Complicated course consequences of a floating thrombus in ascending aorta

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The ascending aorta may be the site of origin of systemic embolization of an unidentifiable cause. We report a case in which a free-floating thrombus of an unknown cause was detected in the ascending aorta via transesophageal echocardiography. The removal of this pedunculated thrombus, which was attached onto a macroscopically and histologically mildly atherosclerotic aortic wall, led to an uneventful recovery for the patient.

KEYWORDS
Aortic root; Thrombosis; Embolism

Introduction

We report a particularly interesting case in which a pedunculated free-floating thrombus of an unknown cause in the ascending aorta was attached to a mildly atherosclerotic aortic wall. The thrombus was thought to have been the origin of a previous left tibial artery embolic event.

Case report

A 50-year-old woman was apparently in good health until she was admitted to the emergency department of another centre for sudden ischaemia of the left leg. A left tibial artery occlusion was demonstrated by angiographic examinations. The patient was treated successfully with a transfemoral embolectomy. The patient had no previous history of thrombotic events, had never been exposed to heparin, and was on no medication at the time. A histological examination of the embolus disclosed a fresh thrombus composed of platelets, red-blood cells, polymorphonuclear cells, and fibrin. No evidence of malignant cells was found, nor did physical examination, chest roentgenogram, electrocardiogram, and Holter monitoring show any abnormalities. Two-dimensional transthoracic echocardiography (TTE) excluded pericardial effusion, mitral valve abnormality, and the presence of a thrombus in the left atrial appendage. The aortic valve was tricuspid with mild insufficiency and no stenosis. The four cardiac chambers and the ascending aorta were of normal dimensions. The left and right ventricular wall thickness and contractility were normal. No patent foramen ovale was detected on a colour Doppler echocardiogram. The patient, having been given heparin and then warfarin, was discharged from hospital.

A week later, however, she came to our hospital for further evaluation. The patient underwent transesophageal echocardiography (TEE), which confirmed the previous TTE findings but revealed the presence of a very mobile lobulated mass >2.5 cm in diameter and attached with a 5-mm base to the posterior wall of the ascending aorta 1.5 cm above the sinotubular junction (Figure 1) and it confirmed by 64-slice CT angiography (Figure 2).

Urgent surgery for the prevention of a recurrent embolic event, particularly brain embolization, seemed to be the only choice. Two hours later, she was in the operating room with her chest open. To ensure the safety of cannulation and find an appropriate clamp site, an epicardial echocardiography probe was employed to evaluate the mass. Unfortunately, the base of the mass had thinned to 1 mm, causing a very exaggerated motion (Figure 3, see Supplementary data online, Video S1). A safe clamp site was found, and through atrial cannulation, the patient was placed on cardiopulmonary bypass pump and her aorta was clamped with minimal manipulation. A cardioplegic injection was then administered followed by a transverse aortotomy 3 cm above the right coronary artery, which failed to reveal the mass. It was tragedy. The aortic wall and leaflets appeared mildly thickened, and mild macroscopic atherosclerotic changes were found on the implantation site of the mass. The left ventricle was explored through the aortic valve, but no thrombus was found. The mitral valve was intact.

Immediately afterwards, the aortic arch was opened directly with total circulatory arrest to expose the origin of the neck vessels. The mass, however, was nowhere to...
be seen. The patient's chest was closed and her limbs' vessels were evaluated with Doppler sonography on the operating table; there were still no signs of the elusive mass. Then, the patient being rushed to the CT scan ward with ambulatory mechanical ventilation. A 64-slice MSCT for an abdominal evaluation detected the mass 6 mm from the origin of the superior mesenteric artery (Figure 4). The patient was, subsequently, transferred back to the operating room, where her abdomen was opened, revealing a change of colour and no peristalsis in the small intestine. The mass was excised 7 mm from the origin of the superior mesenteric artery (within 2 h of embolization) (Figures 5 and 6), as a result of which the artery pulse and colour of intestine were restored. The patient was transferred to ICU and 7 days later was discharged home in good condition.

A histological examination of the mass revealed a fresh thrombus composed of a fibrinous material with polymorphonuclear cells, lymphocytes, and platelets. A histopathological evaluation of the aortic wall demonstrated mild evidence of atherosclerosis. No bacteria or fungus was present in the
found no hypercoaguable or systemic disorder that could have caused unusual thrombosis but only an atherosclerotic plaque, which could have led to endothelial injury and thrombus formation. Be that as it may, the exact mechanisms of giant thrombosis in a high-flow environment such as the ascending aorta are not fully known.

Neither antiplatelet agents nor antivitamin K such as warfarin has been shown to effectively prevent recurrent embolic episodes from aortic atheroma. On the one hand, therapeutic anticoagulation may paradoxically further embolic events by causing plaque haemorrhage, particularly in the presence of protruding atheroma, or by lysing the thin pedicle of the thrombus more rapidly than the mass itself. On the other hand, the efficacy of the anticoagulation in the presence of mobile atherosclerotic debris is questionable. In our patient, it is very likely that warfarin facilitated the embolization of the clot by lysing the base of the thrombus.

**Conclusion**

It is advisable that unusual sources of embolization be sought diligently and thoroughly during diagnostic evaluations because today the majority of sources is detectable and treatable, hence the preventability of the recurrent systemic embolism.

**Supplementary data**

Supplementary data are available at *European Journal of Echocardiography* online.

**Conflict of interest:** none declared.

**References**