

Microembolization from an Abdominal Aortic Aneurysm after Thoracic Aortic Replacement

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A 66-year-old man with thoracic and abdominal aortic aneurysm suffered from microembolism in the lower extremities after total arch replacement. He presented with livedo reticularis with palpable peripheral pulses, and the serum creatinine kinase level elevated up to 7,695. The abdominal aortic aneurysm, but not the thoracic aorta, was the origin of this complication. The morphological change of thrombus in the abdominal aorta detected by ultrasonography was the key to the diagnosis. Graft replacement of the abdominal aorta finally resolved his problem. (Ann Thorac Cardiovasc Surg 2008; 14: 126–128)

Key words: microembolism, abdominal aortic aneurysm, ultrasonography

Introduction

Thrombus in the aortic aneurysm is a rare cause of embolization to the lower extremities. We experienced a rare case of microembolization from an abdominal aortic aneurysm after total arch replacement, and the morphological change of the thrombus detected by ultrasonography was the key to the diagnosis.

Case

The patient was a 66-year-old man who for six months had complained of hoarseness. An enhanced computed tomography (CT) scan revealed a thoracic aortic aneurysm of 84 mm extending from the ascending aorta to the proximal descending aorta and an infrarenal abdominal aortic aneurysm of 64 mm. The luminal surface of the entire aortic wall was covered with shaggy atheroma. He had chronic obstructive pulmonary disease (COPD) and an operative history of gingival carcinoma. The FEV1.0% (ratio of forced expiratory flow volume of one second

and total forced expiratory flow volume) and the PO₂ in room air was 49.5% and 61.5 mmHg, respectively. Institutional approval of this report was obtained, and the patient gave informed consent for serving as a subject.

Because of his poor respiratory function, we planned a staged operation; as the first stage operation, he underwent total arch replacement with an elephant trunk (ET). As arterial inflow sites, bilateral axillary arteries were used to establish antegrade perfusion. The immediate postoperative course was uneventful, and he was extubated on the next morning. He had no clinical symptoms, and the serum creatinine kinase (CK) level was normal.

On the second operative day, he presented with purple color spots and tenderness in his right thigh, and the distal arteries were quite palpable. The livedo reticularis gradually spread over the entire right lower limb and also to the left lower limb (Fig. 1). On the third postoperative day, the CK level elevated to 7,695, and he was unable to move his right leg against gravity. We considered that this was caused by a microembolization originating from the descending thoracic aorta by a flapping motion of the ET graft because there was a large amount of mural thrombus in the proximal descending aorta. We decided to perform an emergent operation on the descending thoracic aorta.

Through the left thoracotomy, we performed a proximal descending aortic replacement, using distal aortic perfusion through the left femoral artery. Immediately after the second operation, we started continuous

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hemodialofiltration. The CK level dropped to 3,795 by the next day, but it again went up to 6,305, and the purple color spots continued to spread (Fig. 2). He presented no abdominal symptoms, and severe acidemia was not present.

The abdominal ultrasonography was performed on the second day after the descending aortic operation. It revealed that the mural thrombus of the abdominal aortic aneurysm was separated and had become mobile, and that blood was passing through it. These findings were not present in the preoperative enhanced CT scan (Fig. 3). Together with the absence of apparent abdominal organ ischemia, we considered that the abdominal aortic aneurysm was the origin of microembolization to the lower extremities. Therefore we decided to perform an emergent replacement of the abdominal aortic aneurysm on the sixth day after the first operation. After that operation, the color change of the patient's legs gradually regressed, and the CK level steadily went down. The paralysis of the right lower limb also disappeared. He was extubated two weeks after the last operation, and was discharged unaffected two weeks later.

Discussion

We experienced a rare case of microembolism to the lower extremities from an abdominal aortic aneurysm after total arch replacement. At first, we considered that the embolic source was the thoracic aorta, which could be scattered by the ET graft inserted just proximal to the descending aorta with a large amount of mural thrombus. However, the second operation for the descending thoracic aorta was not effective to stop the embolic process. The remote infrarenal abdominal aneurysm was the source of embolization. Its resection finally resolved the problem.

It is possible that the use of heparin at the first operation was the trigger of the microembolism from the abdominal aneurysm. Bols et al. reported five cases of blue toe syndrome because of atheromatous embolization precipitated by oral anticoagulant.¹⁾

Shaggy aorta syndrome that has an irregularly shaped inner surface of the aortic wall is one of the causes of multiple cholesterol emboli.^{2,3)} It is characterized by extensive atheromatous disease with diffuse ulcers associated with soft, loosely held debris and a paucity of actual thrombus. Patients of this syndrome with suprarenal lesions and visceral emboli have a high morbidity. If used, the cessation of anticoagulants is recommended. Because



Fig. 1. Purple color spots appeared in his legs after the first operation. He felt tenderness in his thigh.

the resection of the entire diseased aorta can be hazardous, the use of an axillo-femoral bypass with bilateral external iliac artery ligations has been reported for the distal emboli.³⁾ Although the entire thoracic aorta of this case was also shaggy, an embolization of the renal or mesenteric arteries was not evident. So we finally considered that the source was an abdominal aortic aneurysm together with the ultrasonography findings.

Clinically, an identification of the embolic sources is very difficult, which could delay definitive therapy. The most common origin responsible for symptomatic embolization is the aortoiliac segment.⁴⁾ Even a small lesion of penetrating ulceration of the aorta could be the cause of an embolic event.^{5,6)} The diagnosis of the embolic source is quite difficult in a patient like ours, one with multiple lesions. There are no blood or laboratory tests specific to atheroembolism. Eosinophilia has been noted on blood smears and in urine, which was not evident in our case,⁴⁾ in which the morphological change of the thrombus detected by the ultrasonography was the key to diagnosis.

In aortic operations, a change of coagulation system,

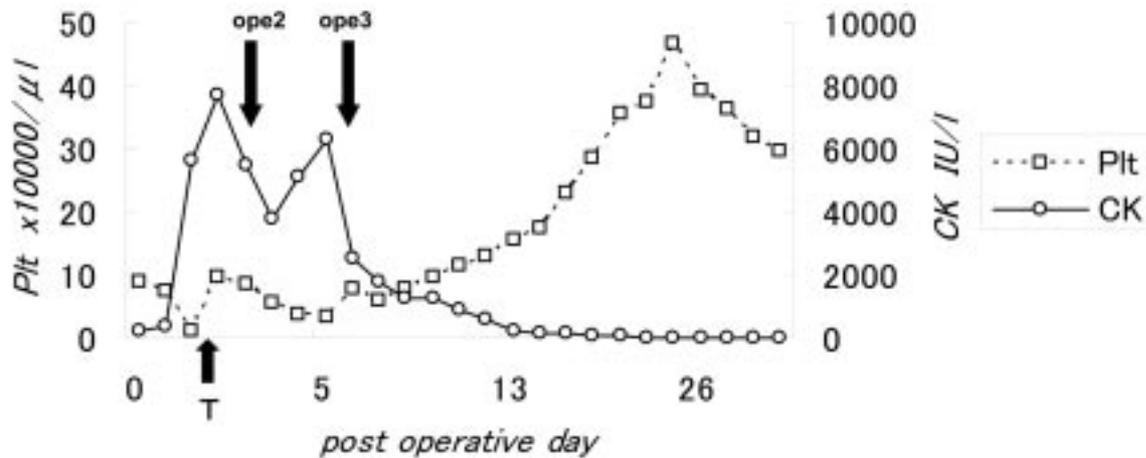


Fig. 2. Clinical course of the platelet count and the creatinine kinase. T, platelet transfusion of 20 units; ope 1, ope 2, and ope 3, the first, second, and third operations, respectively; Plt, platelet count; CK, creatinine kinase.

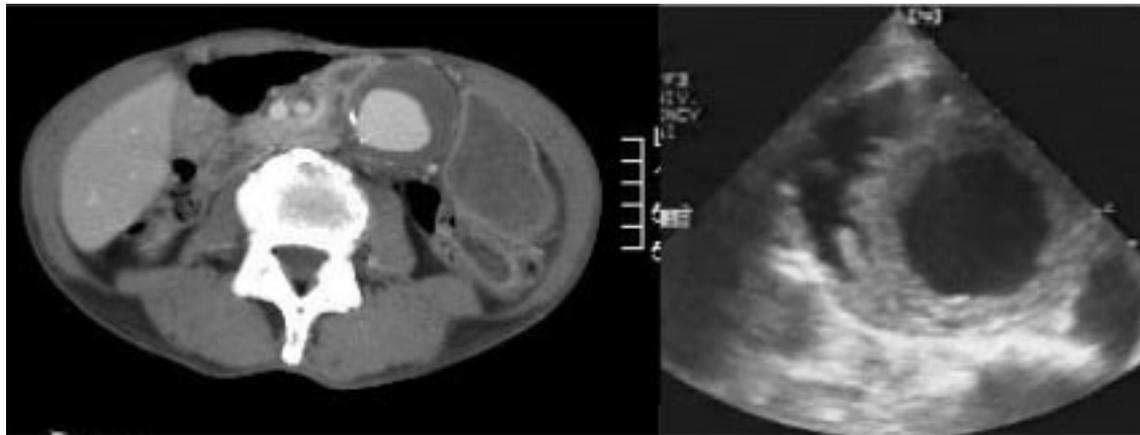


Fig. 3. Abdominal ultrasonography after the second operation revealed blood flow through the mural thrombus and mobile atheroma in the abdominal aortic aneurysm. This flow was not present in the preoperative CT scan. Left, preoperative CT scan; right, abdominal ultrasonography after the second operation.

the use of heparin, and the manipulation of other aneurysms can cause unstabilization of atheroma or clots, which may result in distal microembolization. We should be aware of such a condition, and if it occurs, an ultrasonographic evaluation of the aortic lumen may be helpful to make a diagnosis.

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