Axillofemoral Bypass Markedly Improved Acute Decompensated Heart Failure and Kidney Injury in a Patient with Severely Calcified Stenosis of Thoracoabdominal Aorta (Atypical Aortic Coarctation)

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Summary
Atypical aortic coarctation (AAC) has been reported to occur anywhere along the aorta, except for the ascending aorta. The associated symptoms include hypotension in the lower half of the body, secondary hypertension in the upper half of the body, and heart failure. Here we present an 80-year-old Asian woman complaining of progressive exertional dyspnea. She was diagnosed with acute decompensated heart failure and kidney injury due to severely calcified stenosis of the thoracoabdominal aorta, the so called AAC. She received hemodiafiltration, and pulmonary congestion improved in part. Generally, surgical treatments are quite invasive in elderly patients. Endovascular stent graft placement is less invasive, however, fracture and rupture should be considered at severely calcified lesions like this case. Therefore, we selected extra-anatomical axillofemoral bypass. Her recovery after the surgery was remarkable. In a few days, she became free from hemodiafiltration, intravenous diuretics, and oxygen administration. We thought the contributive factors are the increase in kidney blood flow and the correction of afterload mismatch. The decrease in pulse pressure may reflect the reduction in systemic arterial compliance by axillofemoral bypass. The operative mortality of axillofemoral bypass was reported to be acceptable, although the patency of the axillofemoral bypass graft was not high enough. In conclusion, axillofemoral bypass is effective and feasible for elderly patients with acute decompensated heart failure and kidney injury due to AAC.

Key words: Takayasu’s arteritis, Secondary hypertension, Renovascular hypertension, Hypertensive heart disease, Ischemic nephropathy

Congenital aortic coarctation is typically located either proximal or distal to the ligamentum arteriosum in the aortic isthmus. In contrast, atypical aortic coarctation (AAC) has been reported to occur anywhere along the aorta except for the ascending aorta.1) AAC is a condition associated with Takayasu’s arteritis (TA), congenital hypoplasia, and atherosclerosis. The associated symptoms include hypotension in the lower half of the body (intermittent claudication, ischemic nephropathy, intestinal ischemia), secondary hypertension in the upper half of the body and heart failure due to afterload mismatch. Aortoaoartic bypass has been performed especially in younger patients of TA. However, aortoaoartic bypass is an invasive procedure for elderly patients, because aortoaoartic bypass requires an aortic clamp, thoracotomy, and/or celiotomy. Here, we report a senile patient with AAC who recovered from acute decompensated heart failure and kidney injury after axillofemoral bypass with insight on contributive factors and a review on other treatment options.

Case Report
An 80-year-old Asian woman was hospitalized because of progressive exertional dyspnea. In her 40’s, the medical checkup had found the hypertension and the difference of blood pressure between both arms (right 162/92 mmHg, left 136/96 mmHg). Although antihypertensive medication had started later, it had been resistant (150-180/70-90 mmHg) in spite of taking 5 drugs. Since two years before admission, she had presented exertional dyspnea in 6 months before admission, exertional dyspnea had emerged again and gradually got worse, even with 9 kinds
of antihypertensive medication with diuretics. Finally, she presented orthopaenia and was hospitalized.

On admission, her heart rate was 81 bpm and blood pressure was 154/63 mmHg (right arm) and 114/67 mmHg (left arm). Her blood oxygen saturation was 94% with room air. Her jugular vein showed distention. Auscultation revealed diffuse coarse crackles in her lung and vascular bruit in her anterior chest and back. Marked pitting edema was in her both legs. Pulsations in both femoral arteries were weak and those in both dorsal pedis arteries were not palpable. An ECG showed ST segment depression in II III F V5-6, and a chest x-ray showed cardiomegaly (CTR 55%), mild pleural effusion, pulmonary congestion, and prominent calcification of thoracoabdominal aorta (Figure 1A). A blood sample test revealed microcytic anemia (Hb 6.7 g/dL, rectal ulcer was observed later), elevated serum creatinine (2.69 mg/dL), and serum brain natriuretic peptide (BNP 442.1 pg/mL). C-reactive protein was normal (0.27 mg/dL), but erythrocyte sedimentation rate was a little high (42 mm/hour). A transthoracic echocardiogram showed normal left ventricular contraction (Dd 51 mm, Ds 32 mm, ejection fraction 67%) with mild concentric hypertrophy (interventricular septum 11 mm, posterior wall 11 mm), diastolic dysfunction and elevated left atrial pressure (mitral E 163 cm/s, A 65 cm/s, E/A 2.5, deceleration time 129 ms, septal E’ 6.8 cm/s, E/ E’ 24, right ventricular systolic pressure 62 mmHg). The ankle-brachial indices (ABI) in both legs were low (0.56/ 0.56). An abdominal ultrasound examination showed that right renal arterial blood flow was unmeasurable (left renal artery 58.1 cm/s). The plain computed tomography of her body revealed marked calcification of thoracoabdominal aorta, the orifice of right brachiocephalic artery, left subclavian artery, bilateral renal arteries, and coronary arteries (Figure 1D). The thoracoabdominal aorta showed long stenosis with severe calcification and the lumen diameter was 10 mm from the diaphragm level to the renal artery level (**) The orifice of renal arteries (arrowhead).

Figure 1. Chest x-ray on the admission day (A), the 3rd day (B) and the 31st day (C). They showed prominent calcification of thoracoabdominal aorta (black arrows). The plain computed tomography (D) also revealed marked calcification of thoracoabdominal aorta and the orifice of main branches (white arrows). Right kidney was atrophic (*). Curved planar reconstruction of the plain computed tomography of thoracoabdominal aorta (E) revealed that the lumen diameter was 10 mm from the diaphragm level to the renal artery level (**). The orifice of renal arteries (arrowhead).

On the 3rd day, she showed flash pulmonary edema
Diuretics were not effective and hemodiafiltration was started. Pulmonary congestion improved in part, but in this state, she would keep up hemodialysis and oxygen therapy. We discussed with surgeons and planned an axillofemoral bypass aiming at an increase in kidney blood flow and the correction of the afterload mismatch. The operation was performed on the 12th day. A so-called inverted Y graft was implanted from the right axillar artery to the bilateral femoral arteries. A few days after the operation, her condition improved markedly, then hemodiafiltration, intravenous diuretics, and oxygen administration were not necessary anymore. She was discharged with normal blood pressure, taking 3 hypertension medications with diuretics on the 31st day (Figure 1 C). Her pulse pressure significantly decreased after the operation (from 90 to 60 mmHg, Figure 2). ABI became almost normal (right 0.91, left 0.95). An abdominal ultrasound examination revealed an increase in velocity of the renal arterial blood flow in both sides (right renal artery 54.0 cm/s, left renal artery 96.6 cm/s), and retrograde blood flow in the bilateral femoral arteries from anastomosis sites directed to the abdominal aorta. Serum creatinine improved to 1.1 mg/dL, which was even better than two years ago (around 2 mg/dL). Plasma renin activity markedly reduced from 19 ng/mL/hour on the admission day to 0.6 ng/mL/hour after the operation. BNP also decreased to 147 pg/mL. The echocardiography showed no change in the left ventricular size (Dd 51 mm, Ds 30 mm, ejection fraction 72%), and decreased left atrial pressure (mitral E 98 cm/s, A 98 cm/s, E/A 1.0, deceleration time 284 ms, septal E’ 4.5 cm/s, E/E’ 22, right ventricular systolic pressure 32 mmHg). Colonoscopy after the recovery revealed the rectal ulcer, which resolved itself.

Discussion

There are several etiologies of AAC. It is reported that TA is complicated with lesions (stenosis or aneurysm) of the abdominal aorta in 28% and the renal artery in 13%. “Coral reef aorta” is the condition of focal rock-hard calcification located to the suprarenal aorta. “Mid aortic syndrome” is hypoplastic segment coarctation in the lower thoracic or upper abdominal aorta, complicated with neurofibromatosis or other conditions. In this case, it had the possibility that atherosclerosis was the cause because she had hypertension and was ex-smoker. Although she had no history of fever or vascular pain, the difference of blood pressure between both arms in her 40’s and the calcification in the first branch of the aorta, including the orifice of the right brachiocephalic and left subclavian arteries, indicated the other possibility that she had TA. She also suffered from bleeding from a rectal ulcer, which could be caused by intestinal ischemia. There are no reliable parameters reflecting the disease activity of TA. We should speculate the disease activity from symptoms, systemic inflammatory response and images. The fact that, although erythrocyte sedimentation rate was a little high, C-reactive protein was normal, the vessel lesions were calcified, no increased wall thickness, and edematous, and her age was over a peak age, inferred TA was not so active. We did not use corticosteroids and other immunosuppressive agents even after the operation.

We supposed there were two factors that contributed to the marked improvement of heart failure. The first factor was the increase in kidney blood flow. The ultrasound velocity of renal arteries rose retrogradely from the bypass graft outlets. The renal function (serum creatinine) im-
Several treatment options of AAC have been proposed (Table). End-to-end anastomosis and graft replacement have the risk of damaging main branches and aneurysm at anastomosis. Transaortic endarterectomy has been reported. Some cases were performed with laparoscopy.\(^6\) Aortoaoartic bypass is also considerable, but it has the possibility to hurt the collateral circulation around the stenosis site. They are not suitable for elderly patients and patients whose general statuses are not well because they require quite invasive approaches such as aortic clamp, thoracotomy and/or celiotomy. Endovascular stent graft placement is not very invasive,\(^5\) but, especially in TA patients, it was reported that surgical repair was associated with low mortality and morbidity compared with endovascular repair.\(^5\) In highly calcified lesions, underdilation, fracture, and rupture of stent grafts were expected. Therefore, we chose extra-anatomical axillofemoral bypass. This procedure is not very invasive and needs no stent graft. The operative mortality of the axillofemoral bypass was reported to be acceptable (4.9%),\(^9\) compared with the other report on aortoaoartic bypass (12.1%).\(^8\) On the other hand, it was proposed that the patency of axillofemoral bypass graft was not high enough. Primary graft patency in 3 years was 63% for axillofemoral bypass and 85% for aortofemoral bypass.\(^10\) The patency of the grafts depends on the condition of inflow artery, outflow artery, graft length, and diameter. In TA patients, the vascular lesions distribute mainly in the proximal portion of main branches. So, the condition of outflow artery could be preserved in most cases and the patency of the graft might be better than reported previously.

In conclusion, axillofemoral bypass is effective and feasible for elderly patients with acute decompensated heart failure and kidney injury due to AAC. Detailed clinical course and data indicated the improvement of kidney blood flow and afterload mismatch.

### Disclosures

**Conflict of interest:** The authors declare no conflicts of interest.

### References