
Floating thrombus in the thoracic aorta: what should be done?

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Floating thrombus in a non-aneurysmal and non-atherosclerotic thoracic aorta is a rare event with potentially catastrophic complications. Especially younger patients have a higher risk of embolization. Transesophageal echocardiography is the diagnostic method of choice. However, the treatment of this pathology is still controversial and includes both medical and surgical options. We report our experience with a 43-year-old patient and a review of the literature, and make a proposal for therapeutic interventions.

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Introduction

A floating thrombus in the thoracic aorta is a rare event that may have catastrophic consequences¹ because of the risk of cerebral or systemic embolization. The treatment of patients with this finding is still controversial and includes surgical removal, thrombolysis or anticoagulation. We present a case in which a mobile thrombus in the descending aorta resolved with anticoagulant therapy.

Case report

A 43-year-old male with a previous history of heavy smoking presented to a secondary hospital with acute ischemia of his left lower limb secondary to thromboemboli, which was treated with percutaneous embolectomy. After a few days, he suffered a new embolic episode and underwent a femoro-femoral bypass with a saphenous vein graft. The postoperative course was complicated by persistent ischemia, which eventually led to amputation of the left leg below the knee. At that stage the patient was on anticoagulation therapy with intravenous heparin at 15 000 IU/day. A few days later he had two new episodes of ischemic attacks, one pertaining to the left hand (treated with thromboendarterectomy) and a cerebral transitory ischemic attack afterwards. The patient was then submitted to computed tomography scan and

magnetic resonance imaging which showed an endoluminal mass 1.5 mm in diameter, arising from the proximal descending aorta. No arrhythmia was reported at anytime. For this reason, he was transferred to our hospital.

An ECG, performed at the time of admission, showed sinus rhythm. Blood sample were taken for routine tests inclusive of a coagulation screen. A transesophageal echocardiogram (TEE) confirmed the previous findings (Fig. 1) showing a pedunculated, floating mass just below the left subclavian artery. There was no evidence of an endocavitary mass or of atrial "smoke" which would have been suggestive of stagnant flow. Initially, a surgical approach was contemplated and thrombus removal through a left thoracotomy under intraoperative TEE control was planned. The dose of intravenous heparin was increased to 25 000 IU/day. Three days later, when surgery was scheduled, a preoperative TEE performed in the operating room revealed that the thrombus had resolved. For this reason, the thoracotomy was not performed. The patient was then given oral anticoagulation therapy with warfarin, maintaining his INR between 2.5 and 3.5 (target 3.0) and the heparin was stopped once the therapeutic level was reached. Another TEE performed before discharge showed a normal aortic arch with only a minimal irregularity of the aortic contour.

At 1 month, the patient's conditions were still stable and 6 months later a TEE confirmed the complete resolution of the aortic thrombus (Fig. 2).



Figure 1. Preoperative transesophageal echocardiography showing the floating thrombus in the descending aorta.



Figure 2. Transesophageal echocardiographic image of the descending aorta 6 months following hospital discharge.

Discussion

Arterial embolism is always a dramatic and sudden event that usually reflects left heart disease, such as mitral stenosis, atrial dilation, myxoma, atrial fibrillation, bacterial endocarditis, and ventricular aneurysm. Other important non-cardiac sources of emboli include ulcerated atherosclerotic plaques either in the aorta or in the carotid arteries, while in a smaller percentage of cases the origin could be related to cancer, pregnancy, hypercoagulable states and also autoimmune disease such as the antiphospholipid antibody syndrome. In rare cases², the formation of the thrombus is due to a local endothelial abnormality at the insertion of the ligamentum arteriosus. In our case, we could not find any cardiac arrhythmia or disease to justify the repeated embolic events. In addition all the blood tests done were normal. Many investigations, including computed tomography scan, magnetic resonance imaging and even angiography^{3,4}, are currently employed to reach a de-

finite diagnosis. Just as other authors⁵⁻⁷, we believe that TEE is the gold standard to rule out a thromboembolic source, providing accurate images of the whole aortic lumen and information on the quality of the aortic wall, the presence and morphology of atherosclerotic plaques and of thrombi.

The ideal treatment of mobile aortic thrombi without atheromatosis is still controversial. Some authors^{2,4,8} have suggested an aggressive surgical approach, because of the potential hazard of emboli but Soyer et al.⁷ have shown that in spite of the surgical removal of the thrombus from the aorta, recurrent aortic thrombi and subsequent fatal embolization may still occur. Others have reported complete resolution of the floating thrombi following either thrombolysis⁹ or anticoagulation^{5,6}. In the case we present, despite the clinical history of recurrent emboli that indicated a surgical approach, we had the complete resolution of the pathological and clinical status with only anticoagulant therapy. We excluded thrombolysis because of the potential risk of repeat embolization that could result from a partial lysis of the clot. In addition, the patient had no recurrence at 30 days and 6 months of follow-up. Regarding the INR target of 3.0, this has been chosen because the thrombus was in the arterial and not in the venous bed and there was not any other concomitant disease requiring an INR target > 3.0. The appropriateness of this treatment has been confirmed by the absence of recurrences. In order to prevent the recurrence of thrombi we suggest, in these cases, that the treatment be maintained for life.

In conclusion, we believe that a clinical approach including aggressive intravenous anticoagulation should be the treatment of choice in these patients. It should be begun as early as possible because of the potential source of cerebral (less frequent) and peripheral arterial embolism (more frequent) and because of the significant risk of lysis of the thrombus. Considering the high risk of recurrence even after surgery, this has to be reserved only to those cases in which there are recurrent embolic events despite adequate intravenous anticoagulation. Once discharged, oral anticoagulants should be maintained for life.

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