Case Report

A case of floating thrombus in the ascending aorta that caused recurrent peripheral arterial embolic events

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ARTICLE INFO

Article history:
Received 13 January 2015
Received in revised form 2 March 2015
Accepted 25 April 2015

Keywords:
Peripheral arterial emboli
Floating thrombus
Ascending aorta

ABSTRACT

We report the case of a 62-year-old man with recurrent arterial embolisms to his arms caused by a thrombosis of the ascending aorta. He had developed a left brachial artery embolism 8 years previously, but presented with a right brachial artery embolus on this occasion. A clot-like mass was seen in the ascending aorta on computed tomography without significant atherosclerosis. Magnetic resonance imaging identified multiple asymptomatic cerebral infarctions. Therefore, we surgically removed the thrombus in the ascending aorta, which was an organized fibrin clot. Pathologically, atherosclerosis and plaque formation were evident at the intima where the clot attached. Clot formation was considered to be due to local atherosclerosis.

We report a case of thrombosis of the ascending aorta causing multiple and recurrent arterial embolisms. The patient had no evidence of coagulation disorders, and arteriosclerotic risk factors such as hypertension, diabetes mellitus, and dyslipidemia were absent. Thus, thrombosis may develop in patients without traditional risk factors.

*Learning objective: We report a case of thrombosis of the ascending aorta causing multiple and recurrent arterial embolisms. The patient had no evidence of coagulation disorders, and arteriosclerotic risk factors such as hypertension, diabetes mellitus, and dyslipidemia were absent. Thus, thrombosis may develop in patients without traditional risk factors.

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Introduction

Aortic thrombi can be caused by blood disorders (e.g. protein S or protein C deficiency and anti-phospholipid antibody syndrome), tumors, aortitis, collagen disease, aortic structural abnormalities (e.g. aortic aneurysms), intra-aortic atheroma, hormone therapy, steroid use, and atrial fibrillation. Indeed, aortic thrombi are rare in patients without these causes, and thrombosis of the ascending aorta is rarer still. Here, we describe a patient with thrombosis of the ascending aorta that caused multiple cerebral infarctions and recurrent arterial embolisms of the arms.

Case report

A 62-year-old man presented with sudden-onset numbness and a cold sensation in his right arm in January 2014. He had no history of hypertension, diabetes mellitus, or dyslipidemia, but had previously smoked.

Eight years previously, he had left brachial artery embolism. Contrast-enhanced computed tomography (CT; Fig. 1a) had identified a clot-like mass attached to the aortic arch with asymptomatic cerebral infarction in the distribution of the left vertebral artery. Surgical embolectomy was performed, and postoperative anticoagulant therapy was provided. However, he developed diverticular bleeding that caused hemorrhagic shock, which necessitated surgical colectomy and the termination of anticoagulant therapy. He was subsequently able to tolerate antiplatelet therapy. Approximately 6 months later, the clot-like mass in the aortic arch had disappeared, even though he had stopped the antiplatelet therapy by himself.

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On this occasion, he was fully conscious with a blood pressure of 155/88 mmHg, and a heart rate of 103 beats per minute. His right brachial arterial pulse was palpable, but his right radial artery pulse was weak. In addition, his serum creatine kinase and D-dimer levels were elevated. Protein S and protein C levels were normal, anti-phospholipid antibody was negative (Table 1), and he had not taken steroid therapy. Electrocardiography (Fig. 1b), chest X-ray (Fig. 1c), or echocardiography revealed no abnormal findings.

Ultrasonography suggested that thrombus was likely in his right brachial and ulnar arteries, and contrast-enhanced CT identified arterial obstruction from the site of the upper elbow joint of the right brachial to the proximal ulnar artery (Fig. 1d). We also found a mass measuring approximately 10 mm in the ascending aorta located superior to the previous mass (Fig. 1e) and partial filling defects in both kidneys. CT findings were not consistent with aortic dissection, and there was no strong evidence of aortic sclerosis. There was no evidence of any lower limb arterial obstruction. We diagnosed an acute right brachial artery embolism and performed emergency embolectomy. Postoperatively, blood flow was improved to the right arm.

An echocardiogram revealed a floating mass in the ascending aorta, magnetic resonance imaging identified multiple asymptomatic cerebral infarctions that were thought to be recent, and there were no intra-cardiac thrombi. Thus, the origin of the embolism was thought to be the floating mass in the ascending aorta, and surgical enucleation was performed on day 2 of admission.
was hospitalization, he
mass examination
recurred. Thus, PT–INR,
flowing thromboembolisms
intraoperative echocardiography revealed that the floating
Intraoperative echocardiography revealed that the floating
mass was attached to the anterior wall of the ascending aorta
Fig. 1f). An aortotomy was performed, and a soft mass measuring
approximately 10 mm (Fig. 2a) was removed. The aortic wall
to which the mass attached was thickened (Fig. 2b). Histological
examination revealed a fibrin thrombus with partial organization
and without tumor cells. Severe atherosclerosis and plaque
formation were found on the aortic wall intima where the clot
was attached (Fig. 2c). Elastic fiber of the aortic media was
denatured.
Postoperative anticoagulant therapy was started with heparin.
Oral anticoagulant and antiplatelet drugs were started from day
2 after surgery. Atrial flutter developed on day 6 of admission, but
he returned to sinus rhythm eight hours later and it has not
recovered. His postoperative recovery was good, and he was
discharged on day 17 of admission. At the 1-year follow-up, he
had no signs of recurrence.

Discussion

The mass in the aortic arch was considered a thrombus 8 years
previously on contrast-enhanced CT. On this occasion he presented
with additional embolisms to his right arm and kidneys. Although
he developed atrial flutter for a short period during this
diagnostic period, CT found no clots in the left atria or left auricle.
Thus, we report a case of recurrent arterial embolisms caused by a
floating thrombus in the ascending aorta.
Cases of recurrent ascending aorta thrombi associated with
embolisms to the arms are extremely rare in patients without
underlying disease. To the best of our knowledge, there are no
previous reports of a case such as this. Laperche et al. described
23 cases of floating thrombi of the aorta [1]. In their report,
pathological examination revealed small atherosclerotic plaques in
21 cases with no evidence of severe aortic atherosclerosis or aortic
debris on transesophageal echocardiography.
Our patient had no aortic structural abnormalities, coagulation
disorders, neoplastic diseases, or aortitis. In addition, other than
a history of smoking, he had no risk factors for arteriosclerosis, such
as hypertension, diabetes mellitus, and dyslipidemia. Furthermore,
no significant arteriosclerosis was confirmed on either CT or echocardiography. Nevertheless, there was histological evidence of an atherosclerotic lesion where the thrombus attached to the aorta, suggesting that the thrombus formed at this site.

The most important aspects of this case were severe focal atherosclerotic progression in a localized area of the aorta with no other macro atherosclerosis and the subsequent development of a large thrombus at this small atherosclerotic area. We were unable to fully elucidate the pathophysiology underlying these events. We considered several possibilities. First, many typical atherosclerotic risk factors, such as hypertension, diabetes mellitus, and dyslipidemia, were absent. However, he had a past history of smoking; smoking is a major cause of arteriosclerosis obliterans, and it may have contributed to atherosclerosis in this case. Second, branching of the aorta to the carotid and subclavian arteries can cause turbulent blood flow, which induces shear stress at the aortic wall, leading to endothelial dysfunction and plaque formation at the aortic arch; this further precipitates clot formation. However, the exact cause of focal atherosclerosis progression and large thrombus development in a localized aortic area in this case were not elucidated. It is possible the patient had a coagulation disorder undiagnosed at the time of this report. We thus intend to carefully follow up this patient.

Atherosclerotic plaques larger than 4 mm and located in the aortic arch frequently cause recurrent cerebral infarction [2]. In such cases, the first-choice treatment is anticoagulation. However, due to the presence of multiple embolisms with cerebral and renal infarctions, we opted for emergency surgical extraction in our patient. Endovascular treatment of aortic thrombosis has been reported in some cases [3–5]. However, we performed surgical extraction because we could not completely exclude the possibility of a tumor.

Of note, there was a difference in the site of clot formation between events in this case. Due to the risk that recurrent aortic thrombi could develop at different sites in the aorta, we considered it appropriate to continue oral anticoagulant and antiplatelet treatment (warfarin and aspirin).

We conclude that thrombosis can develop in patients without traditional risk. It is necessary to check an intra-aortic clot in patients presenting with recurrent embolic attacks, even if they do not have traditional risk factors.

**Conflict of interest**

The authors state that they have no conflict of interest.

**References**


