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Upper Extremity Ischemia Complicating Ulcerative Plaque in Descending Aorta

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ABSTRACT

A 54-year-old female patient was presented with upper extremity ischemia. Further investigation revealed ulcerated atherosclerosis plaque in aorta with intramural hematoma and clot formation. The subject underwent a successful resection of mass with the assistance of cardiopulmonary bypass and total circulatory arrest.

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Introduction

In intramural hematoma atherosclerotic plaque of the descending aorta is a common condition, leading to peripheral embolization and ischemia, especially when accompanied with other risk factors (1). Ulceration is caused by advanced atherosclerosis and represents potential thrombosis and consequent embolic events (2). In this report, we presented a case with upper extremity ischemia caused by ulcerous plaque and emboli.

Case Presentation

We presented a 47-year-old female patient with possible symptoms of upper extremity ischemia. The patient was suffering from pain in the left arm, as well as pallor and paresthesia in the past 24 hours. Physical examination of extremities demonstrated the absence of radial and ulnar artery pulses and indicated the capillary refill time (CRT) to be more than three seconds. The patient was first admitted to vascular surgery department, followed by the administration of heparin (3 mg/kg). Afterwards, she was transferred to vascular surgery operating room and embolectomy of brachial artery was successfully accomplished. Following that, intact pulses in all extremities and non-focal neurologic signs were observed in our

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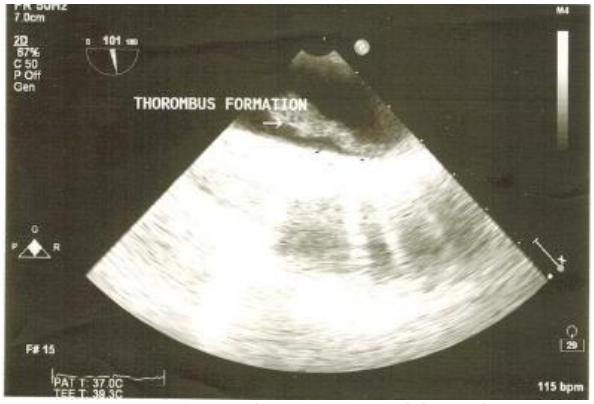

Figure 1. Presence of a mass lesion in proximal descending aorta, located in proximity of PDA and after the left subclavian artery

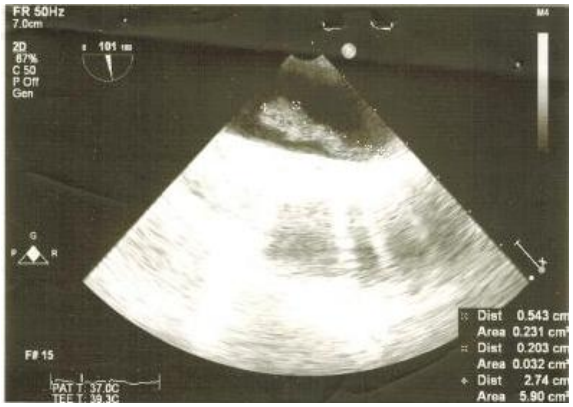

Figure 2.Another picture of thrombosis in descending aorta.

Partial, as well as reversed symptoms of arm ischemia.

Transesophageal echocardiography (TEE) for detecting the sources of embolism, that of indicated no clot or mass lesion in the left atrium and ventricle. Similarly, no lesion was found in the right-sided cardiac chamber. Moreover, there were no abnormalities, such as atrial septal defect (ASD) or patent foramen ovale (PFO) in the subject. Further investigation revealed that there was a mass lesion in proximal descending aorta, located in proximity of posterior descending aorta (PDA) and after the left subclavian artery (Figure 1).

After the patient was admitted to cardiac surgery department, the mass was removed from the aorta to prevent further embolic complications. However, there were controversial issues in optimal management due to the importance of lesion position. After the sternotomy and opening of pericardium, heparin was locally administered and cardiopulmonary bypass was initiated. . The subject was prepared for total circulatory arrest (TCA), and deep hypothermia work-up (18˚c) was performed after aortic cross-clamp and cardiac arrest. After reaching the target temperature of 18˚c (40 minutes), anesthetic management for TCA was performed, followed by the initiation of circulatory arrest. At the next stage, complete dissection of proximal descending aorta and left subclavian artery was detected, resulting in the opening of the descending aorta and removing the observed clot (about 2*2 cm). The clot was extended from PDA to proximal pulmonary artery. Ulcerous plaque was resected and aortic valve was repaired with suture. Following that, cardiopulmonary bypass was restarted and re-warming was initiated at a steady temperature. In the next stage, weaning from cardiopulmonary bypass was successfully managed after reaching 35.5˚c. The patient was fully recovered and discharged from hospital six days later in good clinical condition. Furthermore, early and late follow-ups marked no significant neurologic complications.

Discussion

One of the major sources of embolic complications is cardiac chamber or major arteries (3). Ulcerated atherosclerotic plaque in aorta refers to a penetrating ulcerative atherosclerotic lesion, usually occurring in ascending or proximal descending aorta. Defects in the internal elastic lamina (IEL) into the media layer lead to the formation of intramural hematoma in the aorta, causing aortic intraluminal mass lesions and clots (4-5).

Ulcerated plaque is detected in aorta as acute pain in chest wall or upper and lower back pain. However, some complications might be resulted from the formation of intraluminal clots, such as distal embolization, which affects the central nervous system, visceral, as well as upper and lower extremity ischemia (6-8). Other rare complications of aortic ulcerated plaque include aortic rupture, aneurysm, and the formation of pseudoaneurysm (8).

Several studies have suggested that imaging modalities could be used to detect aortic ulcerated plaque; such examples are TEE, computed tomography (CT), and diffusion weighted MRI (DWI-MRI) (8-9). Accuracy of TEE is an important factor in the diagnosis of plaque ulceration with intral-uminal hematoma. However, it should be noted that echocardiography is an operator dependent modality and physicians should be aware of the presence of intimal tear and false lumen, which usually refers to acute aortic dissection (7, 10).

In this case, no clot or mass lesion was observed in the left atrium and ventricle via TEE. Similarly, no mass lesion was detected in the right-sided cardiac chamber. However, further evaluation revealed a mass lesion in the proximal descending aorta, located in proximity of PDA and after the left subclavian artery.

In case of an episode of embolism, the embolic agents must be identified to correct and prevent

the incidence of further venous thromboembolism. According to the literature, the best controversial treatment of asymptomatic ulcerated aortic plaque is conservative management with anticoagulant or antiplatelet agents, alongside with a close follow-up. In addition, it was reported that the administration of high-dose statins might have an effect on atherosclerosis initiated by anti-inflammatory processes (11). Nevertheless, it is suggested that the underlying causes be surgically modified and corrected in complicated and symptomatic lesions (12-14).

Conclusion

According to the results, any ulcerated atherosclerotic plaque in aorta must be resected since it is a nidus for thrombus formation and thromboembolic events.

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Conflict of Interest

The authors declare no conflict of interest.

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