Case Report

Surgical Treatment of Aortic Coarctation Associated with Multi-vessel Brachiocephalic Involvement in Takayasu's Arteritis

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In Takayasu's arteritis (TA), both atypical coarctation (CO) and brachiocephalic involvement are common features that occasionally require operative correction. A combination of these abnormalities could duplicate underlying illness in patients, posing an increased risk of operative morbidity. We present, herein, two TA patients in which hypertensive heart disease secondary to CO was surgically corrected. Both patients had multi-vessel brachiocephalic disease. One patient who showed occlusion of all brachiocephalic arteries underwent aorto-aortic bypass, while another with two-vessel lesion underwent axillo-bifemoral bypass grafting. Subclavian reconstruction was supplemental to each procedure, resulting in relief of neurologic stigmata. Strategies to avoid intraoperative cerebral ischemia played an important role in the surgical repair of such TA-related extensive vascular lesions. (Ann Thorac Cardiovasc Surg 2003; 9: 202–5)

Key words: Takayasu's arteritis, aortic coarctation, brachiocephalic involvement

Introduction

Takayasu's arteritis (TA) remains a poorly understood diffuse inflammatory disease of the arterial system.[1,2] Vascular insufficiency secondary to occlusive disease can threaten multiple organ systems and strongly affect prognosis. Once a patient presents with uncontrolled hypertension or neurologic symptoms, surgical treatment is the sole measure to prevent greater risk to life. However, surgical repair for extensive arteriopathy requires an ingenious design to avoid morbidity as to respond to the complexities of their pathophysiology.

We successfully treated two patients who had had critical manifestations due to atypical coarctation (CO) associated with multi-vessel brachiocephalic involvement. This report focuses on the revascularization methods used and on prevention of perioperative cerebral complications.

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Case Report

Patient 1

A 61-year old male complaining of severe dyspnea was admitted to the intensive care unit of our hospital. He had a 30-year history of hypertension, and had left hemiparesis resulting from a previous cerebral infarction. A physical examination revealed the absence of a brachial and radial pulse in both upper limbs. The blood pressure in the lower limb was 82/50 mmHg. The chest X-ray revealed pulmonary edema. He was intubated for mechanical ventilation and the transesophageal echocardiography demonstrated left ventricular hypertrophy and CO in the proximal descending aorta. Magnetic resonance imaging (MRI) showed aortic lesions associated with occlusion of all arch branches (Fig. 1). The right subclavian artery was visualized, whereas the left was not. A diagnosis of TA was confirmed on the basis of these findings. The cerebral circulation was supplied by the bilateral vertebral

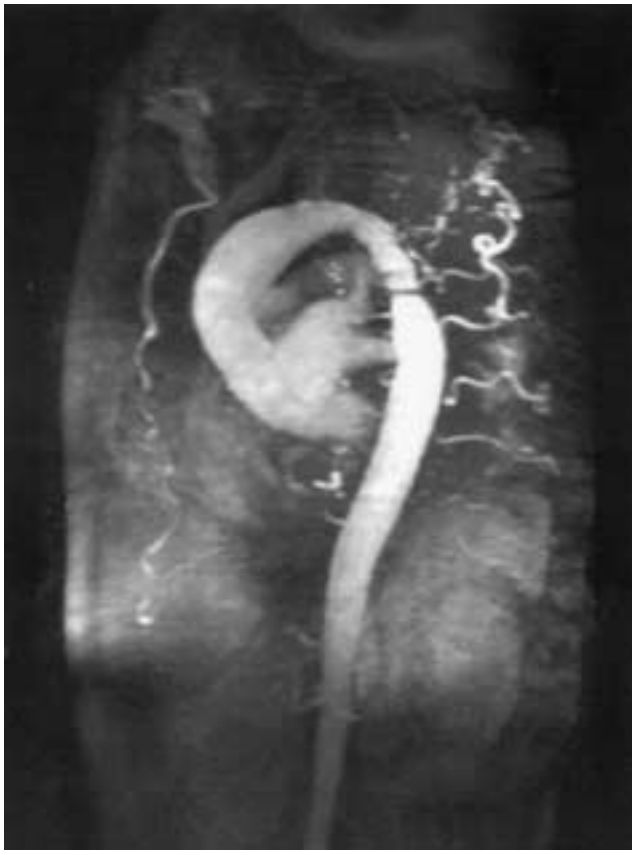

Fig. 1. Preoperative MRI in Patient 1 revealed severe coarctation in the proximal descending aorta associated with occlusion of all arch vessels.

arteries which were perfused via collateral vessels. The patient underwent surgery three months after admission. Ascending-abdominal aortic bypass, not aortic repair via a left lateral thoracotomy, was indicated for this coarctate lesion because right axillary revascularization was simultaneously scheduled. During the operation, regional oxygen saturation (rSO_2) was measured continuously by near-infrared spectroscopy (NIRS) to monitor cerebral perfusion, as the risk of brain damage was potentially high. A 16 mm tube graft was anastomosed to the infrarenal aorta and an 8 mm PTFE graft was anastomosed to the right axillary artery. The blood pressure gradient through the coarctate lesion was disclosed for the first time in an intraoperative study, and was revealed to be 80 mmHg. When a partial clamp was placed on the ascending aorta for testing, the blood pressure there increased from 160 to 250 mmHg. Therefore, we decided to use cardiopulmonary bypass (CPB) to decompress the heart during anastomosis on the proximal aorta. The CPB was instituted with simultaneous perfusion via tube grafts on the abdominal aorta and the right axillary artery. The value of the rSO_2 decreased on initiating circulatory assistance, and was maintained above 60% throughout CPB. After weaning from CPB, the right axillary artery was reconstructed by connecting the 8 mm graft to the aortic tube. There were no new cerebral complications postoperatively. After surgery, the systolic blood pressure of the right upper limb and the lower limb were equal to 144 mmHg.

Currently, at five years after surgery, the patient has no symptoms of heart failure with normal blood pressure.

Patient 2

A 58 year-old female with a long medical history of TA presented to our institution from another hospital. The systolic blood pressure in the right upper limb was 210 mmHg, and in the left upper and lower limbs were 160 and 130 mmHg, respectively. The hypertension was not controlled with several anti-hypertensive drugs. Her performance status was classified as New York Heart Association (NYHA) grade III. She had experienced fainting attacks several times. An echocardiogram showed severe left ventricular hypertrophy. Angiography and computed tomography (CT) scanning revealed a coarctate lesion in the thoracoabdominal aorta and occlusion of the left common carotid and subclavian arteries (Fig. 2). The minimum inner diameter in this aortic lesion was 8 mm. The aorta was calcified circumferentially throughout the infrarenal portion. Axillo-femoral (A-F) bypass was indicated because distal aortic anastomosis was hardly possible. A 10×8 mm Y-shaped conduit was employed for the right A-F bypass grafting. The left axillary artery was revascularized via a 6 mm conduit originating from the A-F conduit. Schemata of the operative design in these two patients are shown in Fig. 3. Postoperatively, the patient's blood pressure was normalized and the blood pressure gradients between the extremities disappeared. Currently, at six months after surgery, her performance status has improved to NYHA grade II and faintness has not recurred.

Discussion

Atypical CO is a rare cause of correctable hypertension in adults.[1)] Either surgical treatment or endovascular stenting has been shown to correct hemodynamic disorders due to this type of aortic lesion.[2,3)] Bypass grafting utilizing aorto-aortic (A-A) or A-F conduits have been applied in the treatment of atypical CO. A-A bypass is

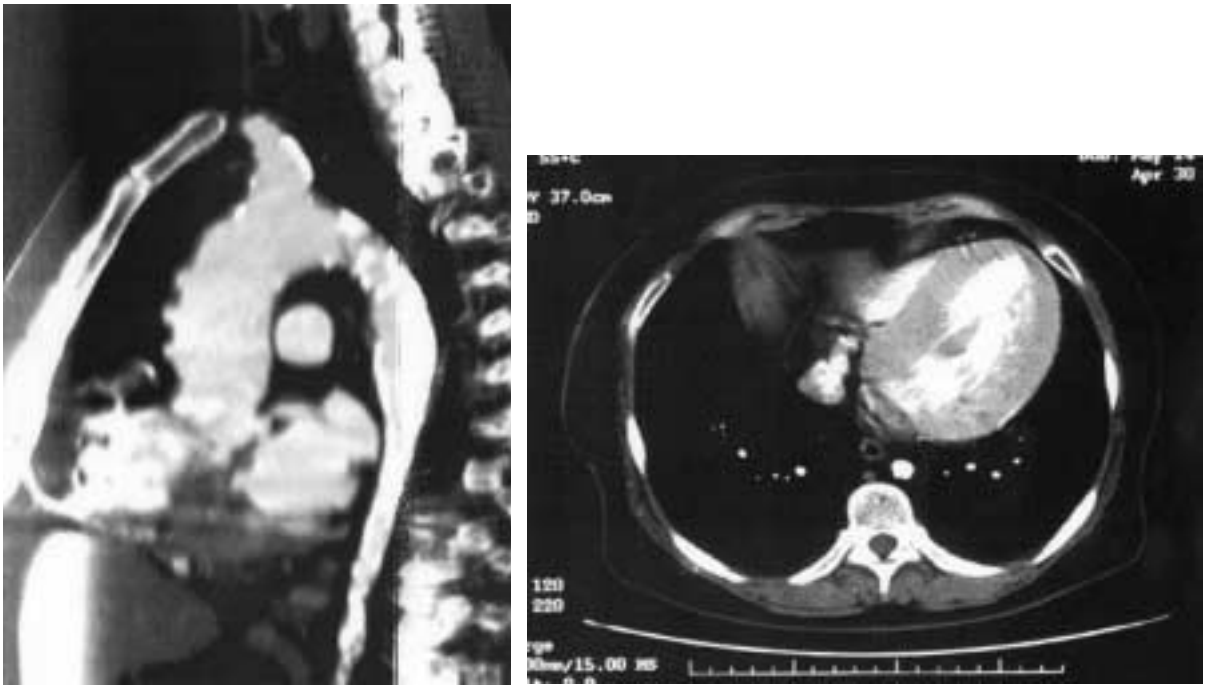

Fig. 2. Preoperative CT findings in Patient 2. A: Left common carotid and subclavian arteries were occluded. B: Coarctate lesion in the descending aorta and left ventricular hypertrophy were revealed.

A | **B**

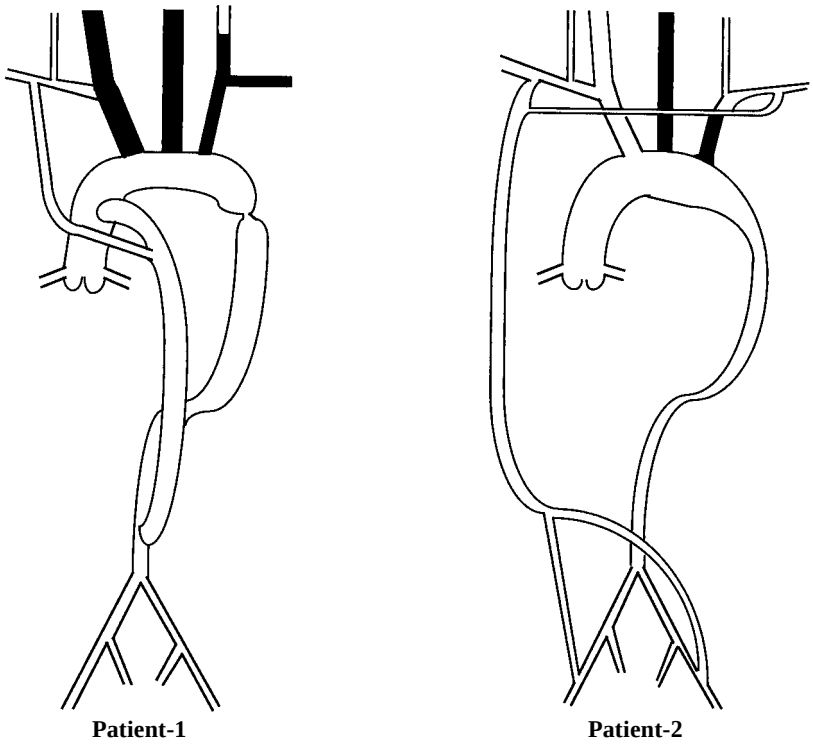

Fig. 3. Schemata of revascularization design in the present patients.

the most promising efficient method to reduce cardiac afterload. Such aortic reconstruction, however, would cause a greater risk of operative mortality. On the other hand, the potential of the A-F bypass to modify this hemodynamic disorder is less reliable because of the inability to employ a conduit with a diameter greater than 10 mm in this plane. Endovascular stenting into atypical CO has been recently introduced as a less-invasive treat-

ment option in one case report.[1] This modality may be more potent than an A-F bypass in selected cases. However, a certain amount of endoluminal stress to the severely diseased aorta can cause definitive mural injury. The coarctate lesions involving the entire thoraco-abdominal region would not be indicated for the endovascular method although this is a typical feature in TA-related CO.

Carotid artery stenosis greater than 50% is a strong risk factor of stroke after cardiac surgery using CPB.[4,5] The incidence of cerebral complication is especially high among patients associated with bilateral carotid lesions. The brachiocephalic trunk is the most frequent involvement site in TA, and multi-vessel lesions are common. Therefore, problems associated with intraoperative cerebral ischemia are inevitable among TA candidates for aortic surgery. Aortic reconstruction necessitated the use of CPB in Patient 1 in whom cerebral circulation was supplied only by collateral flow. Vertebral perfusion via CPB avoided malperfusion of the entire brain. Monitoring of rSO_2 by means of NIRS was very useful to determine pump flow. In Patient 2, application of an A-F conduit dispensed with the use of CPB. Needless to say, the A-F method is preferable in view of reducing morbidity. However, this method necessitates careful monitoring for the occurrence and progression of vasculopathy in the inflow artery.

Surgery for brachiocephalic disease is rarely necessary and is limited to the uncommon patient with neurologic symptoms or patients with severe upper extremity claudication.[2] Fainting attacks threatened both of the present cases. Because any carotid vessel distal to the occlusion could not be visualized through angiographic evaluations, revascularization of one subclavian artery was performed resulting in neurological recovery of both patients. Augmented blood flow through one vertebral artery may have ameliorated perfusion in the affected cerebral region. Revascularizing of carotid vessels may not always be necessary to eliminate neurologic symptoms, even in cases with severe brachiocephalic disease.

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