May-Thurner Syndrome in a Geriatric Patient

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Case Report

A 79-year-old male presented with worsening asymmetric leg swelling, worse on the left. His has end-stage renal disease on hemodialysis with chronic venous insufficiency of bilateral lower extremities and chronic edema despite prior venous stripping surgery. Other medical problems include hypertension, diastolic dysfunction and monoclonal gammopathy of undetermined significance (MGUS).

He reported chronic leg swelling for years but his leg swelling had increased from its baseline over the last several weeks to months. His edema did not improve despite hemodialysis three times weekly for volume overload with fluid removal, furosemide 20mg daily, compression stockings and leg elevation. He reported decreased mobility due to gait and balance problems. He had no associated shortness of breath, orthopnea, chest pain, abdominal pain or abdominal distension. His chronic medications included aspirin 81mg daily, furosemide 20mg daily, amlodipine 5mg daily, atorvastatin and propranolol.

On exam, his vitals were normal. His weight was unchanged. Cardiovascular exam was unremarkable with regular rate and rhythm, no murmur or gallops. Jugular venous pressure was normal. Lungs were clear and abdomen was non-distended, non-tender, and without hepatosplenomegaly. Exam was notable for 1+ pitting edema on the right lower extremity and 2+ pitting edema on the left lower extremity. He has no tenderness upon palpitation of both hyperpigmentation distal legs.

Labs showed mild anemia with hemoglobin 10.4, normal liver chemistries including enzymes and albumin 4.7, and normal brain-naturietic peptide of 84. Recent TSH was within normal limits.

Ultrasound of bilateral lower extremities revealed no evidence of deep venous thrombosis or venous insufficiency. Thoracic echocardiogram showed normal left ventricular ejection fraction of 60-65%, grade 1 diastolic function with normal left ventricular size, and mild concentric left ventricular hypertrophy. An MRI pelvic venogram with and without contrast showed moderate narrowing (approximately 65%) of the left common iliac vein due to external compression from the right common iliac artery, suggestive of May-Thurner syndrome; no intraluminal thrombus was identified.

The patient was referred to a vascular specialist and CT venogram of abdomen and pelvis with and without contrast showed persistent moderate compression of a patent left common iliac vein by the right common iliac artery (May Thurner anatomy), with a venous diameter of 4 mm in this region. Bilateral iliac veins were widely patent and iliac vein stent placement was recommended.

Discussion

May-Thurner Syndrome (MTS), also called iliac vein compression syndrome and Crockett syndrome, should be considered in the differential diagnosis of symptomatic patients with unexplained unilateral leg swelling.1-3 It is an underdiagnosed condition in which venous outflow obstruction occurs from extrinsic venous compression by the arterial system against the bony structures in the iliocaval territory.3,4 MTS is increasingly recognized as a cause of chronic venous insufficiency, and a precipitating factor for DVT.2,5 The most common variant of MTS is when the left common iliac vein is compressed against the lumbar spine by the overlying, right common femoral artery.3,4 The syndrome was initially described by May and Thurner in 1957 who noted that the chronic pulsation of the overlying, right common iliac artery on the left common iliac vein leads to accumulation of elastin and collagen in the vein,6 causing the formation of "spurs" (fibrous bands) within the vein and leading to the development of deep vein thrombosis (DVT).3,4 Other variants MTS include right-sided MTS, in which the right common iliac vein is compressed by the left common iliac artery in association with a congenitally left sided inferior vena cava (IVC),2 and compression of the IVC by the right common iliac artery. Other variants of MTS include iliac vein compression due to pelvic mass (tumor, abscess, or hematoma), uterine enlargement (from fibroids, cancer or pregnancy), aneurysms, vascular tortuosity, and prominent osteophyte of the lumbar spine.5 Iliac vein compression can be thrombotic, combined with acute or chronic deep vein thrombosis (DVT), or non-thrombotic.1,3

The exact prevalence of MTS is unknown but likely underestimated, since patients with MTS anatomy typically are asymptomatic.3 A retrospective study of 50 patients reported 24% prevalence of MTS in an asymptomatic population evaluated by computed tomography.3 It is estimated to be present in 2–5% of patients with venous disease and, in those with DVT, MTS can be present in 18–49%.3 It is also 3 to 8
times more common on the left side. In some studies, asymptomatic iliac vein compression is considered a normal variant rather than a pathologic finding; however, symptoms could develop if the degree of compression is severe.

Risk factors for MTS include female gender (especially those who are postpartum or multiparous), use of oral contraceptives, presence of spinal abnormalities such as scoliosis, hypercoagulability, and prior aortoiliac vascular stent placement. MTS is at least twice as frequent in females than in males. The higher prevalence in females is thought to be related to the female pelvic anatomy in which there is an accentuated lumbar lordosis that pushes the lower lumbar vertebrae anteriorly and, thus, compressing the left common iliac vein against the right common iliac artery. Classic MTS is more common in younger female patients, whereas atypical iliac vein compression is found in the elderly population. A single-center retrospective study by Park reported median age of 76 in patients with atypical iliac vein compression. The younger age at presentation of females compared to males in atypical MTS is attributed to increased hypercoagulable risk with pregnancy and use of hormone replacement therapy. In contrast, the elderly age associated with atypical iliac vein compression is attributed to the aging process, atherosclerosis, and complex comorbidities, such as aneurysmal changes of vessels, vascular tortuosity, and prominent osteophyte of the lumbar spine.

With partial venous obstruction, MTS can be asymptomatic. However, with increased extrinsic venous compression, venous hypertension develops and leads to symptoms. These symptoms can include leg swelling, leg pain, heaviness, venous claudication, hyperpigmentation, and varicosities. Leg swelling is the most common symptom, especially on the left. However, there has been cases of MTS with right-sided and bilateral lower extremity edema. DVT is another common presentation of MTS, in particular iliofemoral DVT. Other presentations of MTS include recurrent superficial vein thrombosis, moderate-to-severe chronic venous insufficiency, and venous ulcers. Rarely MTS can present with pulmonary embolism, ruptured iliac vein, retroperitoneal hematoma from ruptured pelvic varix, acquired MTS due to iliac artery stent or endovascular stent-graft, prostate enlargement, pelvic congestion syndrome, and gastrointestinal bleed from congested rectal varices.

Initial evaluation of patients suspected of having MTS should include color doppler ultrasound to rule out DVT and identify iliac vein stenosis. However, transabdominal doppler ultrasound has low sensitivity in assessing venous compression due to the iliac veins' deep location in the pelvis. CT or MRI venography is recommended due to their non-invasiveness. Although catheter-based contrast venography is considered the definitive diagnostic test for MTS, it is invasive and usually not done unless if there is diagnostic uncertainty or plan for thrombolytic treatment. Intravascular ultrasound (IVUS) is another diagnostic option that is highly sensitive and specific but it is invasive and is used with catheter-based venography at time of anticipated intervention. CT and MR venograms have greater than 95% sensitivity and specificity for diagnosing MTS. CT venography may be better at identifying more severely stenotic iliac vein lesions, and has the advantage of identifying other causes of extrinsic venous compression, such as pelvic tumors. However, a non-diagnostic result on CT venogram can occur if there are technical issues, such as suboptimal contrast opacification of the target vein. MR venography provides better imaging of the pelvic and spinal structures including lumbar vertebral degeneration, bulging or protruding intervertebral discs, osteophytes, or spondylolisthesis. MR venography may overcome the contrast timing limitation of CT scanning, but cannot be performed in patients with metallic implants. Use of CT or MR venography may be limited because of cost.

Treatment of MTS depends on severity of symptoms, presence or absence of a DVT, and the patient's life expectancy, comorbidities and preferences. Since 50% or greater iliac stenosis may be present in one-quarter to one-third of the general population, it is important that anatomic stenosis is not the only criterion for intervention; any measurement of stenosis should be assessed in the context of the patient's symptoms. Those with non-thrombotic MTS with no or mild symptoms, conservative treatment such as compression stockings is recommended. In non-thrombotic MTS patients with moderate to severe symptoms, endovascular therapy with stenting is the primary treatment. Angioplasty and stenting of the affected venous lesion is recommended (rather than angioplasty alone) to improve outflow obstruction since angioplasty alone is associated with recurrent stenosis. In thrombotic MTS without contraindications to lytic therapy, endovascular treatment can reduce the rate of post-thrombotic syndrome. In thrombotic MTS with contraindications to lytic therapy, catheter-directed thrombolysis including suction thrombectomy, or open surgical thrombectomy can be considered.

Although MTS is more common in young females, it was discovered in our geriatric male patient. MTS, a pelvic vein disorder, should be considered in the differential diagnosis when an elderly patient presents with worsening, unexplained leg swelling with no evidence of DVT.

**REFERENCES**


