

Floating Thrombus in the Aortic Arch: A Case Report

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ABSTRACT

Floating thrombi in the aortic arch are very rare, and often go under-diagnosed. Herein, a case of an 8-cm long thrombus in the aortic arch is reported. It was a floating, highly mobile thrombus attached to the atherosclerotic plaque in the proximal aortic arch. The patient was a 59-year-old woman with a history of hypertension. The thrombus was operatively removed, with a favorable outcome. (Korean Circulation J 2005;35:180-182)

KEY WORDS : Arteriosclerosis ; Thoracic aorta ; Thrombus.

Introduction

Thrombosis and embolism are important causes of morbidity and mortality in a variety of disease. An arterial embolism usually reflects heart disease, which accounts for approximately 80% of all cases.¹⁾ Ulcerated atherosclerotic plaques in the aorta and carotid artery are an important noncardiac source of emboli. Embolization of thrombi in the aorta can also be associated with cancer, pregnancy and hypercoagulable states, and in rare cases, with the insertion site of ductus arteriosus.^{2,3)} In 10% of patients, the source of a peripheral embolism cannot be identified. Herein, a case of a large, floating thrombus attached to the atherosclerotic plaque in the aortic arch is reported, with review of some the literature.

Case

A 59-year-old woman presented with continuous numbness in the left arm and hand of 3 days duration, which was followed by mild epigastric pain, vomiting, diarrhea and chills. She had a 10-year history of hypertension. However, she had no

history of cigarette smoking or medical history of arrhythmias, ischemic heart disease, diabetes mellitus or stroke. On admission, she was hemodynamically stable, with blood pressure of 110/70 mmHg in the right arm, but 90/60 mmHg in the left, with a pulse rate of 78 beats per minute.

Her chest X-ray and electrocardiogram were normal. The diagnostic work-up to determine the source of the embolism included a transthoracic echocardiogram, which showed the possibility of vegetation on the aortic valve. On examination of the transesophageal echocardiogram, myxomatous changes of the aortic valve were verified. However, at that time, another important lesion was also detected, which was a long floating echogenic mass, with an irregular margin in the aortic arch (Fig. 1). This appeared to be attached to the posterior wall of the proximal aortic arch, extending to the left subclavian artery and descending aorta. Chest computed tomography showed a filling defect in the aortic arch (Fig. 2).

The laboratory data included: a white blood cell count of $6600 \times 10^3/\mu\text{L}$, hemoglobin 12.3 g/dL, platelets 235,000/ μL , negative ANCA, negative ANA, antithrombin III 90% (normal value: 75–125%), cardiolipin IgM antibody 0.4 MPL U/mL (normal value <4 MPL U/mL), protein C 100% (normal value: 80–120%) and protein S 70% (normal value: 70–123%).

She underwent surgery due to the risk of peripheral embolization. During the operation, a large thrombus, measuring more than 8 cm in length, was removed from the aortic arch,

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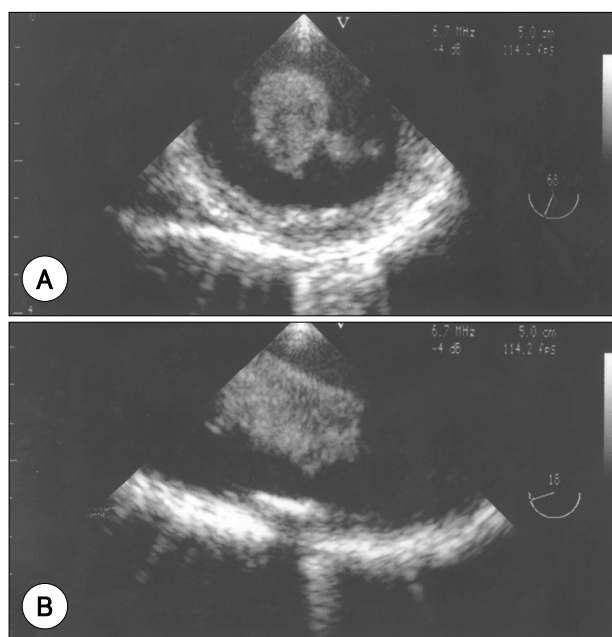


Fig. 1. Transesophageal echocardiography demonstrates a large free-floating thrombus in the aortic arch in transverse (A) and longitudinal (B) views.

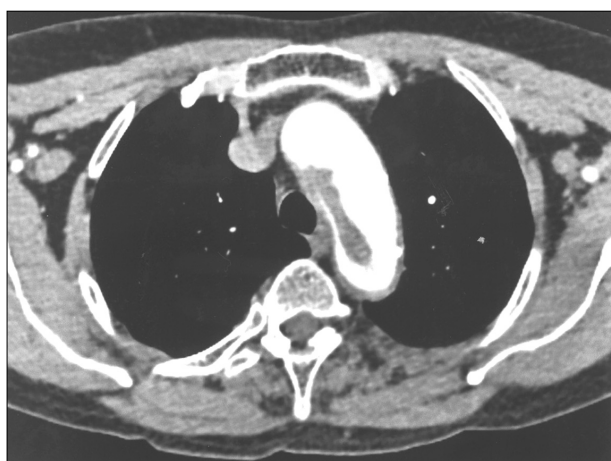


Fig. 2. Chest CT scan demonstrates the thrombus in the aortic arch. CT: computed tomographic.

left subclavian artery and descending aorta (Fig. 3). A small amount of residual thrombus was removed piecemeal, superficially, from the roughened base of the posterior wall of the proximal aortic arch. The base of thrombus was dimpled, like an atheromatous lesion. An organized thrombus, without evidence of malignancy, was found on histopathological examination. Two weeks later, after an uneventful recovery, she was discharged, with warfarin anticoagulation therapy.

Discussion

Most systemic embolisms are caused by thrombi in the left



Fig. 3. Organized thrombus. The base of the thrombus (arrow) and the floating end are shown.

side of the heart. Aortic thrombi, however, are another important cause of arterial thromboembolism.¹⁾ Some hypercoagulable states, e.g., primary polycythemia vera, antiphospholipid antibody syndrome, protein C deficiency, depressed activation of protein C and factor V Leiden deficiency, have been associated with aortic thrombi. Laperche et al.⁴⁾ reported that 17% of patients with a thrombosis of the aortic arch had evidence of a hematostatic disorder.

The presence of pedunculated thrombi in the thoracic aorta is a rare entity. These thrombi move freely in the aortic lumen with each cardiac cycle, and their fragmentation can lead to acute ischemic episodes due to peripheral, visceral or cerebral arterial embolization.⁵⁾ Pathologic studies of the aortic wall in these patients have shown atheromatous lesions, often with minimal atherosclerotic plaques.⁶⁾ The diagnosis of a hypercoagulable disorder in our patient was excluded due to the negative coagulation profile and lack of previously unexplained arterial or venous thromboses. The pathologic mechanism of the lesion in our case appeared to be due to the progressive development of an apposition thrombosis on an ulcerated plaque.

Treatment was considered necessary due to the risk of a massive systemic embolization. Karalis et al.⁷⁾ reported the incidence of embolic events in 73% of highly mobile aortic thrombi, compared with 12% of immobile ones. The optimal treatment of these pedunculated lesions remains undefined. Thrombolysis of clots has been reported in the literature.⁸⁾ Reber et al.⁹⁾ however, discussed the potential danger of thrombolytic agents selectively lysing the stalk of pedunculated lesions, releasing the bulk of the lesion into the bloodstream, thus causing a massive embolization. Thrombolysis for pedunculated thrombi may be contraindicated. In selected

patients, surgical treatment has been successful. Sadony et al.⁵⁾ described the successful removal of thrombi using hypothermic circulatory arrest. In the present case, the thoracic aortic floating thrombus was located in the aortic arch, so a surgical removal of the thrombus was performed using hypothermic circulatory arrest. The exact surgical risk and long-term results are not known, although no embolization has recurred during the 13 months of follow-up.

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