion 1 month after surgery. After 10 months of follow-up she is still asymptomatic.

Discussion

Clinically silent embolism has been found in a variety of clinical contexts including atrial fibrillation, asymptomatic carotid stenosis, symptomatic cerebrovascular disease, and prosthetic heart valves^{4-7,10-12}.

In particular, brain imaging techniques have demonstrated signs of cerebral infarction in the absence of a history or other clinical evidence of stroke in 15 to 26% of patients with atrial fibrillation⁴⁻⁶. Although the cause of silent cerebral lesions remains undetermined and several stroke mechanisms may be present in a particular patient^{5,6}, previous studies support the concept that at least some silent infarctions are caused by emboli⁴. Also, the detection of clinically silent circulating microemboli at transcranial Doppler ultrasonography is relatively frequent in patients with atrial fibrillation⁷; however, the significance of this asymptomatic microembolism has not yet been clearly defined.

Clinical symptoms of cerebral infarction are related to the size and number of the pathologically demonstrated cerebral lesions⁶. Silent infarctions tend

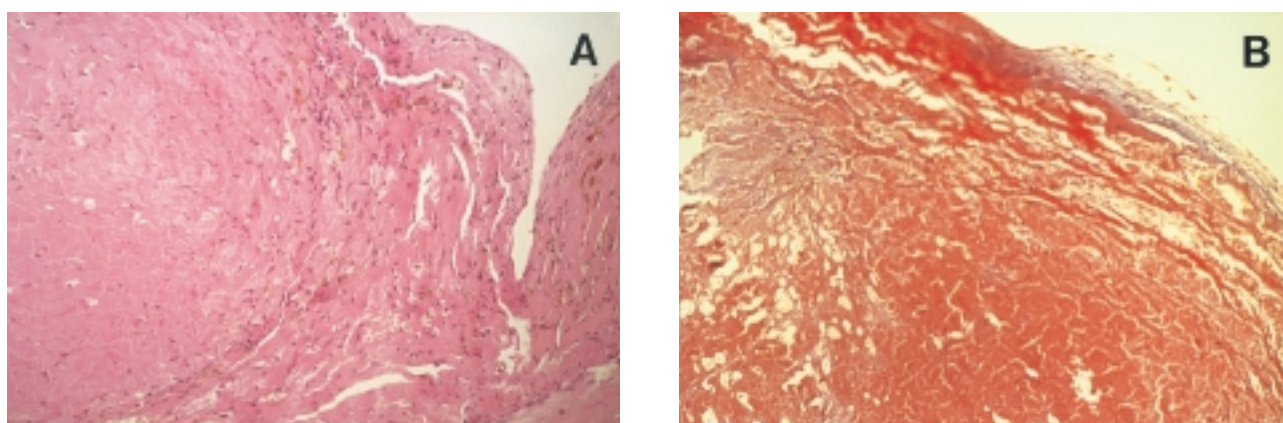


Figure 2. Hematoxylin-eosin (A) and Masson (B) staining: microscopic appearance of the stalk showing aggregated platelets in a scaffolding of fibrin with some incorporated red cells.

to be small and located deep within the brain^{4,6}. Embolism related to a cardiogenic cause, such as a left atrial thrombus, tends to be symptomatic because it is associated with larger infarcts, likely secondary to larger sized emboli and to the abrupt onset of vascular occlusion that may not allow for the development of an adequate collateral blood supply⁹. In our case, despite the size of the thrombus, there were no signs of embolism. We hypothesized that the anticoagulant therapy facilitated the dissolution of the thrombus which occurred beyond the origin of the aortic arch branches, resulting in asymptomatic embolism to the peripheral arteries.

It must be emphasized that we did not perform magnetic resonance imaging of the brain, which, compared with computed tomography, has a higher sensitivity for the detection of infarction; therefore, it cannot be ruled out that some small infarcts have been missed. However, it should be borne in mind that silent embolism of a large left atrial thrombus is a very rare event. To our knowledge, this is the first case report of an echocardiographically diagnosed large left atrial thrombus which dislodged almost completely without signs of embolism.

This case confirms that in patients with atrial fibrillation it may be very difficult to differentiate between a left atrial thrombus and myxoma on the basis of the echocardiographic picture alone^{8,13-15}. In the vast majority of cases, left atrial myxomas are attached by a stalk to the fossa ovalis of the interatrial septum; on the other hand, although the interatrial septum is a possible site of attachment of atrial thrombi^{8,13,14} a left atrial thrombus attached by a stalk to the fossa ovalis of the interatrial septum is a very rare finding. In our patient, the clinical context and the presence of spontaneous echo contrast in the left atrium suggested the diagnosis of thrombus whereas the site of the mass was consistent with the diagnosis of myxoma. New echocardiographic technologies, such as videodensitometry, integrated backscatter and Doppler tissue imaging, may provide additional information when assessing cardiac masses; in particular, Doppler tissue imaging is useful for a more precise definition of the motion pattern of normal and abnormal cardiac structures and, consequently, for a correct differential diagnosis of various cardiac masses¹⁶.

However, in our patient, independently of the nature of the mass, the high embolic potential had prompted urgent surgical intervention.

References

1. Asinger RW, Dyken ML, Fisher M, Hart RG, Sherman DG. Cardiogenic brain embolism. The second report of the Cerebral Embolism Task Force. *Arch Neurol* 1989; 46: 727-43.
2. Hart RG, Halperin JL. Atrial fibrillation and stroke. Revisiting the dilemmas. *Stroke* 1994; 25: 1337-41.
3. Leung DY, Davidson PM, Cranney GB, Walsh WF. Thromboembolic risks of left atrial thrombus detected by transesophageal echocardiogram. *Am J Cardiol* 1997; 79: 626-9.
4. Feinberg WM, Seeger JF, Carmody RF, Anderson DC, Hart RG, Pearce LA. Epidemiologic features of asymptomatic cerebral infarction in patients with nonvalvular atrial fibrillation. *Arch Intern Med* 1990; 150: 2340-4.
5. Ezekowitz MD, James KE, Nazarian SM, et al. Silent cerebral infarction in patients with nonrheumatic atrial fibrillation. The Veterans Affairs Stroke Prevention in Nonrheumatic Atrial Fibrillation Investigators. *Circulation* 1995; 92: 2178-82.
6. Shinkawa A, Ueda K, Kiyohara Y, et al. Silent cerebral infarction in a community-based autopsy series in Japan. The Hisayama Study. *Stroke* 1995; 26: 380-5.
7. Cullinane M, Wainwright R, Brown A, Monaghan M, Markus HS. Asymptomatic embolization in subjects with atrial fibrillation not taking anticoagulants. A prospective study. *Stroke* 1998; 29: 1810-5.
8. Zur-Binenboim C, Ammar R, Grenadier E, Veisler A, Freud M, Palant A. Detection of round floating left atrial thrombus simulating left atrial myxoma by two-dimensional echocardiography. *Am Heart J* 1985; 110: 492-3.
9. Timsit S, Sacco R, Mohr JP, et al. Brain infarction severity differs according to cardiac or arterial embolic source. *Neurology* 1993; 43: 728-33.
10. Kase CS, Wolf PA, Chodosh EH, et al. Prevalence of silent stroke in patients presenting with initial stroke. The Framingham Study. *Stroke* 1989; 20: 850-2.
11. Brott T, Tomsick T, Feinberg W, et al. Baseline silent cerebral infarction in the Asymptomatic Carotid Atherosclerosis Study. *Stroke* 1994; 25: 1122-9.
12. Grosset DG, Cowburn P, Georgiadis D, Dargie HJ, Faichney A, Lee KR. Ultrasound detection of cerebral emboli in patients with prosthetic heart valves. *J Heart Valve Dis* 1994; 3: 128-32.
13. Stoddard MF, Liddell NE, Korfhage L, Arce J, Kupersmith J. The transesophageal echocardiographic diagnosis of left atrial myxoma simulating a left atrial thrombus in the setting of mitral stenosis. *Clin Cardiol* 1992; 15: 379-82.
14. Morelli S, Testa G, Voci P. Large left atrial thrombus mimicking atrial myxoma: successful treatment with anticoagulant therapy. (letter) *Eur Heart J* 1996; 17: 1290.
15. Feinglass NG, Reeder GS, Finck SJ, Shine TSJ, Maniu CV. Myxoma of the left atrial appendage mimicking thrombus during aortic valve replacement. *J Am Soc Echocardiogr* 1998; 11: 677-9.
16. Bartel T, Müller S, Nesser HJ, Möhlenkamp S, Bruch C, Erbel R. Usefulness of motion patterns identified by tissue Doppler echocardiography for diagnosing various cardiac masses, particularly valvular vegetations. *Am J Cardiol* 1999; 84: 1428-33.