May – Thurner Syndrome: A Case Based Review

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Introduction

May-Thurner syndrome is a disorder of left common iliac vein compression by an overriding right common iliac artery that leads to deep venous thrombosis (DVT). This syndrome was first described in 1957 by May and Thurner [1], but has remained under-recognized as an etiology for DVT. Even though the anatomical variant is common, not all patients will develop symptoms and therefore the prevalence of this syndrome is difficult to assess. Patients may present with acute DVT or signs and symptoms of chronic venous hypertension such as venous reflux or hyper pigmented skin changes. The diagnosis is made clinically based on history and exam along with the aid of imaging modalities to document iliac vein compression by the iliac artery. Treatment of May-Thurner syndrome includes anticoagulation in addition to more definitive therapy to correct the anatomical disorder with endovascular stenting or open surgical repair. We present a case of May-Thurner syndrome followed by a review of the current literature on the subject.

Case Report

A 28 year old East Asian male with no significant past medical history presented to the Emergency Department at Jacobi Medical Center (Bronx, N.Y.) with pain and swelling to his left lower extremity for 4 days. The patient was a waiter by profession and reported the onset of pain after prolonged standing during a long work day. Initially his leg was mildly sore and over the next few days he developed pain in the left lower back, thigh and leg with associated swelling. The pain acutely worsened the night before presentation with difficulty ambulating and moving his leg. He denied any injury or recent prolonged travel requiring his leg to be immobile. His family history was non-contributory. He denied any medication or recreational drug use. His vitals were stable and his BMI was 17.7 kg/m². On exam, his left leg was warm and tensely swollen from the ankle to the mid-thigh, and diffusely tender to palpation. His distal pulses were intact. His strength, reflexes and sensation were also intact. His complete blood count, basic metabolic profile and PT/INR were normal.

An X-ray did not show any evidence of fracture. A venous Doppler demonstrated extensive deep vein thrombosis (DVT) extending from the popliteal vein to the common femoral vein with a dilated external iliac vein without flow (Fig 1 A,B). Incidentally he was noted to have concern for left hydrenephrosis. A CT Angiogram demonstrated the right common iliac artery over-riding a dilated left common iliac vein (Fig 2).

**Fig 1 A** – Dilated and non-compressible common femoral vein (White Arrow) with thrombus

**Fig 1 B**- Color Doppler demonstrating flow in the common femoral artery (A) and no flow in the femoral vein (V).
Figure 2 – CT Angiogram demonstrating right common iliac artery (arrow) crossing the left common iliac vein (arrow head). Also incidentally noted to have massive asymptomatic left hydronephrosis.

Interventional Therapy

Given the extent of DVT, patient’s young age, good health and high risk for post-thrombotic syndrome, catheter-directed thrombolysis was initiated after informed consent was obtained. The patient was brought to the cardiac catheterization laboratory and placed in the prone position on the table. Ultrasound-guided access was obtained in the left popliteal vein using a micropuncture kit (Cook Medical, Bloomington, IN). A venogram was performed through the micropuncture sheath which confirmed a large clot burden (Fig 3A) and collaterals from the left iliac to the right iliac veins (Fig 3B). A 7 French Glide sheath (Terumo Corporation, Tokyo, Japan) was placed in the popliteal vein and a 0.035” Glidewire (Terumo Corporation, Tokyo, Japan) was advanced through the thrombus into the inferior vena cava. An EKOS catheter (EKOS Corporation, Bothell, WA) was placed. A 10mg bolus of tissue plasminogen activator (TPA) was given followed by an infusion at a rate of 1.5mg/hr. Heparin at 500units/hr was given via the side port of the venous sheath. After 8 hours, the patient was taken back to the catheterization laboratory and a repeat venogram demonstrated marked reduction in clot burden with mild residual clot in the femoral vein and external iliac vein and reduction of collateral flow but severe stenosis at the left common iliac vein (CIV) - IVC junction. Manual thrombectomy was performed using a 7 French Hockey stick catheter (Cardis Corporation, Bridgewater Township, New Jersey) followed by balloon angioplasty of the left CIV stenosis with a 10mm x 60mm Mustang balloon (Boston Scientific, Natick, MA) and 14mm x 40mm Atlas balloon (Bard Peripheral Vascular Inc, Temple, AZ). Repeat venogram demonstrated resolution of thrombus with residual stenosis at the left CIV-IVC junction (Fig 4A). A 16mm x 60mm self-expanding Wall stent (Boston Scientific, Natick MA) was therefore deployed at the area of stenosis extending into the IVC (Fig 4B). The stent was post dilated successfully.

Fig 3A- Venogram demonstrating occlusion of left femoral vein with heavy thrombus burden (3A, arrow) and patent deep collateral vein.

Fig 3B- Venogram in the AP projection demonstrating thrombus in the Left iliac vein(arrow head) with collaterals from the left to right venous system (Arrow)

Fig 4A- Venogram after thrombolysis demonstrating residual stenosis at the left CIV-IVC junction (white arrow).

Fig 4B- Venogram after stent placement (Arrow) demonstrating brisk flow across the area of stenosis with no residual stenosis or clot. Stent extends into IVC and is anchored against wall.
After stent placement the patient received therapeutic anticoagulation with enoxaparin and was bridged to therapeutic warfarin. A venous Doppler the next day demonstrated no residual venous thrombosis. The pain and swelling in his left leg improved dramatically and he was discharged home on post-operative day three with compression stockings. He followed up in clinic and a repeat ultrasound at 2 weeks demonstrated patent stent with no clot (Fig 5).

A hypercoagulability workup, including Factor V Leiden, Antithrombin III, homocysteine and prothrombin gene mutation, was negative.

**Pathophysiology**

May-Thurner syndrome occurs when the iliac vein develops evidence of venous hypertension or deep venous thrombosis due to chronic compression by an overlying right common iliac artery against the lumbar vertebrae [5]. Although there are other parts of the body where an artery apposes a vein, the vein is simply displaced in those circumstances and therefore does not feel the effects of arterial pulsation or compression. In the case of May-Thurner syndrome, the position of the iliac vein in between the right iliac artery and vertebral body leads to the compression effect. It has been postulated that chronic compression and arterial pulsations lead to the development of endovascular intimal proliferation causing venous “spur” formation, impaired venous return and blood stasis leading to thrombus formation. Autopsy studies of patients with lower extremity DVT revealed presence of large amounts of elastin and collagen in the thickened venous wall, without inflammatory cellular infiltration or scar tissue. There are three morphologic types of May-Thurner syndrome: focal compression by the overlying iliac arteries, diffuse atrophy and cordlike obliteration of the iliac vein. Diffuse atrophy and cordlike obliteration represent chronic effects of focal compression leading to fibrosis. Once the venous walls have formed spurs leading to luminal narrowing, the process is felt to be irreversible. Patients with venous spurs may remain asymptomatic for an extended time due to the formation of collateral venous drainage.

Left common iliac vein compression is the most common variant seen in May-Thurner, but there are other less common variants, including left common iliac vein compression from a left common iliac artery, compression of the right common iliac vein by the right internal iliac artery and right-sided May-Thurner in a patient with a left-sided IVC [6]. There are also reports of thrombophilia in patients with May-Thurner and some experts have advocated performing a hypercoagulable work-up in all patients who present with this syndrome [7].

**Clinical Manifestations**

The clinical presentation of May-Thurner can vary widely. There are many patients with iliac vein compression without any symptoms while others may present with chronic venous insufficiency and/or DVT, the hallmark of May-Thurner syndrome. Patients will present with unilateral lower extremity edema and pain, predominantly on the left side. Some also present with venous claudication. However, because of the chronic and progressive nature of the disease process, patients may have stigmata of post-thrombotic syndrome such as venous reflux, hyperpigmentation of the skin, varicose veins (retroperitoneal and pudendal venous collaterals to shunt the distal iliofemoral venous system), chronic leg pain, phlebitis, lipodermatosclerosis and recurrent skin ulcers [5,8].

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**Literature Review**

**Prevalence**

May-Thurner syndrome is a rare condition whereby the left common iliac vein is compressed in between an overriding right common iliac artery and the posterior vertebral spine leading to DVT and or chronic venous hypertension. The true prevalence of this anatomical variant is unknown, but quoted to be as high as 22% based on autopsy series [2,3]. Despite this, the syndrome remains under-recognized, mainly because a small percentage of those with DVT have this anatomical variant (2-3%) [4]. It is seen predominantly in women in the second to fourth decade of life, after prolonged immobilization, use of oral contraceptives or multiple pregnancies. Only 30% of those with May-Thurner syndrome are male.

**Fig 5-** Follow up ultrasound at 2 weeks with patent stent (arrow) within the left EIV extending into the CIV and normal phasic flow (arrowhead).
Kim et al. have described clinical stages of iliac vein compression. Stage I is asymptomatic iliac vein compression. Stage II is development of venous spur. Stage III is development of left iliac vein DVT. Patients with DVT in the past are at increased risk for developing recurrent DVT. It has also been noted that patients with DVT and who have spur formation in the vein had a 72% of recurrence despite treatment with anticoagulation. Although DVT may be common in May-Thurner syndrome, very few patients progress to large pulmonary embolism. One study found an inverse correlation between the severity of stenosis and symptomatic pulmonary embolism in these patients [9]. This may be due to a “protective” effect of luminal narrowing from spur formation and possible trapping of large emboli that may otherwise go to the lungs [10].

**Diagnosis**

The diagnosis of May-Thurner is based on presence of DVT and subsequent confirmation of the anatomy of the iliac arteries and veins. This syndrome should be suspected in patients who present with recurrent DVT without another apparent etiology or have extensive DVT on the left side. The presence of DVT can be confirmed non-invasively with lower extremity ultrasound. With regards to confirming venous compression, there have been various proposed methods such as measuring pressure differentials between the two iliac veins at rest (difference of 2 mmHg) and with exercise (difference of 3 mmHg). Others have utilized inferior vena cava pressures as a surrogate for contra lateral iliac vein pressure. Imaging modalities are increasingly used to make the diagnosis. While lower extremity vascular ultrasound will confirm presence of DVT, it will not provide information on the anatomical position of the iliac arteries and veins or presence of spurs. Abdominal helical CT and MRI venography, and intravenous ultrasound have been utilized for diagnosis of DVT and delineation of venous anatomy consistent with May-Thurner syndrome with good yield compared to the gold standard of traditional invasive venography. MRI can also provide accurate estimation of collateral blood flow. Intravascular ultrasound plays an important role in May-Thurner because it has the ability to assess luminal diameter and presence of spurs and aid in endovascular intervention. One must keep in mind that there may be significant variability in the degree of venous compression noted in these imaging modalities in the same patient depending on volume status. However, in true May-Thurner, the vein should remain narrowed regardless of volume status and patient position on serial imaging [11]. CT and MRI venography are also useful to evaluate for alternative etiologies of DVT (tumor, lymphadenopathy and hematoma) [8].

**Treatment**

The goal of treatment in May-Thurner syndrome is to reduce the incidence of post-thrombotic syndrome (PTS) by eliminating obstructing thrombus and correcting the precipitating factor. Most studies evaluating treatment for iliac vein compression syndrome are either retrospective or observational [5,12]. Anticoagulation with unfractionated heparin, low molecular heparin and warfarin is the mainstay of treatment for patients with DVT to reduce propagation of thrombus and prevent recurrence. If early clot lysis can be achieved with catheter directed thrombolysis or mechanical thrombectomy, the incidence of PTS can be reduced [13-15]. Despite the success of these measures in routine iliofemoral DVT, clot lysis therapy is inadequate for patients with May-Thurner syndrome [14,16, 17].

Surgical treatments to relieve the obstruction include a variety of novel techniques that include creation of a bypass (the Palma cross over), iliac vein patch angioplasty and relocation of the artery behind the vein [10,18]. These procedures incur a high morbidity and patients still need long-term anticoagulation. With the improvement in percutaneous endovascular techniques, balloon angioplasty and stenting have become the standard of care [10,17]. An endovascular approach reduces morbidity and permits assessment of stenosis characteristics and collateral circulation during the procedure. In cases of large thrombus burden mechanical and catheter directed thrombolysis (CDT) are also be employed to improve clinical symptoms and vessel patency [19].

First described by erger, a stent placement for iliofemoral stenosis is now being used routinely [14,17,20]. Isolated balloon angioplasty and or CDT alone has a high re-occlusion rate (100% in one study)[1] suggesting it should be supplemented with stent placement to improve patency. The technical success of the endovascular approach is upwards of 96% with a 1 year primary and secondary patency rate of 58-100% and 76-98% [1,21,22]. The durability, long length and flexibility of self-expanding stents make them the preferred stent compared to balloon expandable stents in the treatment of iliofemoral stenosis. The average stents used are 1 Ato 1mm Â± 2cm Wall stents. The stented area should cover the area of stenosis and if needed can be extended proximally into the IVC [1]. A tenting Â± the IVC has shown to be safe with no increased risk of contralateral iliac vein occlusion [23]. When successfully treated, patients experience some immediate relief in their symptoms that may not be reflected in the conventional hemodynamic measurements [1,23]. In the absence of randomized trials, expert opinion recommends 3-6 months of anticoagulation followed by lifelong antiplatelet therapy if no evidence of hypercoagulability is found.
**Conclusion**

May-Thurner syndrome is an underappreciated cause of DVT and chronic venous insufficiency. The syndrome should be suspected in all young and middle aged patients who present with left sided ilio-femoral DVT or isolated left sided chronic venous disease. We advocate an aggressive approach in appropriate candidates utilizing catheter directed thrombolysis, and mechanical thrombectomy followed by angioplasty and stenting of the common iliac vein. Although surgical interventions are available, there is significant more risk associated with surgical repair compared to endovascular techniques.

**References**


