

Giant Floating Thrombus Localized in the Ascending Aorta

Mahmut Akyildiz,¹ Yilmaz Zorman,¹ Tamer Aksoy,² Mustafa S. Yilmazer,³ Esra Erturk,¹ Cagatay L. Onar,¹ Ahmet Midi⁴

Departments of ¹Cardiovascular Surgery, ²Anesthesiology, ³Cardiology, and ⁴Pathology, Faculty of Medicine, Maltepe University, Istanbul, Turkey

ABSTRACT

We report the case of a 32-year-old male patient with symptoms of cerebrovascular accident manifesting with dysarthria. A transesophageal echocardiogram showed a floating mass localized in the ascending aorta, and a multislice computed tomography evaluation confirmed the diagnosis. With a comprehensive assessment of the mass, we decided on surgical intervention. A pedunculated and fragile mass was seen just near the right coronary ostium. The measured dimensions were 7.7 × 1.0 × 1.5 cm. The removed mass has been analyzed histopathologically and found to be the cause of the neurologic findings with an uncertain underlying etiology.

INTRODUCTION

It is rare to observe a floating mass originating from the ascending aorta. Such structures in this location are caused by an injury to the endothelium, such as atherosclerotic changes, arterial hypertension, or vasculitis [Culliford 1993]. The aorta as a source of embolism should be sought in patients with thromboembolic events with no obvious source.

CASE REPORT

A 32-year-old, previously healthy male patient was admitted to the emergency department with complaints of acute decline of speech and weakness in the right upper extremity. Dysarthria, sensorial aphasia, and right hemiparesis were observed in his neurologic examination at the time of admission to the emergency department.

The patient had a history of type II diabetes mellitus and had smoked 1 pack of cigarettes each day for 17 years. Magnetic resonance imaging showed a nonhemorrhagic infarct in the left cerebral hemisphere, the corona radiata, and the basal ganglia. We then performed lower-extremity venous Doppler ultrasonography, transthoracic echocardiography, and carotid-vertebral Doppler ultrasonography evaluations, which showed no evidence of a thrombogenic origin.

Received May 3, 2009; received in revised form October 30, 2009; accepted November 20, 2009.

Correspondence: Cagatay L. Onar, MD, Maltepe University, Faculty of Medicine, Feyzullah Cad No. 39, 34844 Maltepe, Istanbul, Turkey; +90-532-2827683; fax: +90-216-3830271 (e-mail: cagatay00@gmail.com).

A transesophageal echocardiogram revealed a floating, pedunculated mass originating from the ascending aorta and extending into the aortic arch. A thoracic multislice computed tomography (CT) examination showed the exact location, which was compatible with the transesophageal echocardiography results (Figure 1).

The patient's total cholesterol level was 253 mg/dL, the low-density lipoprotein concentration was 64 mg/dL, his triglycerides were 275 mg/dL, and the high-density lipoprotein level was 34 mg/dL. Lipoprotein electrophoresis showed a normal distribution.

Blood fibrinogen, homocysteine, protein C, protein S, and antithrombin III levels were measured to evaluate these variables as risk factors for formation of the thrombus. The results of these tests were within the reference intervals. The results of a genotypic analysis for the factor V Leiden mutation (G1691A) were also negative. The results of all other tests to clarify the etiology (tests for anticardiolipin

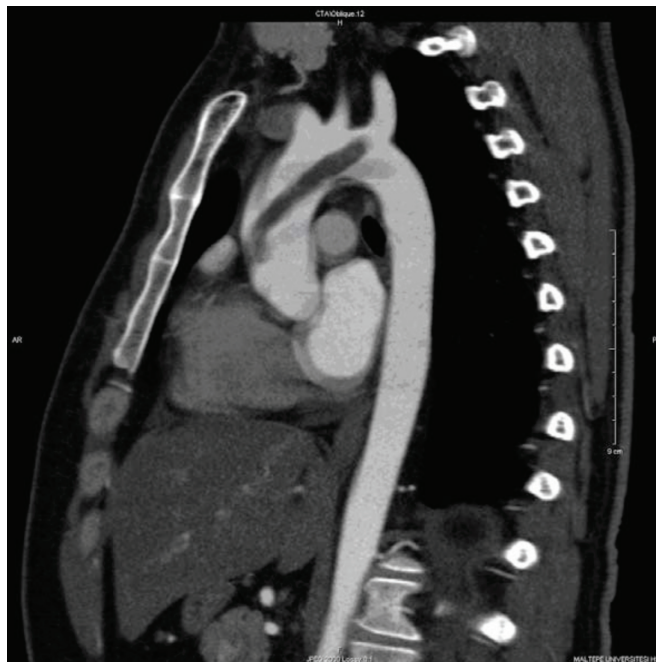


Figure 1. Thoracic multislice computed tomography scan showing the exact location of a floating, pedunculated mass originating from the ascending aorta and extending into the aortic arch.

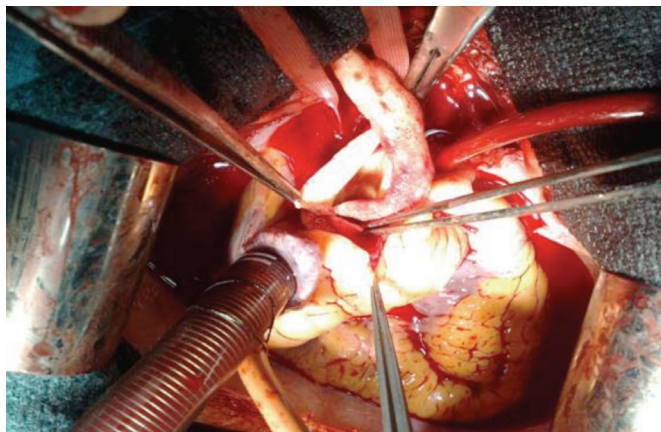


Figure 2. The aorta was incised longitudinally, and the mass was seen and delivered via the incision.

immunoglobulin M [IgM], IgG, anti-native DNA, anti-double-stranded DNA, antineutrophil cytoplasmic antibodies, and antinuclear antibodies) were negative.

The patient was scheduled for open heart surgery for excision of the thrombus. Arterial cannulation was made via the right femoral artery, and a venous 2-stage cannula was placed through the right atrium. A vent was inserted into the pulmonary artery. Extracorporeal circulation was initiated. Spontaneous fibrillation was maintained by decreasing the body temperature to 28°C. A very short period of total circulatory arrest was achieved as a step in the planned surgical intervention. The aorta was incised longitudinally, and the mass was seen and delivered via the incision (Figure 2). The cross-clamp was placed on the distal aorta. The aortic incision was repaired with continuous 5-0 polypropylene suture. The patient was weaned off the pump uneventfully after rewarming.

Macroscopically, the mass's dimensions were 7.7 × 1.0 × 1.5 cm, with a peduncle 2 mm long. The color of the mass was light pink to beige; the mass was fragile (Figure 3).

There were no problems during the postoperative follow-up period. The patient was discharged with the prescription of anticoagulant and antiaggregant treatment on the sixth postoperative day.

The patient has been seen by his hematologist at monthly intervals; all blood test results have been within reference intervals.

DISCUSSION

In our case, the pathology was attributable to the atherosclerotic degeneration of the ascending aorta and was diagnosed with transesophageal echocardiography. A plaque or mass >4 mm located in the ascending aorta or the proximal aortic arch must be considered the most important risk factor for an ischemic stroke, especially when it is mobile or floating [Amarenco 1994]. Nonetheless, a mobile thrombus located in the ascending aorta is a rare condition compared with other causes of ischemic stroke [Bruno 2001]. Thrombi originating from the



Figure 3. The dimensions of the light pink to beige mass were 7.7 × 1.0 × 1.5 cm, with a peduncle 2 mm in length.

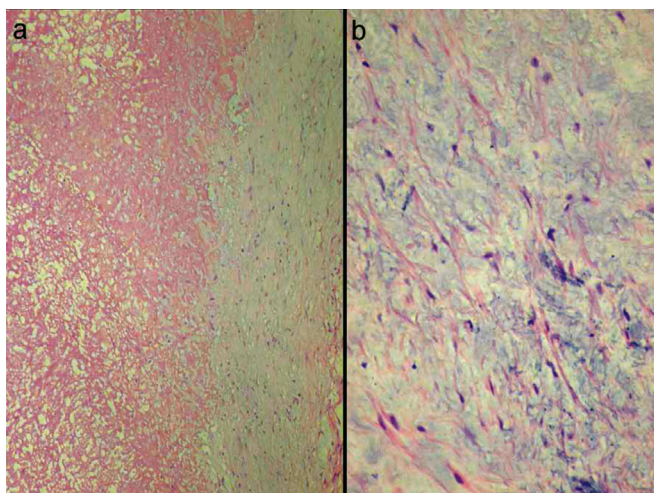


Figure 4. Histopathologic analysis of the mass. A, Hematoxylin-eosin staining showed neovascular structures with signs of early organization, especially around the pedicle. B, Periodic acid-Schiff-Alcian Blue pH 2.5 cytochemical staining showed mucinous depositions.

ascending aorta or the proximal aortic arch are reported to occlude coronary ostia and can clinically present with symptoms of acute myocardial infarction [Culliford 1993].

Atherosclerotic plaques, systemic diseases, or hypercoagulable situations are generally considered as the cause of thrombus in the ascending aorta. When there is no underlying condition, it is an interesting phenomenon to find a floating thrombus of that size and location, where the blood velocity and pressure are the highest in the body.

In our case, we found no evidence for any condition that may have predisposed the patient to the formation of thrombus.

A histopathologic analysis of the mass showed high myxoid degeneration and inflammatory cell infiltration over

the aortic wall. The samples stained with hematoxylin-eosin showed neovascular structures with signs of early organization, especially around the pedicle (Figure 4A). The endothelium of these vascular structures showed CD34 staining, and histiocytic infiltrations showed CD68 staining. Besides the histiocytic infiltration, rare lymphocytes were observed. The aortic wall from which the mass originated showed high myxoid degeneration and chronic inflammation. Periodic acid–Schiff–Alcian Blue pH 2.5 cytochemical staining showed mucinous depositions (Figure 4B).

Chronic infiltrations of inflammatory cells prompted the consideration of vasculitis, but there was no positive marker for vasculitis. Anticardiolipin IgG, IgM, and antinuclear antibody levels were normal. Systemic lupus erythematosus also was ruled out.

Mesenchymal/monocytic incidental cardiac excrescences (MICE) are benign, nonneoplastic proliferations of mesothelial cells admixed with histiocytes to form small excrescences [Luthringer 1990]. In our case, the histopathologic findings did not match those expected for MICE.

In patients who have symptoms of an ischemic stroke, the first attempts to investigate the etiology must consist of arterial carotid and vertebral Doppler ultrasonography and transthoracic echocardiography evaluations. When the results of these studies cannot explain the etiology, transesophageal echocardiography and contrast CT scans must be performed.

To decide on the treatment options, we performed transesophageal echocardiography and contrast CT scans to determine the exact location and size of the mass. After establishing that it was very large and so close to the right coronary ostium and the branches of the aortic arch, we decided on a surgical intervention.

Surgical excision or administration of thrombolytic therapy to pedunculated and mobile thrombi of the ascending

aorta and the proximal arch are the methods that have been reported by some authors [Hausmann 1992; Farah 1993]. Regarding the size and the location of the mass and its clinical presentation, surgical intervention was preferred and performed.

In this case, we have reported the successful surgery to remove a giant floating thrombus localized in the ascending aorta, before it was able to give rise to fatal complications [Sodian 2002].

REFERENCES

- Amarenco P, Cohen A, Tzourio C, et al. 1994. Atherosclerotic disease of the aortic arch and the risk of ischemic stroke. *N Engl J Med* 331:1474-9.
- Bruno P, Massetti M, Babatasi G, Khayat A. 2001. Catastrophic consequences of a free floating thrombus in ascending aorta. *Eur J Cardiothorac Surg* 19:99-101.
- Culliford AT, Tunick PA, Katz ES, et al. 1993. Initial experience with removal of protruding atheroma from the aortic arch: diagnosis by transesophageal echo, operative technique, and follow-up [abstract]. *J Am Coll Cardiol* 21:342A.
- Farah MG, Hawawini H. 1993. Thrombus of the ascending aorta as a source of cerebral embolism. *Chest* 104:1604-5.
- Hausmann D, Gulba D, Bargheer K, Niedermeyer J, Comess KA, Daniel WG. 1992. Successful thrombolysis of an aortic-arch thrombus in a patient after mesenteric embolism. *N Engl J Med* 327:500-1.
- Luthringer DJ, Virmani R, Weiss SW, Rosai J. 1990. A distinctive cardiovascular lesion resembling histiocytoid (epithelioid) hemangioma. Evidence suggesting mesothelial participation. *Am J Surg Pathol* 14:993-1000.
- Sodian R, Bauer M, Weng YG, Siniawski H, Koster A, Hetzer R. 2002. Floating nonocclusive thrombus in the ascending aorta. *Ann Thorac Surg* 74:588-90.