

Venous thromboembolism in a patient with an uncommon etiology of May-Thurner syndrome

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ABSTRACT

May-Thurner syndrome (MTS) consists of common iliac vein compression from an extrinsic source. Patients with MTS can present with a variety of symptoms, potentially making the diagnosis difficult. Classically, MTS will result in left iliac vein compression from the right iliac artery. In rare cases, it can be secondarily caused by compression from other anatomic structures in the pelvis. We present the case of a 43-year-old woman with MTS with iliofemoral deep vein thrombosis and pulmonary embolism caused by a large uterine leiomyoma. Our findings underscore the need to consider various etiologies of venous compression in patients with extensive unilateral venous thromboembolism. (*J Vasc Surg Cases Innov Tech* 2021;7:549-52.)

Keywords: May-Thurner syndrome; Uterine leiomyoma; Venous thromboembolism

Classic May-Thurner syndrome (MTS) results in compression of the left common iliac vein (LCIV) between the right common iliac artery (RCIA) and the fifth lumbar vertebral body. This compression causes endovenous fibrosis, which can lead to an acute thrombotic event or long-term symptoms associated with chronic venous hypertension. MTS can cause symptoms ranging from mild left leg edema and discomfort to chronic skin changes to venous thromboembolism (VTE). MTS is present in ~14% to 32% of the general population, although it causes <5% of all lower extremity VTEs.^{1,2} The risk of VTE increases in patients with prothrombotic risk factors such as pregnancy, genetic hypercoagulability, or long periods of immobilization. MTS presents most frequently in women aged 20 to 40 years.^{1,2} We have presented a case of VTE in a patient with apparent MTS of an unusual etiology. The patient provided written informed consent for the report of her case.

CASE REPORT

A 43-year-old white woman had presented to the emergency department complaining of left leg pain and edema that had begun 3 days earlier. She reported chest pain but denied shortness of breath and had no other systemic symptoms. She had

no history of hypercoagulability but was taking oral contraceptive pills and reported current electronic cigarette use. Her examination findings were significant for left leg swelling with pain. She was tachycardic, with a heart rate of 120 bpm and an oxygen saturation of 95%. Her laboratory test results were remarkable for a creatinine of 1.4 mg/dL and a white blood cell count of $12.7 \times 10^9/L$. A D-dimer level was not obtained, because her pretest probability for pulmonary embolus was high. A chest computed tomography angiogram indicated the presence of bilateral pulmonary emboli extending to the subsegmental branches in all lobes without evidence of pulmonary infarction or right heart strain. Lower extremity duplex ultrasound revealed extensive thrombosis in the left common femoral, femoral, popliteal, peroneal, and posterior tibial veins (*Fig 1*). Heparin was started, and she was admitted for further evaluation.

A diagnostic venogram demonstrated thrombus extending through the iliac veins into the inferior vena cava (IVC). She was deemed a candidate for iliofemoral lysis given her young age, presumed duration of the venous thrombosis, and severity of the ongoing symptoms. Given the presence of the significant pulmonary clot burden, we placed an IVC filter (IVCF) via an internal jugular approach and initiated lysis via a left popliteal approach. She underwent catheter-directed lysis for 24 hours, which resulted in resolution of the femoral and iliac vein thrombus. We interrogated the LCIV with intravascular ultrasound and discovered extrinsic LCIV compression, attributed to the RCIA, and intraluminal synechiae, suggesting acute on chronic thrombosis. We decided to place a 14-mm \times 12-cm VICI stent (Boston Scientific Corp, Marlborough, Mass), which was postdilated with a 12-mm balloon with good results (*Fig 2*). The IVCF was not removed because of a moderate amount of thrombus within the cone.

The patient was discharged in stable condition 5 days after the initial presentation with a 3-month course of apixaban (Eliquis) and instructions to stop taking the oral contraceptive pills pending follow-up. She was seen 2 weeks later, with a duplex ultrasound scan confirming the patency of the LCIV stent. During the examination, an astute ultrasound technician noted a 6.7-cm \times 7-cm vascular mass compressing the vein and stent

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(Fig 3). The physical examination demonstrated minimal leg edema and a nontender mass along the anterior abdominal wall. A contrast-enhanced computed tomography scan was performed 2 days later, which revealed a large multifibroid uterus with an exophytic 8.4-cm fibroid on the dome of the fundus that was compressing the LCIV and stent (Fig 4).

A follow-up ultrasound 3 months later demonstrated continued stent compression and patency. The patient received a gynecologic referral after the fibroid discovery and, ultimately, underwent complete hysterectomy without issue 8 months later. At that time, her IVCF was also removed. An ultrasound examination 10 months after the fibroid discovery and 2 months after the hysterectomy demonstrated stent patency without compression. The patient continued taking apixaban (Eliquis) for 7 months after the fibroid discovery without adverse effects.

DISCUSSION

Classic MTS is described as RCIA compression of the LCIV. Symptoms include swelling, pain, and VTE.³ In rare cases, however, pelvic or abdominal masses can exacerbate this preexisting compression or mimic it.^{4,5} The incidence of MTS is two times greater in women than in men, which might suggest the female pelvic anatomy as a complicating or exacerbating factor.^{1,6}

Traditional venography (with or without intravascular ultrasound) often cannot differentiate the sources of extrinsic compression. Differentiation with duplex ultrasound is operator dependent and can be challenging. Duplex ultrasound has relatively low sensitivity and should not be relied on solely to rule out MTS or other forms of iliac vein compression. Computed tomography or magnetic resonance venography are generally the preferred modalities for identifying the sources of external anatomic compression in MTS.⁷

The present patient had developed MTS secondary to compression from a large uterine fibroid. Therefore, we propose conceptualizing the etiology of ambiguous cases such as the present case as either primary MTS, with LCIV compression caused solely by the RCIA, or secondary MTS, with LCIV compression caused by pelvic or abdominal structures, with or without involvement of the RCIA.

The American Vein and Lymphatic Society developed the symptoms, varices, and pathophysiology classification to clarify and standardize the description of pelvic venous disease. Although a nonthrombotic etiology of pelvic venous obstruction is included in the classification, it does not differentiate between vein wall degeneration, a proximal luminal obstruction, or various sources of extrinsic compression—a distinction with important implications for management. We hope that our etiologic conceptualization can complement the symptom, varices, and pathophysiology system in the classification of MTS.⁸

Park et al⁹ recently studied MTS variants, including what we have referred to as secondary MTS, and right

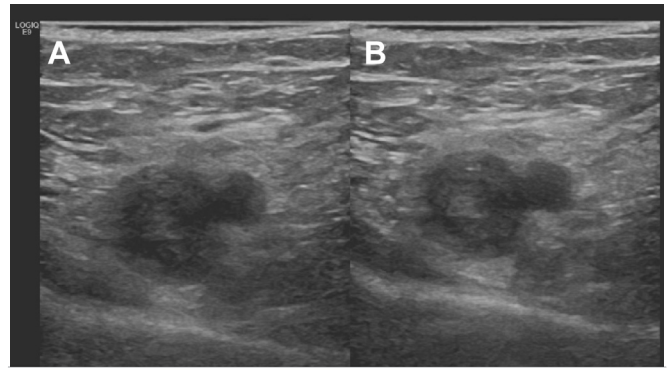


Fig 1. Lower extremity duplex ultrasound from day of admission demonstrating hyperechoic thrombus in the lumen of the left common femoral vein (A) and lack of compressibility (B).

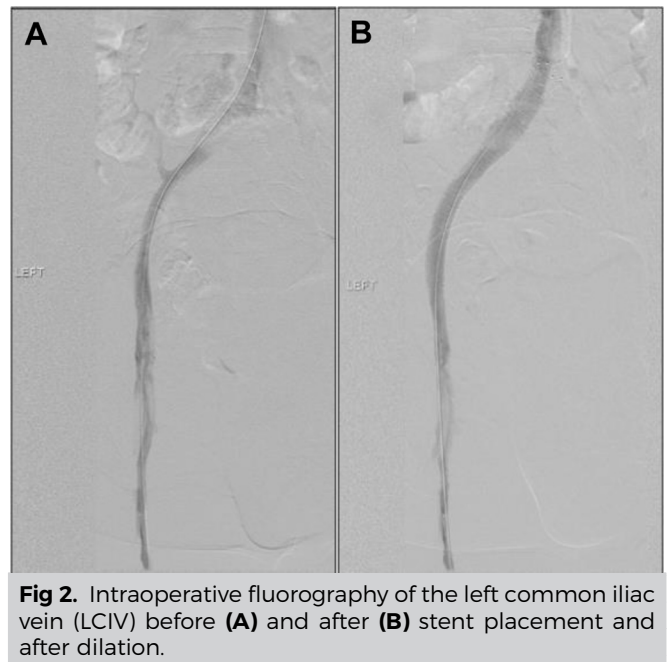


Fig 2. Intraoperative fluorography of the left common iliac vein (LCIV) before (A) and after (B) stent placement and after dilation.

common iliac vein compression. They noted that these MTS variants occurred in ~6% of patients with symptomatic MTS and that these patients will usually be elderly, which they attributed to the aging process's effects on the vasculature and nearby structures.⁹

Although an association between uterine fibroids and VTE is well-documented in the literature, few studies have considered this relationship in the context of secondary MTS from benign gynecologic etiologies.¹⁰⁻¹⁶ Also, standardized guidelines for the long-term treatment of these patients are lacking. Suggestions have included long-term anticoagulation and gynecology referral for potential hysterectomy or myomectomy.^{17,18} Uterine artery embolization is also an option, although this can be associated with a higher risk of fibroid recurrence than hysterectomy or myomectomy and also

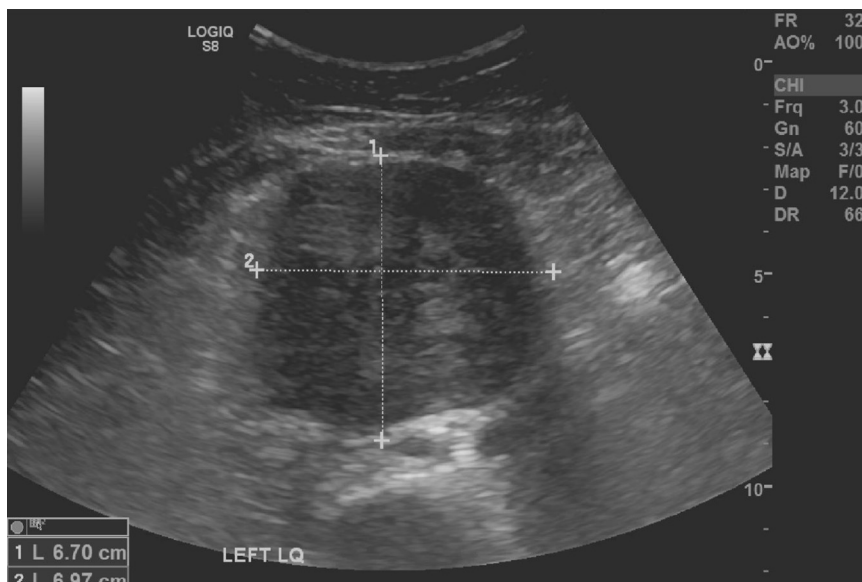


Fig 3. Abdominal duplex ultrasound demonstrating a large vascular mass in the left lower quadrant.

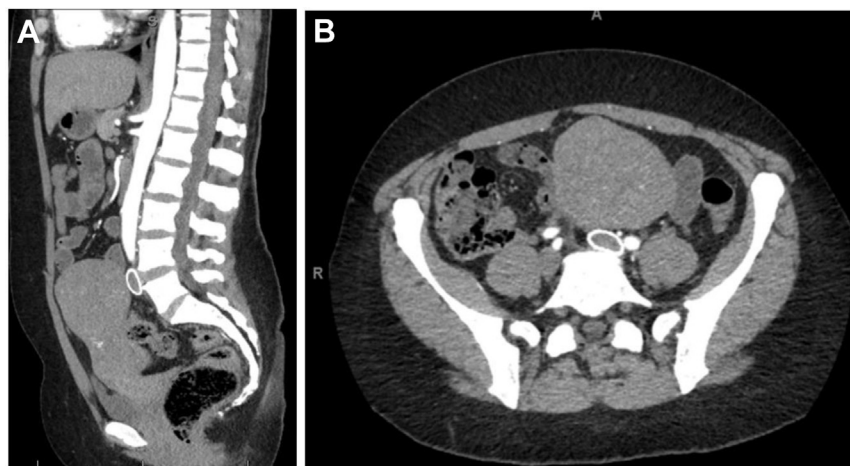


Fig 4. Sagittal (A) and axial (B) arterial phase contrast-enhanced computed tomography scans demonstrating compression of the left common iliac vein (LCIV) stent between a uterine fibroid and the lumbar vertebrae.

carries the risk of postembolization syndrome.¹⁹ Patients with persistent symptoms should undergo evaluation of the left iliac vein and might require stenting.

CONCLUSIONS

We have described the case of a patient with secondary MTS from a uterine fibroid. Our findings reinforce the importance of a thorough search for a possible cause of venous compression when a patient presents with an extensive, unilateral VTE.

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