



Endovascular treatment of acute deep venous thrombosis secondary to may-thurner syndrome

May-thurner sendromuna sekonder olarak gelişen derin ven trombozunun endovasküler tedavisi

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Abstract

Iliofemoral deep venous thrombosis (DVT) is five times more likely to occur in the left leg when compared with the right leg. May and Thurner provided an explanation for this phenomenon by discovering an anatomical variation of left common iliac vein in 1957. This syndrome is caused by vascular thickening of the left common iliac vein at the crossing point and compression between the fifth lumbar vertebrae and overlying right common iliac artery. Cases with Iliofemoral extension of the thrombus including iliofemoral obstruction create a major challenge. Although in MTS, anticoagulant therapy alone does not prevent long-term complications. To avoid re-thrombosis, venous outflow should be re-established, ensuring that there is no residual stenosis in the left iliac vein. Here in, we present a case of our experience about treatment for DVT due to MTS with endovascular method and evaluate our result of this treatment.

Keywords: May-Thurner Syndrome; Endovascular Treatment; Deep Venous Thrombosis.

Öz

İlio-femoral derin ven trombozu (DVT) sol alt ekstremitede sağ alt ekstremiteye oranla 5 kat daha sık izlenir. May ve Thurner 1957'de anatomik varyasyonu keşfetmesi ile bu fenomene bir açıklama getirmiştir. May-Thurner sendromu (MTS) olarak anılan bu sendrom, sol ana iliak venin sağ ana iliak arteri çaprazladığı noktada, arter ve 5. lumbar vertebranın yapmış olduğu basya sekonder gelişen ven duvarının kalınlaşmasıdır. İlio-femoral bölgeye kadar uzanan DVT tedavisi zordur. MTS gibi altta anatomik bir nedenin olduğu durumlarda tek başına antikoagülan tedavi uzun dönem komplikasyonları engellemede yeterli kalmamaktadır. Re-tromboz riskini azaltmak ve venöz dönüşü tekrar sağlamak için iliak vende rezidüel stenozun kalmadığından emin olmak gereklidir. Bu olgumuzda MTS sendromuna sekonder olarak gelişen DVT'nin endovasküler yöntemle tedavisi ve sonuçlarını inceleyeceğiz.

Anahtar Kelimeler: May-Thurner Sendromu; Endovasküler Tedavi; Derin Ven Trombozu.

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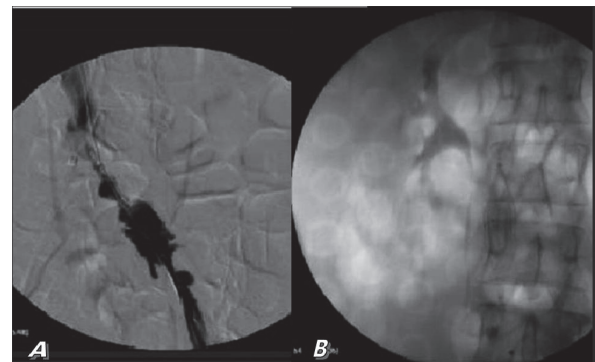
INTRODUCTION

Iliofemoral deep venous thrombosis (DVT) is five times more likely to occur in the left leg when compared with the right leg. May and Thurner provided an explanation for this phenomenon by discovering an anatomical variation of left common iliac vein in 1957. May-Thurner syndrome (MTS) is also known as iliac vein compression syndrome. This syndrome is caused by vascular thickening of the left common iliac vein at the crossing point and compression between the fifth lumbar vertebrae and overlying right common iliac artery (1, 2). Cases with iliofemoral extension of the thrombus including iliofemoral obstruction create a major challenge. The treatment should prevent thrombus extension, pulmonary embolism and long-term venous insufficiency, such as post-thrombotic syndrome (PTS). Although in MTS, anticoagulant therapy alone does not prevent long-term complications (3, 4, and 5). To avoid re-thrombosis, venous outflow should be re-established, ensuring that there is no residual stenosis in the left iliac vein (6). Herein, we present a case of our experience about treatment for DVT due to MTS and evaluate our result of this treatment.

CASE REPORT

A 21-year-old female patient with no past medical history, presented for evaluation of a new onset, left lower limb swelling and pain. The swelling was first appeared 3 days earlier and progressed over time. There was no prior history of DVT, immobilization or use of oral contraceptives. She was hemodynamically stable. The physical examination was normal, despite the pitting left lower limb edema. Lower limb pulses were all normal. Blood investigations showed no obvious blood clotting disorder. Duplex ultrasound (DUS) was performed and it confirmed left lower limb DVT extending to the left common iliac vein. According to DUS; the common iliac vein, which is beneath the right common iliac artery, appeared mildly compressed, because of the overlying artery on B-mode US. In DUS there was no flow pattern of the external and common iliac veins distal to the arterial compression point. Anticoagulation was carried out right after the diagnosis. Unfractionated heparin was given with an initial intravenous bolus dose of 1000 U and then its infusion rate was titrated according to activated coagulation time at a rate of 600-1000 U/h. Venography was scheduled for definitive diagnosis and placement of the ultrasound accelerated thrombolysis (UAT) catheter. The procedure was explained in detail to the patient and her family and written informed consent for venography was obtained. Venographic examination was performed in an angiography suite and using a digital subtraction angiography unit (BV Pulsera, Philips Healthcare, and Holland) five days after the DUS examination. She was placed in the prone position and an ascending venogram was obtained from the left vena saphena parva (VSP) through popliteal vein to observe the extent and location of thrombosis. Venography revealed severe compression of the left common iliac vein with significant stenosis (Figure 1a).

After the examination; we placed the distal tip of the UAT catheter, 1 cm beyond the stenotic area into the common iliac vein and proximal tip was in superficial femoral vein. After thirty six hours of UAT therapy with tissue plasminogen activator (t-PA) which was continuously infused at a rate of 1mg/h and unfractionated heparin which was continuously infused at a rate 600-1000U/h, DUS showed that the distal common, external iliac veins and common femoral vein were patent with partial thrombosis. The common iliac vein beneath the right common iliac artery appeared mildly compressed by the overlying artery on B-mode US and the flow pattern of the external and common iliac veins distal to the arterial compression point was monotonous and monophasic. We ended up the UAT therapy and scheduled endovascular therapy on the next day. During this time anticoagulation with unfractionated heparin was continued. Next day before the endovascular treatment, control DUS was performed and according to DUS, there was no flow pattern of the external, common iliac veins and common femoral vein distal to the arterial compression point again. Vena saphena magna (VSM) remained the main collecting vein for lower extremity. For endovascular treatment; patient was placed in the supine position, and an ascending venogram was obtained from the superficial femoral vein to observe the extent and location of thrombosis and stenosis. Anticoagulation was carried out during the endovascular treatment. In view of the risk of pulmonary embolism during endovascular treatment, we prophylactically placed the deployment of retrievable inferior vena cava filter (IVCF) from the right common femoral vein. IVC filter placed at 2 cm below the renal veins right after iliac bifurcation (Cordis, Johnson and Johnson Medical NV) (Figure 1b).



Figür 1a-1b. Venography revealed severe compression of the left common iliac vein with significant stenosis, inferior vena cava filter placed at 2 cm below the renal veins right after iliac bifurcation

After placement of IVCF, left common femoral vein was punctured directly under ultrasound guidance. A 10F vascular sheath was inserted to obtain a diagnostic venogram. Catheter was successfully manipulated over hydrophilic stiff guide wire, and successfully transversed through occluded segment to left common iliac vein. Venogram was performed to obtain the position of catheter and multiple balloon dilatation was done to common and external iliac veins. It was performed

starting by 9.0 × 80 mm (Boston Scientific) which was followed by 14, 16 × 40 mm balloon (Boston Scientific). A 16 × 60-mm self expanding wallstent (Boston Scientific) was deployed across the occluded segment of the common iliac vein. The stent was dilated to 16 × 40 mm with the angioplasty balloon (Boston Scientific) (Figure 2a). A post-angioplasty venogram demonstrated a patent stent (Figure 2b). A post-angioplasty venogram demonstrated a patent stent, but the contrast material flow was very slow through the stent into the IVC. The probable reason of the inefficient flow throughout the stent was the thrombosis in the distal common and superficial femoral vein. VSM was the main vessel which brought the venous flow from lower extremities to proximal common femoral vein. After the endovascular therapy, we reperformed UAT therapy for the next thirty six hours. Controlled DUS showed VSM flow was much better and there was a partial flow in superficial femoral vein and common femoral vein. The flow throughout the stent was accelerated. We ended up the UAT therapy and continued with unfractionated therapy alone for the next ten days. In the tenth day we retrieved VCIF without any complication. After this procedure we began warfarin therapy.

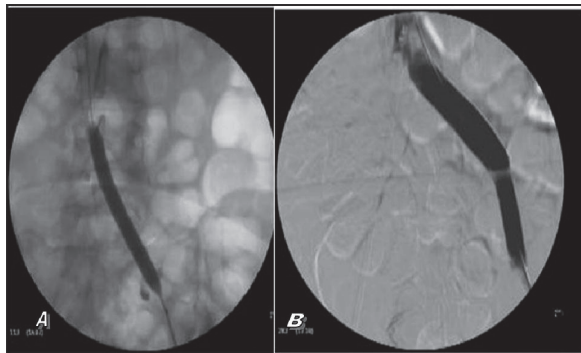


Figure 2a-2b. The stent was dilated to 16 × 40 mm with the angioplasty balloon at common iliac vein, post-angioplasty venogram demonstrated a patent stent)

DISCUSSION

DVT can be either acute or chronic. Patients with acute DVT mostly present with sudden pain and onset of unilateral leg swelling. It is more common in the left than the right lower extremity, and MTS is considered to be a risk factor for patients with left-sided iliofemoral DVT. It occurs more likely in young to middle aged women (7, 8). A retrospective study conducted by Kibbe MR et al. showed the prevalence of MTS was around 22-24% (9). Despite this high incidence, the clinical incidence of MTS related DVT reported is around 2-3% (10). The optimal treatment of MTS related DVT is removing the clot burden, restoring the venous outflow and reducing the extremity vein pressure. Anticoagulation therapy alone is ineffective for complete removal of clot. Today increasing use of UAT therapy and stent implantation are an effective treatments for MTS related DVT. It is superior to surgery in terms of minimal trauma, preserves of vein valve function and it doesn't require any incision (11). There also studies show that UAT

therapy does not always prevents postthrombotic damages (12). The present case showed that UAT therapy combined with endovascular stent intervention can be safely and effectively used for the treatment of acute massive proximal DVT secondary to MTS. O'Sullivan et al. (1 year 93.1%) and Kawk et al. (2 year 95%) showed good stent patency in their studies (13, 14). It is important that, early medical and endovascular treatment of MTS associated DVT can prevent pulmonary embolism, long-term venous damage and PTS (15). After UAT therapy, treating only with heparin was inefficient without rapid dilatation of the stenosis caused by MTS. In our case, duration between UAT therapy and endovascular stent procedure was more than 18 hours. During this time period, in spite of continuation of heparin therapy, we observed worsening findings in DUS. Even we were successful after all, we suggest the duration between these two procedures should be as short as possible. This strategy will reduce the risk of re-thrombosis and improve the outcome of this procedure. Duration of posttreatment therapeutic anticoagulation is controversial and should be individualized for each patient.

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