Revascularization for controlling hypertension and improving cardiorenal failure in Leriche syndrome

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Abstract

Leriche syndrome usually occurs when atherosclerotic obstructions result in luminal narrowing of the abdominal aorta or iliac arteries and leads to thrombosis; it rarely causes heart or renal failure. We report the case of a 58-year-old Asian man with heart and renal failure as the dominant clinical manifestations of renovascular hypertension caused by Leriche syndrome. We performed an aorto-bifemoral bypass and unilateral renal artery stenting. Post-operative echocardiography showed improved cardiac function, with the left ventricular ejection fraction increasing from 30% before surgery to 54.2% after surgery. Moreover, his heart rate and blood pressure became stable, and his serum creatinine and brain natriuretic peptide levels decreased from 3.46 to 1.08 mg/dL and 685 to 4 pg/mL, respectively. Our case report shows that aorto-bifemoral bypass and unilateral renal artery stenting can effectively treat heart and renal failure resulting from renovascular hypertension caused by Leriche syndrome.

Keywords  Leriche syndrome; Renovascular hypertension; Heart failure; Renal failure; Aorto-bifemoral bypass; Renal artery stenting

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Introduction

Leriche syndrome, or aortoiliac occlusive disease, results from thrombotic occlusion of the abdominal aorta immediately above the bifurcation site. Characteristic symptoms include intermittent bilateral claudication with ischaemic pain, absent or diminished femoral pulses, pallor, leg coldness, fatigue, and inability to maintain penile erection; risk factors include hyperlipidaemia, hypertension, diabetes mellitus, and smoking. Physical examination findings include weak or absent femoral and distal pulse rates.

Pathologically, Leriche syndrome is primarily caused by luminal narrowing of the abdominal aorta and/or iliac arteries by atherosclerotic obstruction. It typically begins at the distal aorta or common iliac artery origins and gradually progresses proximally and distally; it rarely affects the visceral or renal arteries. According to Bergen and Trippel, aortic thrombosis progression involves one or both renal arteries in 3–15% of patients with distal aortic occlusion. During progression, the thrombosis may gradually encroach on the renal artery, causing increasingly severe hypertension and heart and renal failure. Atherosclerotic renal artery diseases are common causes of renovascular hypertension, which often leads to resistant hypertension.

We describe a case of Leriche syndrome with renal artery occlusion-induced resistant hypertension associated with cardiorenal failure caused by atherosclerotic renal disease and abdominal aorta occlusion. Revascularization therapy was performed to control hypertension and improve heart and renal function.

Case report

The patient provided informed consent and institutional approval was obtained.

A 58-year-old Asian male visited a hospital due to progressive exertional dyspnoea [New York Heart Association (NYHA) Class IV] and intermittent claudication; tests revealed hypertension (243/127 mmHg), heart failure, and renal failure. Anti-hypertensive medications (nifedipine, 80 mg/
day; azilsartan, 40 mg/day; bisoprolol, 5 mg/day; and
doxazosin, 4 mg/day) were administered but ineffective.
Plain computed tomography (CT) and abdominal ultrasound
revealed occlusion of the abdominal aorta below the superior
mesenteric artery (Figure 1A) and absence of bilateral renal
arterial blood flow. The left kidney was atrophic (70 mm
along the long axis), whereas the right kidney was not
(95 mm along the long axis).

The patient was transferred to our hospital for detailed ex-
amination and treatment. His heart rate, blood pressure, and
blood oxygen saturation in room air were 95 beats per
minute with sinus rhythm, 202/131 mmHg, and 94%, respec-
tively. Auscultation revealed diffuse coarse crackles in the
lungs and vascular bruits in the anterior chest and back;
marked pitting oedema was observed in both legs. Pulsations
in the femoral arteries were weak and not palpable in the
dorsalis pedis arteries. Chest radiography showed cardiomeg-
aly (cardiothoracic ratio (CTR), 55%), pleural effusion, and
flash pulmonary oedema (Figure 1B); electrocardiography
(ECG) showed left ventricular hypertrophy (Figure 1C). A
blood test revealed elevated serum creatinine (3.46 mg/dL)
and brain natriuretic peptide (BNP; 685 pg/mL) levels. Trans-
thoracic echocardiography showed low left ventricular
contraction (Dd, 53.2 mm; Ds, 47.8 mm; ejection fraction,
30%) with mild concentric hypertrophy (interventricular sep-
tum, 11 mm; posterior wall, 11 mm) and diastolic dysfunction
(E/A, 0.6; deceleration time, 166 ms; E/E', 9.5) (Figure 1D);
however, no findings indicated valvular disease, cardiomyo-
pathy, or ischaemic heart disease, and the ankle–brachial index
(ABI) for both legs was low (right, 0.58; left, 0.51) (Figure 1E).

Magnetic resonance angiography revealed occlusion of the
abdominal aorta below the right renal artery (Figure 2A), left
renal artery occlusion, and right renal artery stenosis with se-
vere ostial lesions associated with aortic atherosclerosis
(Figure 2B). Pre-operative contrast-enhanced CT and angiog-
raphy were not performed because the estimated glomerular
filtration rate was 13 mL/min.

The patient was diagnosed with decompensated heart fail-
ure and Stage 2 acute kidney injury—defined using the Kidney
Disease Improving Global Outcomes criteria—secondary to
Leriche syndrome. However, fluid overload was obvious; thus,
a diuretic was intravenously administered without consulting
heart and renal failure specialists, as they were absent. The
diuretic (80 mg/day furosemide) was effective, and the

Figure 1 Pre-operative examination findings. (A) Abdominal ultrasound examination showing occlusion of the abdominal aorta below the superior mesenteric artery. The abdominal aorta around the renal arteries was filled with a large number of clots (white arrow). Ao, aorta; SMA, superior mesenteric artery. (B) Chest radiography showing cardiomegaly (cardiothoracic ratio, 55%), pleural effusion, and flash pulmonary oedema. (C) Electrocardiogram showing left ventricular hypertrophy. (D) Transthoracic echocardiogram showing low left ventricular contraction (Dd, 53.2 mm; Ds, 47.8 mm; ejection fraction, 30%) with mild concentric hypertrophy (interventricular septum, 11 mm; posterior wall, 11 mm). (E) Blood pressure readings showing that the ankle–brachial index for both the legs was low (right, 0.58; left, 0.51).
Dyspnoea immediately improved from NYHA Class IV to III; however, heart and renal functions only partially improved. After consulting with interventionalists, an aorto-bifemoral bypass and left renal angioplasty were performed immediately after admission to increase blood flow in the left kidney and correct the afterload mismatch. The abdomen was explored via upper and lower median incisions, and the femoral arteries under the inguinal ligament were examined as potential anastomotic sites. The aorta was below the renal artery with no pulsation or marked arteriosclerotic changes. The abdominal aorta was explored, and the appropriate anastomotic site selected. Transperitoneal tunnels were created between the femoral and anastomotic sites, and the patient was heparinized. Heparin was administered for an activated clotting time of 200–250 s during the operation.

As numerous blood clots were attached to the suprarenal abdominal aorta, an aortic clamp was applied above the celiac artery. An aortic incision was made at the renal artery level, and a thrombus was found in the lumen; when removed, no blood flow was observed in the renal arteries. After aortic clamping, aortic anastomosis using a 16 × 8-mm J-Graft (Japan Lifeline Inc., Tokyo, Japan) and bifemoral anastomosis were performed. Angiography revealed that the aorto-bifemoral bypass was patent, with total occlusion of the left renal artery and 90% stenosis of the right renal artery ostium (Figure 3A). Percutaneous transluminal renal angioplasty (PTRA) was performed using an Express Vascular LD 5 × 15-mm stent (Boston Scientific Corporation, Natick, MA, USA) implanted in the right renal artery (Figure 3B). PTRA in the left renal artery was technically difficult due to an unclear left renal artery ostium.

Several days post-operation, the patient’s condition markedly improved; intravenous diuretics and oxygen administration were not required. He was discharged on Day 11 with normal blood pressure and was only prescribed anti-hypertensive medication (2.5 mg/day bisoprolol). Post-operation contrast-enhanced CT showed a patent aorto-bifemoral bypass and right renal angioplasty and the collateral artery from the aorta to left renal artery (Figure 3C). His heart rate significantly decreased post-operatively, and chest radiography and ECG showed reduced cardiomegaly (CTR, 45%), pleural effusion, pulmonary congestion, and left ventricular hypertrophy (Figure 4A and 4B). Post-operative serum creatinine levels improved to 1.08 mg/dL, and BNP levels decreased to 4 pg/mL. Echocardiography showed reduction in the left ventricular size and improvement in wall motion and diastolic dysfunction (Dd, 44.8 mm; Ds, 32.2 mm; ejection fraction, 54.2%; E/A, 0.6; deceleration time, 166 ms; E/E', 9.5) (Figure 4C); the ABI was almost normal (right, 1.01; left, 1.01) (Figure 4D).
Figure 3  Operative angiography and post-operative contrast-enhanced computed tomography (CT). (A) Angiography showing total occlusion of the left renal artery and 90% stenosis of the right renal artery ostium. (B) Percutaneous transluminal renal angioplasty was performed using an Express Vascular LD 5 × 15-mm stent (Boston Scientific Corporation, Natick, MA, USA) implanted in the right renal artery. (C) Post-operative contrast-enhanced CT showing the aorto-bifemoral bypass, patent left renal artery, and collateral artery from the aorta to the left renal artery (white arrow).

Figure 4  Post-operative examination findings. (A) Chest radiography showing reduced cardiomegaly (cardiothoracic ratio, 45%), pleural effusion, and pulmonary congestion (compared with Figure 1B). (B) Electrocardiogram showing reduced left ventricular hypertrophy (compared with Figure 1C). (C) Echocardiography showing reduction in the left ventricular size and improvement in wall motion (Dd, 44.8 mm; Ds, 32.2 mm; ejection fraction, 54.2%) (compared with Figure 1D). (D) Blood pressure readings showing that the ankle–brachial indices were almost normal (right, 1.01; left, 1.01).
Discussion

Decompensated heart and renal failure caused by Leriche syndrome rarely occurs,5 and no published studies have reported the post-operative improvements in heart and renal failure symptoms seen in our case. We suggest that an increased vascular bed and decreased cardiac afterload contributed to the post-operative improvement of heart failure. Our patient had extended refractory hypertension in the upper half of his body. Cardiac function post-operatively improved based on echocardiographic findings (left ventricular ejection fraction, 30% pre-operatively to 54.2% post-operatively) and serum BNP levels (685 pg/mL pre-operatively to 4 pg/mL post-operatively), which may reflect a decrease in systemic arterial resistance due to the aorto-bifemoral bypass, possibly leading to a reduction in cardiac afterload.

Atherosclerotic renal artery disease presents a broad spectrum of clinical features, including heart failure, hypertension, and renal failure. The key to improving renal failure is increasing renal blood flow.6 Accordingly, we performed PTRA in the right artery, which was technically easy following the removal of the thrombus in the abdominal aorta around the renal arteries. Contrast-enhanced CT showed the collateral artery from the aorta to left renal artery and patency of the right renal artery stent. As the left renal ostium was unclear, bilateral revascularization was technically difficult and thus not performed. In bilateral renal artery stenosis, stent placement in one or both arteries prevents recurrent pulmonary oedema.7

Increasing attention has been paid to flash pulmonary oedema and congestive heart failure associated with atherosclerotic renal artery disease.8 Kawarada et al.9 reported that renal stenting significantly improved left ventricular filling pressure in patients with atherosclerotic renal artery disease and heart failure; however, in previous studies, renal artery stenting did not reduce blood pressure or the number of cardiovascular or renal events better than optimal medical therapy.6

Axillofemoral bypass reportedly improved congestive heart failure in a patient with aortic coarctation complicating Takayasu arteritis10; however, post-operative improvement was not observed with heart or renal failure caused by Leriche syndrome. With recent advancements in catheter technology and angiographic techniques, there has been a shift towards an endovascular-first strategy for Leriche syndrome. In our case, a large thrombus was attached to the aorta around the renal artery; therefore, the risk of embolism would have been high, and PTRA in the right renal artery would have been technically difficult if endovascular therapy was selected. Although waiting for heart function to improve before performing surgery may have been beneficial, we believe that the improvement provided by surgery outweighed this need; hence, surgery was performed relatively early.

In conclusion, aorto-bifemoral bypass and unilateral renal artery stenting can effectively treat heart and renal failure, the dominant clinical manifestations of renovascular hypertension caused by Leriche syndrome. As Leriche syndrome progresses, it primarily presents with fatal cardiorenal failure; thus, careful follow-up is required if untreated, as are preparations for intervention at any time.

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Conflict of interest

None declared.

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