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Ascending Aorta Thrombose: A Rare Cause of Simultaneous Acute Myocardial Infarction and Upper Limb Ischemia

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INTRODUCTION

Acute myocardial infarction (AMI) is generally caused by thrombotic occlusion of atherosclerotic coronary arteries. Although, the origin of the thrombus can be caused by diseased aorta with extension or emboli to coronary arteries;[1] or in rare cases we are faced with intact aorta (nonaneurysmal and/or atherosclerotic).[2] Floating thrombus of the normal ascending aorta is usually associated with hypercoagulative state including genetic disorders, malignancy, trauma, medications and medical instrumentations without clear mechanism.[3] Anyway, ascending aortic thrombus may lead to life-threatening thromboembolic events that carries increased morbidity and mortality. The optimal management of these patients is still controversial and depends on location and anatomy of lesions, duration of the occlusion, clinical presentation, patient risk factors (co-morbidities), and risk of the procedure.[4]

Herein, we report a particularly interesting case of acute inferior MI and left upper limb ischemia via extensive thrombosis from ascending aorta that treated via thrombectomy and medically with fibrinolytic and anticoagulation.

CASE REPORT

A 45 years old woman was transferred to our emergency department because of a 2 hours history of persistent central burning chest discomfort and pain begans suddenly at sleep time with associated nausea, vomiting and cold sweating that goes with paresthesia and pain in left arm.

In past history she had diabetes mellitus on oral hypoglycemic agent therapy including Metformin 500 mg twice a daily and not mentioned other disease, but she used oral contraceptive pills (LD type) for 25 years since 20 years old. Her family history for cardiovascular diseases was negative.

Initially the patient was awake and hemodynamically stable with arterial blood pressure 120/80 mmHg in right arm, heart rate about 88 beat per minute, respiratory rate 20 breath per minute, body temperature of 37.1 °C and arterial O2 saturation 90%. The blood pressure in left arm was not detected. In left arm physical examination, she had no focal motor or sensory deficits, but she experienced numbness and the arm was pale and pulseless with slow capillary filling. Exam of heart, lung and other limbs were normal.

At first a 12 leads electrocardiogram (ECG) was taken that showed ST-segment elevation in inferior leads (acute inferior MI) (Figure 1A).

Laboratory findings were almost within the normal limits, with the exception of a slightly normochromic and normocytic anemia. Initial coagulation markers, such as platelet count, prothrombin and activated partial thrombin times, were also within normal ranges. Cardiac enzymes, including troponin I and D-dimmer were increased.

A highly mobile echo dense mass (10 × 8 mm) in ascending aorta near the origin of right coronary cusp was detected accidentally during echocardiography assessment before coronary angiography (CAG) (Figure 1B). Other finding in echocardiography was normal except of a hypokinesia in base of inferior wall.

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According to above data, emergency coronary computed tomography (CT) angiography was performed to roll out of aorta dissection as source of aorta thrombosis and AMI. There was a thrombus formation in size of 11 × 10 mm in proximal part of tubular portion of ascending aorta with extension to right coronary artery (RCA) without evidence of aorta dissection or aneurysm (Figure 1C, D). Concurrent coronary CT angiography also showed thrombotic stenosis at proximal part and complete thrombotic occlusion at distal part of RCA before the posterior descending artery (PDA). Left brachial artery also was occluded at proximal probably due to embolization from the aorta thrombosis.

Regarding to high risk cases for systemic embolization, the coronary artery intervention was not performed and we continue the antiplatelet and anticoagulation therapy as AMI and limb ischemia treatment. For the best decision, the cardiac surgeon and cardiac interventionist (heart team) were consulted and we decided to emergency upper limb angiography and thrombectomy as a suitable intravascular treatment considering to marginally threatened (class II) acute arterial thromboembolism.

Left upper limb angiography was performed via femoral artery approach that revealed complete cut-off of the brachial artery flow at proximal part (Figure 2A). The 0.014 Asahi Sion Blue guide wire was crossed the thromboses and percutaneous aspiration thrombectomy was done successfully. Simultaneously, reteplase (Retelies, OSVE pharmaceutical Co. Iran) was infused at total doses of 10 unit (approximately 18 mg) in two separate stages with stepwise and blousing infusion method. Then we complete the reteplase dosage for AMI treatment and other 10 unit was injected intravenously. After this proceeding, the arm flow was established (Figure 2B) and she experienced improvement in pain and numbness of the hand and the radial pulse was detectable. The patient was transferred to cardiac care unit with heparin infusion intravenously (IV) concurrent with other MI medication treatment. Patient was asymptomatic and in control coronary CT angiography and echocardiography [transthoracic echocardiography (TTE) and transesophageal echocardiography (TEE)] there was not any thrombose in ascending aorta and left arm as well as normal epicardial coronary arteries (Figure 2C, D). The patient was started on warfarin therapy [with target international normalized ratio (INR) 2-3] with a covering heparin infusion until her INR became therapeutic. Be-

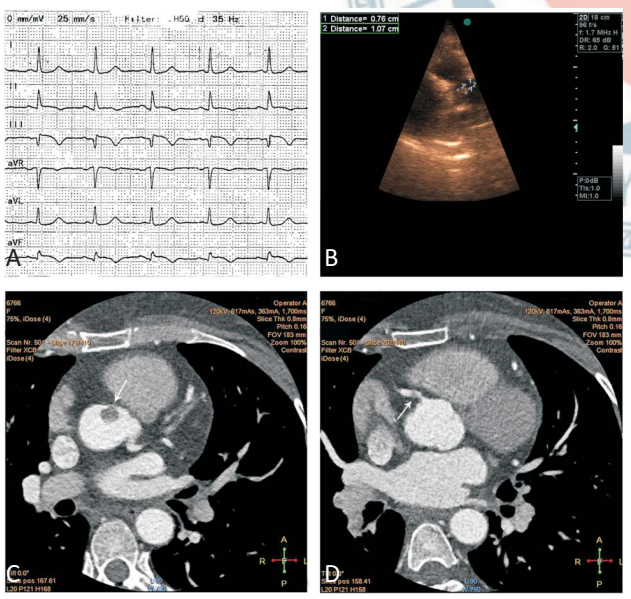

Figure 1. *ECG: show acute inferior MI (A), TTE: show echo dense mass in ascending aorta (B), CT angiography: show thrombus formation in ascending aorta (C) with extension to RCA (D). CT, computed tomography; ECG, electrocardiography; MI, myocardial infarction; TTE, transthoracic echocardiography; RCA, right coronary artery.*

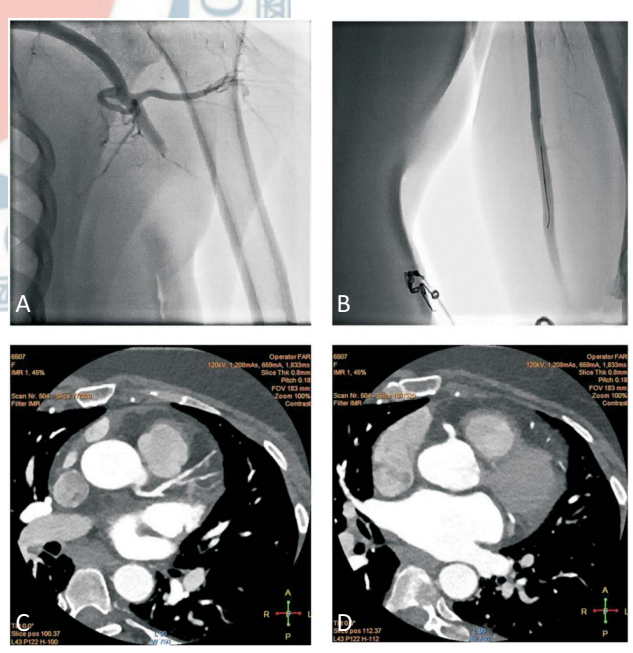

Figure 2. *Left upper limb angiography showed complete cut-off of the brachial artery flow at proximal part (A); and re-establishing blood flow after thrombectomy and direct fibrinolytic therapy (B). Control CT angiography shows no evidence of thrombosis in ascending aorta and RCA (C, D). CT, computed tomography; RCA, right coronary artery.*

cause of extensive clots in root of aorta and left brachial artery, some additional lab data was requested (inclusive of C3/C4/CH50/anti dsDNA/Anti cardiolipin Ab/Anti CCP/RA factor) with rheumatology service consult; that all of them were in normal range. Once stabilized on warfarin therapy (INR 2.8), the patient was discharged home.

Hereditary Hypercoagulability Panel (including factor V leiden, prothrombin 20210A, antithrombin III, protein C and S) was checked after 6 weeks that all of them were also normal.

DISCUSSION

The thrombose formation in normal ascending aorta is an uncommon and life-threatening condition for high risk thromboembolic events.[5] The precise mechanism is not yet fully understood, but its occurrence is low due to high blood flow and sheer stress of aorta. In most cases, we should considering to underlying malignant disease, acquired and hereditary hypercoagulable disorders, primary endothelial disorders or even iatrogenic causes.[5] Our case just had one risk factor inclusive chronic consumption of oral contraceptive pills (LD type) which is associated with increase in coagulation status.

Clinical presentation has a wide spectrum from asymptomatic to symptoms related to coronary, cerebral, peripheral, or visceral embolization; although many cases of aortic thrombosis are diagnosed after an embolic event usually involving the extremities.

There are various methods for the management of aortic thrombus and its complications including anticoagulant therapy, systemic (IV) thrombolytic therapy, local thrombolytic therapy, thrombectomy and surgical resection.[6-8] Currently no evidence exists that which one method is provides the better result and we cannot define a stepwise approach for all the patients.

Surely, the first therapeutic step is anticoagulation therapy that in some studies it is known as a preferred treatment.[9] In this case presentation we encountered with two critical events; AMI and acute symptomatic limb ischemia. In previous case reports with MI alone the recommended options were anticoagulation and when this measure fails, thrombectomy.[2] Okoronkwo et al. in a case of ascending and aortic arch thrombus which cause of MI and lower extremity ischemia reported successfully resolution of the aorta thrombus with medically treatment via systemic anticoagulation.[6]

In this case, there were two major events, and we had to make a conscious decision for both of them. The patient's hemodynamic was stable, chest pain severity was less than arm pain, area of infarction was inferior and primary PCI was accompanied by high risk of systemic embolization. So, given the severity of the limb ischemia we did act on the basis of Inter-Society Consensus for the Management of Peripheral arterial Disease (TASC II) and classified the limb ischemia after the IV heparin therapy.[4]

LEARNING POINTS

- Ascending aorta thrombosis with concurrent MI and upper limb ischemia is very rare but it would be in mind.
- There are various therapeutic strategies for ascending aorta thrombose that it should be decided on the basis of the patient's concurrent clinical conditions.
- Consideration to risk factors of thrombus formation are necessary to prevent recurrence of events.

CONFLICTS OF INTEREST

All authors declare no conflicts of interest.

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