» Case Report «

Endovascular Management of May-Thurner Syndrome

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May-Thurner syndrome or iliac vein compression syndrome is associated with deep vein thrombosis (DVT) resulting from chronic compression of the left iliac vein against lumbar vertebrae by the overlying right common iliac artery. Historically, May-Thurner syndrome has been treated with anticoagulation therapy. However, this therapy can be problematic when given alone, because it prevents the propagation of the thrombus without eliminating the existing clot. Furthermore, it does not treat the underlying mechanical compression. Consequently, syndrome who was managed by anticoagulation therapy alone, there is a significant chance that the patient will develop recurrent deep vein thrombosis or post thrombotic syndrome or both. Recently, both retrospective and prospective studies have suggested that endovascular management should be front-line treatment; endovascular management actively treats both the mechanical compression with stent placement and the thrombus burden with chemical dissolution. We report our case of 53 years old male patient with May Thurner syndrome who managed by endovascular treatment.

Keywords: may-thurner syndrome, iliac vein compression syndrome

INTRODUCTION

In 1957, May and Thurner described three varieties of Intra-luminal spurs occurring in the left common iliac vein close to its junction with the inferior vena cava (IVC).¹⁾ The incidence of May-Thurner syndrome is unknown and ranges from 18%–49% among patients with left-sided lower extremity DVT.²⁾ May and Thurner postulated that the chronic pulsations of the overriding right iliac artery led to the development of a “spur” in the vein wall and that this spur would result in partial venous obstruction, Chronic trauma to the inner side of the vein wall due to adjacent arterial pulsations leads to the accumulation of elastin and collagen, contributing to spur formation.³⁾ In addition to the chronic arterial pulsations, mechanic compression of the iliac vein by the thick-walled overriding iliac artery (**Fig. 1**) leads to ex-

tensive local intimal proliferation, impaired venous return and venous thrombosis. In addition to the mechanical alterations to the vessel wall, hypercoagulable states, when tested, are found in the majority of patients. Kolbel et al.⁴⁾ found underlying hypercoagulable disorders in 67% of patients screened prior to treatment of chronic iliac vein occlusion. Left iliac vein compression is the most common variant seen in May-Thurner syndrome; however, several other variants have been described in the literature. Compression of the left common iliac vein by the left internal iliac artery,⁵⁾ compression of the right common iliac vein by the right internal iliac artery,⁶⁾ compression of the IVC by the right common iliac artery⁷⁾ and right-sided May-Thurner syndrome in a patient with a left-sided IVC⁸⁾ have all been described.

CASE REPORT

53 years old male with diabetes mellitus and hypertension who persistent with painless swelling of the left thigh of about 7 years duration. The swelling had gradually become more severe. Physical examination revealed a swollen left thigh, circumference of which was 15 cm larger than the right one. The patient had two episodes of venous thrombosis, and the last episode occurred 2

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Received: January 19, 2012; Accepted: March 20, 2012
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Fig. 1 CT of the abdomen and pelvis with contrast showing compression of left iliac vein by left iliac artery, multiple collaterals seen at abdominal wall.

weeks earlier, which dramatically aggravated his symptoms. He was treated with warfarin 6 mg and aspirin 300 mg with no appreciable improvement. Venous Doppler ultrasonography (U.S.); revealed left common femoral thrombosis, occluded left common iliac vein with subsequent aneurysmal dilatation of external iliac vein. Abdominopelvic computed tomography (CT) was done with dual-phase thin-section (section thickness, 1.25 mm). Abdominal CT was performed at arterial (angiography) and venous (venography) phases with four-channel multi-detector row CT. Three-dimensional volume rendering and abdominal CT demonstrated normal arterial anatomy and compression of the left common iliac vein by the left common iliac artery with total occlusion of the common iliac vein, aneurysmal dilatation of left external iliac vein with multiple collaterals seen along left thigh and anterior abdominal wall, as well as evident thrombosis of common femoral vein which extended down to involve superficial femoral and popliteal veins. No pelvic mass was noted. Endovascular venoplasty was decided.

A 6-F sheath was placed into the right common femoral vein; Rt iliac venography and cavogram was done followed by deployment of retrievable IVC filter just below renal vein retrievable OptEase filter (Johnson and Johnson, USA), another 8 F sheath was placed into left common femoral vein, venogram was done, and total occlusion of iliac vein was observed with subsequent aneurysmal dilatation of left external iliac vein with multiple collaterals (**Fig. 2**), micro-catheter (Renegade by Boston scientific, USA) was successfully manipulated over 0.018 wire (Transend 0.018 wire, Boston Scientific,

Fig. 2 8F sheath was placed into left common femoral vein, venogram revealed total occlusion of left common iliac vein with subsequent aneurysmal dilatation of left external iliac vein.

USA) and successfully transverse through occluded segment to IVC, cavogram was done to confirm the position of micro-catheter, multiple balloon dilatation were done starting by 2 mm × 6 mm (Sleek balloon by Clear Stream Technologies) followed by 4, 6, 8, 10, and 12 × 40 mm Power flex balloon by (Johnson and Johnson, USA). A 14 mm × 40-mm Wallstent (Boston Scientific, USA) was deployed across the occluded segment of the vein. The stent was dilated to 14 × 40 mm with the angioplasty balloon. A post-angioplasty venogram demonstrated a widely patent stent and good contrast material flow through the stent into the IVC, without filling of the cross-pelvic collaterals (**Fig. 3**). Measurement demonstrated no pressure gradient across the stent in the supine position.

While the patient was lying prone on the angiographic table, we accessed the popliteal vein under ultrasonographic (U.S.) guidance with a small-gauge echogenic needle to avoid inadvertent puncture of the adjacent popliteal artery. A 5-F sheath was inserted through which all subsequent catheter and wire exchanges were performed. Using (multi-side port catheter infusion set, Cook Medical, USA). 10 mg of tissue plasminogen activator (tPA) (actilyse, Boehringer Ingelheim, Germany) was injected through infusion catheter as a loading dose, and then infusion catheter was connected to actilyse

Fig. 3 A post-angioplasty venogram demonstrated a widely patent stent and good contrast material flow through the stent into the inferior vena cava, without filling of the cross-pelvic collaterals.

infusion pump with a dose of 0.5 mg/hour, Lysis progress was monitored at venography at 8 hours interval, total lysis was achieved after 16 hours.

The patient was discharged from the hospital on the same day and prescribed the following drug regimen: warfarin 6 mg for 3 months, then 300 mg of aspirin daily for life and 75 mg of Clopidogrel (Plavix, Sanofi-Dogu, Istanbul, Turkey) daily for 6 weeks. At 2 weeks follow-up, Doppler U.S. revealed patent venous stent, and left thigh edema dramatically decreased. Then IVC filter was retrieved. At 3-month follow-up, Doppler U.S. revealed patent venous stent, and left thigh edema was substantially decreased.

DISCUSSION

A history of persistent left lower extremity swelling with or without DVT in a woman between the 2nd and 4th decades of life, without an obvious cause, is highly suggestive of May-Thurner syndrome, and this possibility should be assessed with CT and iliac venography. It is not an infrequent source of venous abnormalities in the left lower extremity. The true prevalence of this disorder is unknown. With the move away from venography and the increased use of noninvasive diagnostic measures

to confirm the presence of venous thrombosis, many cases of the left iliofemoral venous thrombosis associated with May-Thurner syndrome are probably not recognized.

The overlying artery appears to induce a partial obstruction of the vein in two ways: by its anatomic orientation with subsequent physical entrapment of the left common iliac vein and by extensive intimal hypertrophy of the vein resulting from the chronic pulsatile force of the right common iliac artery. This condition has been estimated to occur in 2%–5% of patients who undergo evaluation for lower extremity venous disorders, and it is not known why the normal anatomic relationship between the left common iliac vein and right common iliac artery is disrupted and begins to interfere with venous flow.

Reported findings show that lower extremity DVT occurs three to eight times more frequently in the left side than on the right.⁹ In 1943, Ehrlich and Krumbhaar performed anatomic dissections in 412 cadavers and found obstructive lesions in 23.8% of the left common iliac veins. Histologically, these lesions did not represent chronic recanalized clot; rather, they were composed of elastin and collagen, without inflammatory cellular infiltration or irregular arrangement of scar. They also found that 33.8% of lesions occurred after the 1st decade of life and concluded that the lesions were acquired and not congenital.

Patients with May-Thurner syndrome typically present with unilateral (left) lower extremity edema and pain. A propensity for this disorder is seen in young women in their second to fourth decade of life, after prolonged immobilization or pregnancy. Because of the chronic nature of the disease process, patients may also present with stigmata associated with post-thrombotic syndrome such as pigmentation changes, varicose veins, chronic leg pain, phlebitis and recurrent skin ulcers. The clinical stages of iliac vein compression were described by Kim et al.,¹⁰ and include Stage I, asymptomatic iliac vein compression; Stage II, development of a venous spur; Stage III, development of left iliac vein DVT.

Although the diagnosis of May-Thurner syndrome is based on the clinical presentation of left lower extremity swelling and pain in association with radiologic evidence of compression, however, the diagnosis of May-Thurner syndrome may not always be straight forward. May and Thurner,¹¹ have advocated the use of pressure differentials to support the diagnosis of hemodynamically significant obstruction. They have suggested¹¹ that a pressure differential between the two iliac veins of 2 mmHg at rest or 3 mmHg with exercise is significant and that an

exaggerated pressure response to exercise is a marker of significant obstruction.¹¹⁾ Other authors have utilized inferior vena caval pressure as a surrogate for contralateral iliac vein pressures with the assumption that there should be little or no gradient between the inferior vena cava and the iliac vein unless obstruction is present.¹¹⁾

The finding that many patients with iliofemoral compression remain asymptomatic raises the question of whether this represents a normal anatomic variant. On the other hand, some patients with this vascular abnormality develop significant symptoms of chronic venous outflow obstruction, including pain, unilateral left lower extremity edema, venous claudication, varicose vein formation, venous stasis ulceration, and unprovoked DVT. Although, the true incidence of thrombosis in the setting of iliac compression syndrome is uncertain, there is evidence that patients in whom thrombosis develops may be at higher risk for recurrent DVT, so treatment should be started as soon as possible. Mickley et al.¹²⁾ have reported on a series of selected patients who underwent thrombectomy for iliofemoral thrombosis. Of patients with left-sided thrombosis, 49% had venous spurs found intraoperatively. Prior to 1994, those spurs were untreated, and 72% of those patients had recurrent occlusive thrombosis despite treatment of at least 1 year with vitamin K antagonists. In comparison, recurrence occurred in only 1 of 28 similarly treated patients (4%) with either right-sided thrombosis or left-sided thrombosis without spur formation.

In patients in whom suspicious signs or symptoms develop, the diagnosis of May-Thurner syndrome is best made radiographically. Other causes of these symptoms, including trauma, postsurgical changes, pelvic masses, and radiation, must be excluded. Doppler ultrasound will detect the lesion if a DVT is present in the iliac vessels, but is unable to visualize iliac vein compression and spurs.¹³⁾ Other diagnostic modalities include helical abdominal CT, CT venography, magnetic resonance venography, intravenous ultrasound, and conventional venography. Kibbe et al.¹⁴⁾ used abdominal helical CT scanning to determine the incidence of left common iliac vein compression in an asymptomatic population. They found that two-thirds of all patients who were studied had at least 25% compression of the left iliac vein. The authors concluded that compression of the left iliac vein may be a normal anatomic finding and that abdominal CT scanning is accurate in determining if left iliac vein compression is present. There are, however, limitations for the use of abdominal CT scanning in determining if iliac vein compression is present. The CT scans were obtained

during the arterial phase of the intravenous bolus, which limits the type of vessel reconstruction and analysis that can be performed. CT venography may be used as an effective adjunctive modality when there is a known DVT. Chung et al.¹⁵⁾ found that CT venography was just as specific and highly sensitive in the diagnosis of DVT compared with ultrasound and accurately delineated venous anatomy and the extent of thrombus present. A limitation of CT venography involves the inability to control for the volume status of the patient, which could lead to overemphasis of the degree of compression of the left iliac vein in a dehydrated patient. The traditional “gold standard” for diagnosis of May-Thurner syndrome is conventional venography, which can be diagnostic and therapeutic when endovascular therapy is used. Non-invasive imaging methods are being used increasingly to diagnose DVT and iliac compression. The aforementioned imaging modalities may help in planning catheter-directed thrombolysis without the initial need for conventional venography. These non-invasive imaging modalities are simple, efficient and cost-effective in diagnosing DVT associated with iliac compression.³⁾

May-Thurner syndrome is a progressive disease with substantial long-term disabling complications. An aggressive approach designed to relieve the mechanical compression should be strongly considered. Multiple surgical treatment options have been advocated. These include vein-patch angioplasty with excision of intra luminal bands, division of the right common iliac artery and relocation behind the left common iliac vein or IVC, and contra lateral saphenous vein graft bypass to the ipsilateral common femoral vein with the creation of a temporary arteriovenous fistula (Palma cross over). Overall, the reported long-term success, defined primarily as patency of the left common iliac vein or venous bypass, is 40%–88%. More recently, treatment with endovascular techniques has been described. The first known report of treatment of May-Thurner syndrome solely by endovascular means was by Berger et al. in 1995,¹⁶⁾ who successfully placed a venous stent to relieve iliac compression. Several subsequent studies have demonstrated efficacy in the treatment of iliac vein compression with thrombectomy and endovascular stenting. Self-expandable stents are used in the venous system as they can cover long distances, are easy to re-sheath and have adequate durability. Balloon-expandable stents may be used if needed (insufficient response to pre dilatation/self-expandable stent). Extending the stent into the IVC may be done without increasing the risk of contra lateral iliac vein occlusion.¹⁷⁾

In summary, it is important to recognize that persistent edema of the left leg may be caused by May-Thurner syndrome, especially in young women. This diagnosis is confirmed with ascending iliac venography, which demonstrates the iliac vein compression. The mechanical compression should be relieved prior to the onset of DVT and venous insufficiency. Management of May-Thurner syndrome has evolved over the past few decades favoring endovascular management as the primary treatment. With early recognition and aggressive management, May-Thurner syndrome can be a well-managed disease.

DISCLOSURE STATEMENT

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article. The author(s) received no financial support for the research, authorship, and/or publication of this article.

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