

Abdominal Aortic Grafting for Renal and Lower Extremity Hypoperfusion Due to Aortic Dissection with Progressive Thrombosis of a False Channel

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We present the case of a 53-year-old man with renal and bilateral limb ischemia due to Stanford B aortic dissection. The thrombosis of the false lumen had progressed and compressed the true lumen, developing renal and leg ischemia. Urgent graft replacement of the infrarenal abdominal aorta with proximal fenestration was successfully performed and the patient was discharged without complications. (Ann Thorac Cardiovasc Surg 2006; 12: 152–4)

Key words: aortic dissection, renal and leg ischemia, thrombosis of false lumen

Introduction

Management of patients with ischemic peripheral vascular complications due to aortic dissection is still controversial, especially in patients with progressive thrombosis of the false lumen. Effective treatment such as fenestration, tailoring aortoplasty and catheter intervention is limited because of the thrombus filled in false lumen. We describe a direct approach with graft replacement of the dissected abdominal aorta with proximal fenestration for resolving this complicated situation.

Clinical Summary

A 53-year-old man was referred to our hospital for further treatment of a complicated acute aortic dissection (Stanford B). He had presented with severe back pain of sudden onset, and was diagnosed with acute aortic dissection (Stanford B), one month earlier. Computed tomography (CT) had shown an aortic dissection from the descending thoracic aorta to the terminal abdominal aorta with a persistent false channel. However, no sign of pe-

ripheral ischemic complication was recognized and he was treated initially by medical means. Following medical treatment, he complained of worsening leg fatigue on walking. The groin pulsation of the femoral arteries was weakened (ankle-brachial index of 0.6), and the serum creatinine level was elevated to 2.0 mg/dl on laboratory examination. Reevaluation by CT scan revealed a narrowing of the true lumen, with progression of thrombosis of the false channel at the level of the proximal abdominal aorta, with renal hypoperfusion (Figs. 1A and 1B). MRI also showed interrupted forward flow of the abdominal aorta below the renal arteries (Fig. 2). Surgical treatment was indicated for improvement of the blood supply to the lower extremities and to both kidneys. Direct intervention for a dissected abdominal aorta was selected for radical resolution of this difficult situation.

At operation, the abdominal aorta was approached via median laparotomy. The left-side inferior caval vein and the left renal vein were encountered before dissecting the proximal abdominal aorta. The suprarenal abdominal aorta, and the two right renal, left renal, and both common iliac arteries were also dissected and taped. After heparinization, these were clamped in sequence. The aorta was transected just below the renal arteries and old thrombi in the false channel were carefully removed. The superior mesenteric and renal arteries originated from the true lumen. The septum between the false and true lumens was widely resected extending proximal at the level of renal arteries. An 18×9 mm artificial graft was sutured

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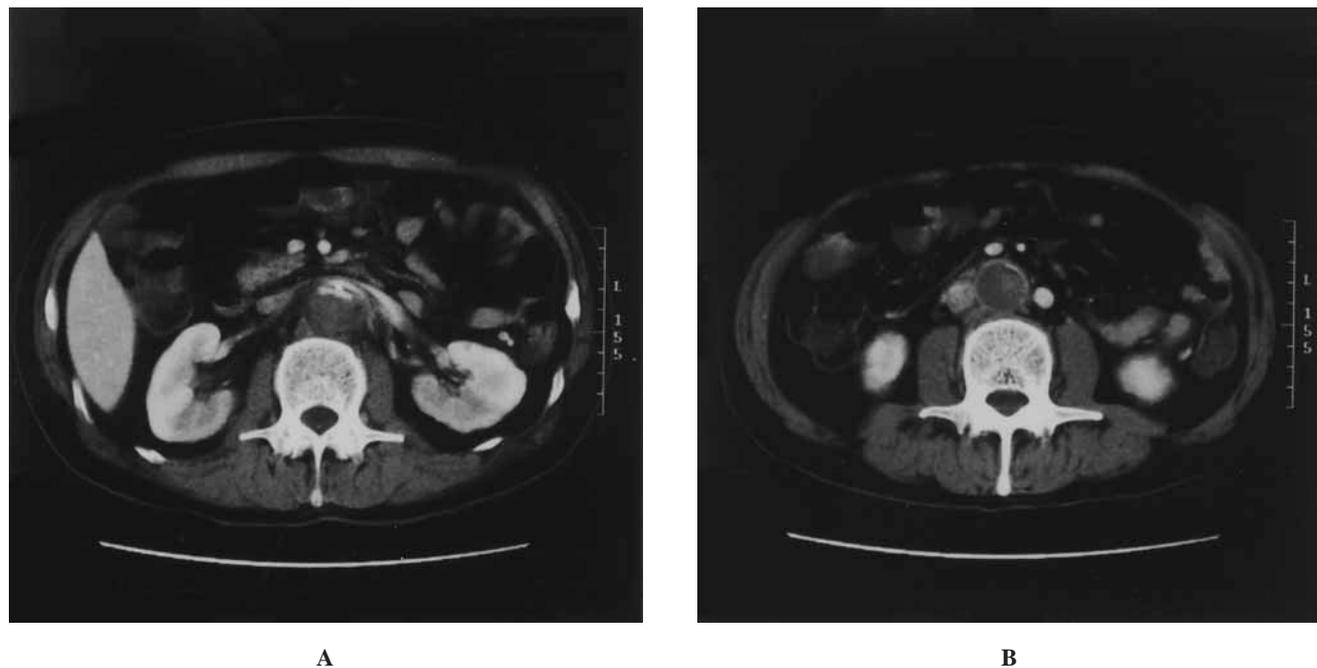


Fig. 1.

- A:** Preoperative CT scan showing a narrowed true lumen with renal hypoperfusion.
- B:** Subtotal occlusion of the abdominal aorta due to progressive thrombosis of the false channel.

to the transected and fenestrated aorta, applying Teflon felt on the outside. After flushing out fragile thrombi, the limbs of the graft were sutured to both common iliac arteries. The renal artery occlusion time was 18 minutes and the patient recovered well. His leg discomfort disappeared completely, and the serum creatinine level decreased promptly. Postoperative CT scan revealed adequate flow in both renal arteries and in the lower extremities. The patient was discharged without complications on the 15th postoperative day.

Discussion

Management of patients with ischemic peripheral vascular complications that develop during treatment of acute aortic dissection is still controversial.¹⁻³ Clarification of the mechanism of branch artery ischemia — which includes compression of the true lumen by the false channel, lack of reentry with the branch arteries originating from the false channel, obstruction due to dissected intimal flap, or thromboembolic genesis — is important for refining the treatment strategy. In patients with distal reentry and a patent false channel, various techniques including fenestration,^{4,5} aortic tailoring,⁶ percutaneous balloon fenestration, and percutaneous stenting⁷ could re-



Fig. 2. Preoperative MRI showing Stanford B aortic dissection and interrupted bloodflow in the abdominal aorta near the renal arteries.

solve organ ischemia. However, in patients with renal, mesenteric, and lower extremity hypoperfusion due to progressive thrombosis of the false lumen, the interventional strategy remains equivocal and challenging. Absence of distal reentry leads to a blind sac with thrombosis, which compresses the true lumen within the undilated aorta, resulting in malperfusion. In this situation effective treatment is limited because the creation of reentry by fenestration, and by tailoring aortoplasty is impossible. Stenting and balloon dilatation of the true lumen may lead to aortic rupture or thrombus migration. A direct approach to the narrowed site of the diseased aorta or an extra-anatomical bypass are the only options for resolving this catastrophic situation.

In the present case, extra-anatomical bypass (such as axillo-bifemoral bypass) would have improved the blood supply to the lower extremities. However, retrograde blood supply to the renal arteries was not expected because of severe and extended narrowing of the true lumen below the renal arteries. Therefore, we selected graft replacement of the dissected abdominal aorta with proximal fenestration — a decision that produced success. In conclusion, a direct approach with grafting appears to be an effective procedure for resolving visceral malperfusion due to aortic dissection with a thrombosed false channel, even during the subacute period.

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