



Spontaneous Resolution of Lower Extremity Hypoperfusion in Type B Aortic Dissection: A Case Report

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ABSTRACT

Lower extremity hypoperfusion occurs in 5.7-30% of Stanford type B aortic dissection cases. A 53-year-old male patient presented with type B aortic dissection. His left femoral pulse was not palpable. The proximal left common iliac artery was nearly occluded in computed tomography angiography. There was no extremity-threatening rest pain, but there was intermittent claudication at 100 meters. Cross femoral bypass was planned for the patient under elective conditions. After two months, the left femoral pulse was palpable, and the patient no longer had intermittent claudication. In this study, we report that lower extremity hypoperfusion, which developed after acute type B aortic dissection resolved without open and endovascular surgery.

Key Words: Dissection; lower extremity; reperfusion; intermittent claudication

Tip B Aort Diseksiyonda Gelişen Alt Ekstremitte Hipoperfüzyonunun Spontan Rezolüsyonu: Bir Olgu Sunumu

ÖZET

Stanford tip B aort diseksiyonu olan vakaların %5.7-30'unda alt ekstremitelerde hipoperfüzyon görülebilir. Elli üç yaşında erkek hasta tip B aort diseksiyonu tanısıyla acil serviste değerlendirildi. Sol femoral nabızın palpe edilemediği hastanın bilgisayarlı tomografi anjiyografisinde proksimal sol ana iliyak arter neredeyse total okludedydi. Ekstremiteleri tehdit eden istirahat ağrısı yoktu, ancak 100 metrede intermitan klodikasyon mevcuttu. Hastaya elektif şartlarda *cross* femoral bypass planlandı. İki ay sonra operasyon öncesinde değerlendirilen hastanın sol femoral nabızı palpe edilebiliyordu ve intermitan klodikasyon şikayeti kalmamıştı. Çalışmamızda akut tip B aort diseksiyonu sonrası gelişen alt ekstremitte hipoperfüzyonunun açık ve endovasküler cerrahiye gerek kalmadan düzeldiğini bildirdik.

Anahtar Kelimeler: Diseksiyon; alt ekstremitte; reperfüzyon; intermitan klodikasyon

INTRODUCTION

Complicated Stanford type B aortic dissection (TBAD) shows different clinical presentations such as aortic rupture, hypotension, shock, neurologic sequelae, and malperfusion syndrome (visceral or lower extremity)⁽¹⁾. Approximately 25-40% of TBAD are of complicated nature⁽²⁾. The early mortality rate of patients with complicated TBAD is significantly higher than patients with uncomplicated TBAD.

Current guidelines recommend that patients diagnosed with complicated acute TBAD should receive immediate TEVAR after initiation of anti-impulse medical therapy⁽³⁾. The purpose of TEVAR is to endovascularly close the entry tear, reduce the pressure of the false lumen, induce thrombosis and restore normal blood flow⁽⁴⁾.

Lower extremity hypoperfusion (LEH) due to TBAD is an abnormal pulse examination accompanying leg pain, pallor, paresthesia, or paralysis⁽⁵⁾. LEH occurs in 5.7-30% of TBAD cases⁽⁶⁾. In this case study, we report that LEH developing after acute TBAD resolves without open and endovascular surgery.

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CASE REPORT

A 53-year-old male patient presented to the emergency department with a sudden onset of chest and back pain. On examination, blood pressure was 175/85 mmHg, rhythm was 105 per minute, and the left femoral pulse was not palpable. There were no problems in the transthoracic echocardiography and electrocardiogram. Thoracoabdominal computed tomography angiography (CTA) was performed with suspicion of aortic dissection. On CTA, TBAD was starting from the left subclavian artery orifice and ending at the bilateral common iliac arteries. Thoracic aorta diameter was 37 mm and false lumen diameter was 17 mm. The proximal left common iliac artery was nearly occluded by hypodense thrombus material. Blood flow was supplied to the left lower extremity by a very thin true lumen (TL) in the common iliac artery (Figure 1.a-c). The patient was started on anti-impulse, antihypertensive, and anticoagulation treatment in the intensive care unit (ICU).

Shock, neurological sequelae, visceral malperfusion syndrome, and persistent pain did not develop. Although the left femoral pulse was not palpable, the patient did not develop rest pain threatening the left lower extremity. Since there was no acute ischemia threatening the extremity; TEVAR or emergency surgery was not planned.

After two days, the patient was transferred to the service. At the follow-up visit, the patient had intermittent claudication (IC) in the left lower extremity at 100 meters. The left ankle brachial pressure index (ABI) measurement was 0.69. Right ABI measurement was 1.01. Cross-femoral bypass was planned for the patient under elective conditions. The patient was discharged with oral antihypertensive and anti-impulse therapy on the fifth day of hospitalization.

When the patient was checked for planned vascular surgery two months later, the IC had decreased. The walking distance was over 500 meters. Left ABI measurement was 0.95. It was decided to perform CTA. In CTA, type B chronic dissection was observed. However, there was no appearance of thrombus occluding the left common iliac artery orifice. The left common iliac artery was supplied with a wider TL (Figure 2.a-c). Under these circumstances, it was decided not to perform cross-femoral bypass surgery on the patient. In the one-year follow-up of the patient, claudication symptoms did not recur.

DISCUSSION

LEH due to AD is one of the most feared clinical scenarios. The concern about LEH is not just the risk of amputation. The presence of LEH is statistically associated with higher in-hospital mortality⁽⁷⁾. The pulse examination in LEH is definitely

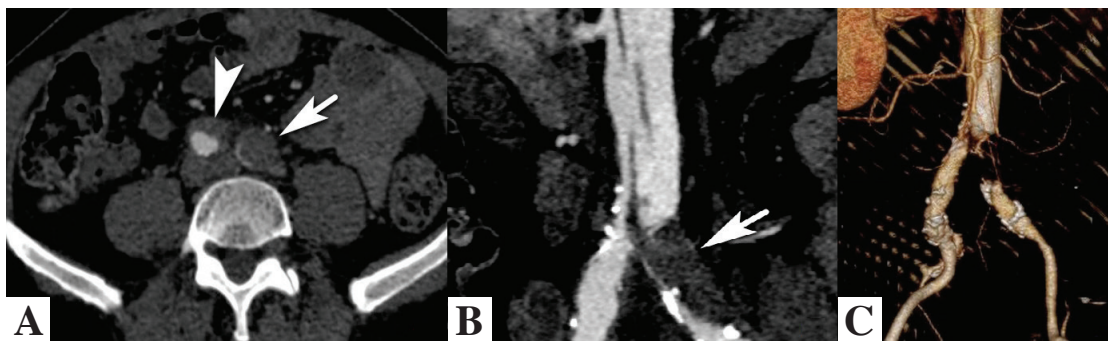


Figure 1. Initial CT angiography of the patient. Axial A, coronal reformatted B, and 3D reconstruction C. CT images of aortic dissection extending in the iliac arteries. The clotted false lumen partially compressed the true lumen in the right iliac artery (arrow-head), where the left iliac artery was almost completely occluded (arrow).

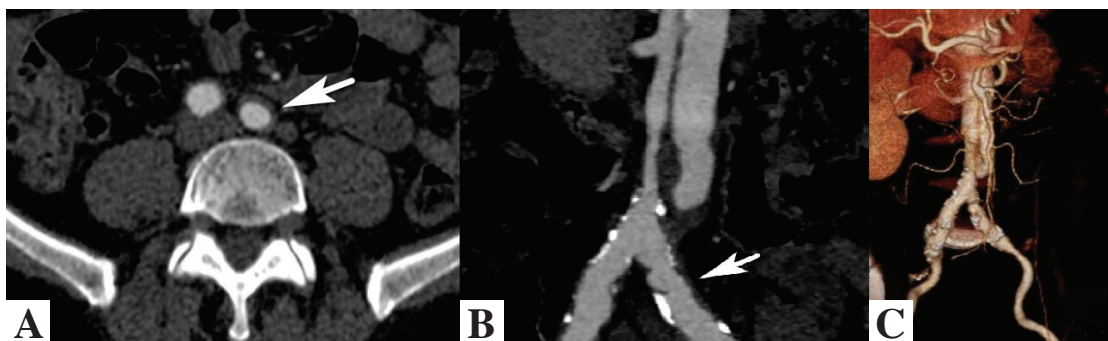


Figure 2. Two-month follow-up CT angiography of the patient after medical therapy. Axial A, coronal reformatted B and 3D reconstruction C. CT images demonstrate the expanded true lumen in the left iliac artery (arrows).

abnormal. In the absence of a pulse, it is necessary to evaluate whether the extremity is under threat. In a systematic review by Gargiulo et al., only 13% of cases that developed LEH due to TBAD did not have leg-threatening ischemia⁽⁶⁾. In these cases, medical treatment such as uncomplicated TBAD is seen as the gold standard. However, if there is ischemia threatening the leg, endovascular or open surgery should be planned according to the morphology of the dissection. Causes such as the collapse of the true aortic lumen in the abdominal aorta or iliac obstruction may require different treatments⁽⁸⁾. As in renal and visceral ischemia, TEVAR, fenestration, or both can be performed endovascularly. In TEVAR, it is planned to close the entrance tear, restore normal blood flow, and correct ischemia. TEVAR is a good option if conditions such as abnormal capillary filling time, paresthesia, paralysis, or rest pain in the leg are present. We did not plan TEVAR as there was no leg-threatening ischemia. The current approach in surgical treatment is extra-anatomical bypass. Cross-femoral bypass is preferred if the flow and structure of the contralateral femoral artery are suitable for bypass. If there is a flow problem in both femoral arteries, axillo-bifemoral bypass is preferred⁽⁶⁾.

In our case, dissection was the most probable diagnosis, since the left femoral artery pulse was not palpable and there was chest and back pain. At this stage, we believe it is crucial to determine whether the lack of a pulse is due to dissection or peripheral arterial embolism, because otherwise, there may be delays in diagnosis and perhaps inappropriate treatment.

We did not plan a left lower extremity flow reconstruction at the first stage as there was no ischemic rest pain in the extremity. In clinical follow-ups, a cross-femoral bypass was planned because the patient had IC.

Changes in the flow pattern due to the dynamic nature of the dissections may resolve extremity hypoperfusion⁽⁹⁾. Dissection-induced hypoperfusions are caused by branch vessel obstructions. Williams et al.⁽¹⁰⁾ reported two types of branch vessel obstruction due to dissection: static and dynamic obstruction. In our case, it was seen that both static and dynamic obstruction caused hypoperfusion. Thrombus causing near-total stenosis at the level of the left common iliac artery was causing the static obstruction. The false lumen located to the left at the level of the abdominal aorta bifurcation caused dynamic obstruction in the true lumen.

In the two-month follow-up, the patient's IC resolved spontaneously. As seen on the CTA, FL thrombosis at the level of the abdominal aorta bifurcation reduced the dynamic obstruction. Also, static obstruction regressed with the resolution of thrombus in the left common iliac artery. We believe that the synergy formed by the regression of both obstruction mechanisms resulted in minimal residual stenosis in the iliac artery.

In conclusion, peripheral artery surgery can be postponed for some time in TBAD patients with stable IC symptoms. We recommend a comprehensive reassessment of patients' ischemic symptoms prior to peripheral artery surgery.

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