Chronic Occlusion of an Abdominal Aortic Aneurysm

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A 67-year-old woman with peripheral arterial occlusive disease in both lower extremities, secondary to an abdominal aortic aneurysm, developed chronic total occlusion of the abdominal aortic aneurysm during the 3-year follow-up period. She suffered from sudden onset of paraplegia 3 months after palliative axillo-bifemoral bypass grafting and died of pneumonia. The paraplegia was considered to have been caused by thrombosis of lumbar arteries that might have served as an important collateral pathway in the distal spinal cord, due to proximally propagated infrarenal aortic thrombosis. It is necessary to recognize that chronically thrombosed abdominal aortic aneurysm (AAA) still has a risk of causing serious complications with a high mortality rate, especially in cases treated medically or with palliative operations.

Key words: abdominal aortic aneurysm, thrombosis, claudication

INTRODUCTION

Patients with unruptured infrarenal types of abdominal aortic aneurysm (AAA) have a low mortality with treatment; however, several problems associated with AAAs remain unresolved. Chronic total occlusion of an AAA is a rare presentation, but it can cause serious complications with a high mortality rate.

CASE REPORT

A 67-year-old woman with complaints of progressive intermittent claudication was referred to our hospital for evaluation of an AAA with peripheral arterial occlusive disease in both lower extremities and renal dysfunction due to diabetes mellitus. She had a history of anteroseptal myocardial infarction that was treated by percutaneous coronary intervention. She had right hemiplegia due to a previous cerebral infarction. At the first medical examination, the AAA was palpable above the umbilicus. Peripheral arterial pulse examination showed weak pulsations in bilateral femoral arteries, but the popliteal, posterior tibial, and dorsalis pedis arteries were not palpable in both lower extremities. The ankle-brachial pressure index (ABPI) was 0.61 on the right side and 0.57 on the left side. A plain computed tomography (CT) showed an infrarenal type of AAA (maximum diameter, 48 mm), and aortography showed that the AAA was positively enhanced, with arterial occlusive disease below bilateral common iliac arteries (Fig. 1a and b). Though intermittent claudication had worsened during the 3-year follow-up period, she had refused surgical treatment until she could not walk even 10 meters. Anticoagulants and prostaglandins had been administered until she accepted surgical treatment. After admission, digital subtraction angiography revealed that the AAA was completely occluded with thrombus, which extended just below the level of bilateral renal arteries to both external iliac arteries (Fig. 2). The preoperative ABPI had deteriorated to 0.45 on the right side and 0.39 on the left side. An echocardiogram showed severely depressed left ventricular function with hypokinesis in the anteroseptal segment and an estimated ejection fraction of 38%. Coronary artery angiography showed total
Fig. 1  **a**: Plain computed tomography shows an abdominal aortic aneurysm, with a maximum diameter of 48 mm.  
**b**: Aortography shows that the aneurysmal cavity is positively enhanced, but the right common iliac artery is occluded at the proximal portion just before the bifurcation into the bilateral common iliac arteries, with distal collateral compensation. The left common iliac artery is patent, but multiple severe stenoses are seen in the left common iliac artery to the left external iliac artery. *right external iliac artery, **left external iliac artery.

Fig. 2 Preoperative digital subtraction angiography shows that the abdominal aortic aneurysm is entirely thrombosed from just below bilateral renal arteries to bilateral external iliac arteries (arrow). Collateral compensation is confirmed just below bilateral external iliac arteries. *right external iliac artery, **left external iliac artery.
occlusion in the proximal left anterior descending coronary artery segment with collateral compensation. Ultrasound carotid artery examination revealed diffuse stenoses in bilateral carotid arteries. Considering her severely impaired condition (almost wheelchair dependent, renal dysfunction (serum creatinine, 5.7 mg/dl), and chronic heart failure due to ischemic heart disease), less invasive treatment was chosen instead of radical surgery including replacement of the occluded AAA with a prosthesis. Hematological examination results were as follows: bleeding time (Duke), 1.5 minutes; fibrinogen, 212.8 mg/dl; activated partial thromboplastin time (APTT), 41.3 sec (control 35.0 sec); prothrombin time expressed as international normalized ratio (PT INR), 1.81; D-dimer, 23.5 μg/ml; and platelet count, 9.4 × 10^4/ml. These data suggested a consumptive coagulopathy state. Therefore, axillobifemoral bypass grafting was performed. Although temporary hemodialysis was required, the patient’s postoperative course was uneventful. The postoperative ABPI was improved to 0.82 on the right side and 0.73 on the left side. Three months after discharge, the patient was readmitted to our hospital because of sudden onset of paraplegia. Hematological examination results at the re-admission were: bleeding time (Duke), 2.3 minutes; fibrinogen, 244.0 mg/dl; APTT, 48.8 sec (control 35.0 sec); PT INR, 2.11; D-dimer, 73.0 μg/ml; and platelet count, 9.4 × 10^4/ml. Enhanced CT showed that the previously recognized stump thrombus extended upward to occlude bilateral renal arteries. However, there was no sign of aneurysmal rupture, and the patency of the prosthesis was confirmed by enhanced CT and arterial pulse examination. Her renal function deteriorated rapidly. Since she refused renal artery revascularization, regular hemodialysis was required. Her paraplegia was treated medically, but she died of pneumonia 7 months after developing paraplegia. Permission for autopsy could not be obtained.

**Discussion**

In the present case, a palliative operation was performed because of the generally impaired status of the patient. However, shortly after the operation, she developed sudden onset of paraplegia and rapid deterioration of renal function. These findings suggested that rapid proximal thrombus propagation had occurred, leading to renal and lumbar arterial occlusions. Lumbar arteries of the patient were considered to have served as an important collateral pathway to the distal spinal cord. The proximal extension of thrombus was confirmed by the enhanced CT at re-admission. Chronic total occlusion of an AAA is a relatively rare presentation; its reported incidence is 0.6%–2.8%. This presentation has several typical clinical findings including progressive intermittent claudication. Factors that are considered to favor complete aneurysmal occlusion tend to be small aneurysms less than 50 mm in diameter, which are often associated with peripheral arterial occlusive disease, embolisms caused by cardiac embolism, thromboembolisms due to dislodgement of intramural thrombosis, and cardiogenic hypotension or low-flow state. However, operative procedures are known to influence the hemodynamic outcome and patency rate. Aortic reconstruction is superior to axillobifemoral bypass. Late complications should be anticipated following palliative operations. Chronically thrombosed AAA still has a risk of rupture and proximal extension of the infrarenal thrombosis, which can lead to renal, distal spinal cord, lower extremities and visceral ischemia. On the basis of the anatomy of the blood supply to the distal spinal cord, the lumbar, iliolumbar, internal iliac, and lateral sacral arteries play an important role in perfusion, with anastomoses involving the spinal arteries at the level of the conus medullaris. These arteries may be critically important when the greater radicular artery is compromised due to aorto-iliac occlusive disease. In such patients, these arteries may serve as an important collateral pathway to the distal spinal cord. Therefore, abrupt disruption of these arteries due to thrombosis can easily lead to irreversible ischemia of the distal spinal cord. In the present case, the occlusion had already extended to just between the level of the renal arteries and bilateral external iliac arteries, including complete occlusion of the bilateral internal iliac arteries. Under such circumstances, the lumbar arteries may serve as the only collateral pathway to the distal spinal cord. Therefore, paraplegia was thought to have developed when the lumbar arteries were occluded due to proximal extension of thrombus. Paraplegia seems to be more frequent in patients with AAA occlusion than in patients with aorto-iliac occlusive disease. Several factors would affect this phenomenon. Aorto-iliac occlusive disease is a common entity that is slowly progressive, permitting the development of collateral circulation. Moreover, coagulation abnormalities
may play an important role. Parry et al. reported that development of thrombosis is considered to involve the interaction between the process of inflammation and coagulation, which remains pathological in patients with AAA but not in patients with peripheral arterial occlusive disease. These conditions seem to result in patients with AAA having a hypercoagulable state. Moreover, continued slow and turbulent blood flow in the aneurysm also may predispose patients to the develop thrombosis. In impaired patients, as in the present case, endovascular or hybrid repair of an AAA with widespread severe peripheral arterial occlusive disease should be considered in the early stage.

**CONCLUSION**

A patient with peripheral arterial occlusive disease secondary to an AAA developed chronic infrarenal aortic occlusion during follow-up, which resulted in sudden onset paraplegia and acute exacerbation of renal function after palliative surgery. A possible mechanism to explain these symptoms was suprarenal extension of the aortic thrombus. Elective removal of the thrombosed AAA and anatomical reconstruction are required to prevent this complication in patients who can tolerate such procedures.

**REFERENCES**