A rare case of May-Thurner syndrome due to external compression of the right and left common iliac veins

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ABSTRACT

May-Thurner syndrome, also known as external iliac compression syndrome, is a rare but commonly underdiagnosed cause of asymmetric lower extremity edema. Here we describe a case of May-Thurner syndrome owing to external compression of the right and left common iliac veins presenting as chronic worsening asymmetric right greater than left lower extremity edema. Initial etiology workup was unremarkable, and further diagnostics revealed compression of the right common iliac vein at the bifurcation of the right common iliac artery between the right external and internal iliac arteries with concomitant compression of the left common iliac vein. Stenting of the right common iliac vein was completed, with significant symptomatic improvement at 30-day follow-up. This case documents a unique variant of May-Thurner syndrome rarely described in the literature. (J Vasc Surg Cases Innov Tech 2025;11:101658.)

Keywords: May-Thurner syndrome; Unilateral edema; Iliac vein compression

May-Thurner syndrome, a type of iliac compression syndrome, is a commonly underdiagnosed etiology of asymmetric lower extremity edema.[1-3] May-Thurner syndrome involves compression of the left common iliac vein (LCIV) by the right common iliac artery (RCIA) and the lumbar spine.[2,3] Although most cases of iliac compression anatomical abnormalities are asymptomatic, May-Thurner syndrome commonly presents as asymmetric left lower extremity edema and a significantly increased risk for left lower extremity deep vein thrombosis (DVT).[2-4] The worsening degree of stenosis has been shown to cause increasingly elevated risks of DVT formation.[3,4] Here we present a unique case of May-Thurner syndrome causing asymmetric right worse than left lower extremity edema with diagnostic imaging and successful management.

CASE REPORT

A 54-year-old man with hypertension, chronic kidney disease (CKD) stage IIIa, compensated hepatitis B cirrhosis, and coarctation of the aorta status post aortic stenting with no residual complications, presented with chronic worsening bilateral right greater than left lower extremity edema. He was limited to ambulating two blocks before experiencing significant bilateral leg pain prohibiting further ambulation. Vital signs were within normal limits. Cardiac examination was significant for regular rate and rhythm with normal S1 and S2 sounds and no murmurs, S3, or S4, present. Pulmonary examination was clear to auscultation bilaterally and normal work of breathing. There was no hepatojugular reflux present. There was significant asymmetric lower extremity edema with 4+ right lower extremity edema and 2+ left lower extremity edema to the mid-thighs. The preprocedural Clinical Etiological Anatomical Pathological clinical class was C4a. Electrocardiography revealed sinus rhythm with old inferior myocardial infarction and left axis deviation. Medical therapy with increasing titration of diuretics was unsuccessful in mitigating progression of his asymmetric lower extremity edema. The initial differential diagnosis included DVT, decompensated heart failure, decompensation of previously controlled hepatitis B cirrhosis, lymphedema, and mass effect from malignancy. Laboratory assessment disclosed stable CKD stage IIIa, normal hepatic function and no other significant findings. A transthoracic echocardiogram showed a left ventricle ejection fraction of 55% to 60% with normal right ventricle size and function, a normal gradient across his previously stented aortic coarctation, and no significant valve abnormalities. Bilateral duplex ultrasound examinations were performed showing no evidence of DVT in the left or right lower extremity veins. Reflux testing was not performed. Computed tomography (CT) abdomen and pelvis showed liver surface nodularity suggestive of cirrhosis, no significant bilateral hydronephrosis, no iliac vein compression and no evidence of compressive masses. CT venogram was not performed given his CKD stage IIIa to reduce his contrast exposure. Given the unclear etiology of the chronic progressive bilateral, right greater than left lower extremity edema, the patient was referred for right heart catheterization. Right heart catheterization revealed a right atrial pressure of 13 mm Hg, right ventricle pressure of 45/12 mm Hg, pulmonary artery pressure of 40/13 mm Hg (mean 27 mm Hg), and pulmonary capillary wedge pressure of 12 mm Hg, consistent with normal right and left ventricular filling pressures. Bilateral peripheral venogram was performed showing bilateral common iliac vein stenosis (Fig, *A*). Intravascular ultrasound (IVUS)

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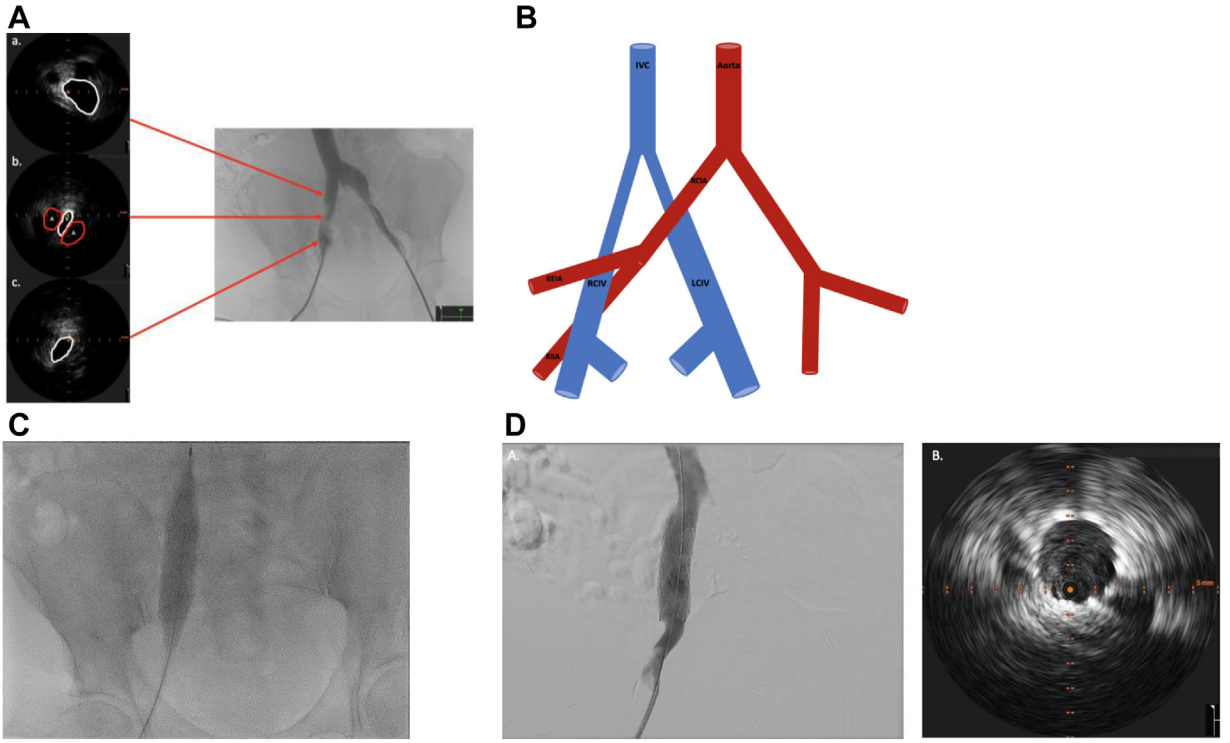

Fig. (A) Preprocedure peripheral venogram and intravascular ultrasound (IVUS) examination: Bilateral iliocavogram of bilateral internal iliac veins demonstrating bilateral common iliac vein stenosis. IVUS of the right common iliac vein showing segments cranial to stenosis **(A-A)**, segment with maximal stenosis **(A-B)** with compression of the vein (*V*) between the internal and external iliac arteries **(A-A)** and a segment caudal to the stenosis **(A-C)**. **(B)** Example compression diagram. A diagram demonstrating the right common iliac artery (*RCIA*) compression of the left common iliac vein (*LCIV*) and the right common iliac vein (*RCIV*) compressed between the right external and internal iliac arteries. **(C)** Balloon dilation of the RCIV. Poststent balloon dilation using an 18 × 40 mm Armada 35 Balloon Dilation Catheter (Abbott Vascular, Santa Clara, CA) at 6 atm of the right common iliac vein. **(D)** Postprocedure peripheral venogram and intravascular ultrasound (IVUS) examination. **(D-A)** Digital subtraction angiography of the right common iliac vein after the 18 × 60 mm Abre stent deployment and balloon dilation demonstrating resolution of the stenotic segment. **(D-B)** IVUS image demonstrating appropriate stent deployment and expansion with resolution of the right common iliac vein stenosis.

examination showed external compression of the right common iliac vein (RCIV) between the right external and internal iliac arteries with concomitant external compression of the LCIV by the RCIA against the lumbar spine (Fig, *A*). A diagram of the compression observed in this case is included (Fig, *B*). On catheter pullback, the LCIV pressure was 15 mm Hg, the RCIV pressure just caudal to the stenosis was 30 mm Hg, and the IVC pressure was 15 mm Hg. The pressure gradient across the RCIV stenosis, between the origin of the RCIV and the IVC, was 15 mm Hg with no significant gradient across the LCIV. IVUS examination demonstrated the luminal area of the RCIV lesion to be 0.57 cm^2. Given the patient's CKD, the decision was made to pursue staged intervention of the RCIV first because this side had worse symptoms and gradient. The RCIV was stented using an 18 × 60 mm Abre Venous Self-Expanding Stent (Medtronic, Inc, Minneapolis, MN) followed by dilation using an 18 × 40 mm Armada 35 Balloon Dilation Catheter (Abbott Vascular, Santa Clara, CA) at 6 atm (Fig, *C*). Poststent IVUS examination showed improved lesion luminal area of 1.97 cm^2. Final IVUS examination and venogram showed no residual stenosis, edge dissection or perforation (Fig, *D*). The patient was discharged on dual antiplatelet therapy for 3 months and aspirin therapy thereafter, along with continuation of an aggressive diuretic regimen. The patient reported a significant reduction in bilateral lower extremity edema with marked improvement in ambulation capacity and no further lower extremity asymmetry at the 30-day and 60-day follow-up visits. The postprocedure Clinical Etiological Anatomical Pathological clinical class was C2a. He continues to be followed in our outpatient cardiology clinic.

DISCUSSION

In 1851, Virchow described a roughly five times increased incidence of DVT in the left compared with the right leg, which he hypothesized was due to the RCIA compressing the LCIV.[5] It was not until 1957 when May and Thurner studied cadavers finding vascular thickening of the left iliofemoral venous wall where the RCIA

intersected the LCIV that May-Thurner syndrome was first described.[5] Since then, May-Thurner syndrome remains an underdiagnosed disease in patients with asymmetric lower extremity edema, with a prevalence of between 2% and 5% percent of isolated lower extremity venous disorders.[5-7] Iliac compression syndrome is often discovered after negative diagnostic imaging for more common etiologies, such as DVT or lymphedema and failed diuretic therapy, thus suggesting an alternative cause for the asymmetric lower extremity edema. Neither DVT nor lymphedema were present in our patient. Thus, it is important to maintain a broad differential diagnosis for patients with persistent asymmetric lower extremity edema despite aggressive medical management, as highlighted in this case as well. There remains extensive variability in the location and extent of compression of the LCIV; however, most cases occur in women presenting with unilateral left greater than right lower extremity edema owing to compression of the LCIV by the RCIA.[6-10] Furthermore, there is evidence to suggest the possibility of a normal anatomical variant of LCIV compression by the RCIA, as well in asymptomatic individuals.[10,11] It is exceedingly rare for compression of both the LCIV and RCIV by the RCIA against the lumbar vertebrae on the left and between the internal and external iliac arteries on the right, which makes this case unique.[9] Despite the presence of compression of the LCIV on IVUS examination, it is unclear whether this compression is contributing to the patient's left lower extremity edema given the lack of gradient; thus, no stenting was performed in the LCIV.

Jayaraj et al[12] described contralateral limb swelling owing to excess collateral load on the opposite limb circulation as a possible etiology of contralateral leg improvement with ipsilateral stenting. This may help to explain the improvement in the left lower extremity edema after stenting of the RCIV, which resolved this obstruction and removed the excess collateral load on the left limb circulation. The initial plan for a staged procedure to return for stenting of the LCIV was forgone after the patient experienced resolution of his symptoms, further supporting a staged approach to iliofemoral stenting.[12] Unfortunately, routine gradients are not performed typically; thus, it is difficult to determine the significance of the compression of the LCIV in this case definitively. It is possible that the left lower extremity edema may have been associated with the patient's other comorbidities, including renal dysfunction or cirrhosis, which cannot be excluded as a possible etiology given the lack of gradient. IVUS examination is the standard method to diagnose chronic iliac venous obstructions.[13] However, noninvasive diagnostic studies including cross-sectional imaging with computed tomography and magnetic resonance venography can help to guide decision-making when chronic iliac venous obstruction is suspected.[7,9,12-16] The mainstay of treatment for patients who have failed conservative therapy is endovascular stenting.[7,9,13,16]

CONCLUSIONS

In this article, we present a rare case of May-Thurner syndrome via external compression of the LCIV by the RCIA and the RCIV by the right internal and external iliac arteries. We provide a successful management approach using multimodality imaging and successful stenting of the RCIV resulting in marked symptomatic improvement for our patient.

PATIENT CONSENT

The patient consented to publication of the case.

DISCLOSURES

None.

REFERENCES

1. Cockett FB, Thomas ML. The iliac compression syndrome. *Br J Surg*. 1965;52:816–821.
2. Harbin MM, Lutsey PL. May-Thurner syndrome: history of understanding and need for defining population prevalence. *J Thromb Haemostasis*. 2020;18:534–542.
3. Poyyamoli S, Mehta P, Cherian M, et al. May-Thurner syndrome. *Cardiovasc Diagn Ther*. 2021;11:1104.
4. Carr S, Chan K, Rosenberg J, et al. Correlation of the diameter of the left common iliac vein with the risk of lower-extremity deep venous thrombosis. *J Vasc Intervent Radiol*. 2012;23:1467–1472.
5. Virchow R. Uber die erweiterung kleiner gefasse. *Arch Path Anat*. 1851;3:427–462.
6. May R, Thurner J. The cause of the predominantly sinistral occurrence of thrombosis of the pelvic veins. *Angiology*. 1957;8:419–427.
7. Mousa AY, AbuRahma AF. May–Thurner syndrome: update and review. *Ann Vasc Surg*. 2013;27:984–995.
8. Brazeau NF, Harvey HB, Pinto EG, Deipolyi A, Hesketh RL, Oklu R. May-Thurner syndrome: diagnosis and management. *Vasa*. 2013;42:96–105.
9. McDermott S, Oliveira G, Ergül E, Brazeau N, Wicky S, Oklu R. May-Thurner syndrome: can it be diagnosed by a single MR venography study? *Diagn Interv Radiol*. 2013;19:44.
10. Nazzal M, El-Fedaly M, Kazan V, et al. Incidence and clinical significance of iliac vein compression. *Vascular*. 2015;23:337–343.
11. Molloy S, Jacob S, Buckenham T, Khaw KT, Taylor RS. Arterial compression of the right common iliac vein; an unusual anatomical variant. *Cardiovasc Surg*. 2002;10:291–292.
12. Jayaraj A, Noel C, Raju S. Contralateral limb improvement after unilateral iliac vein stenting argues against simultaneous bilateral stenting. *J Vasc Surg Venous and Lymphatic Disorders*. 2020;8:565–571.
13. Jayaraj A, Rossi FH, Lurie F, Muck P. Diagnosis of chronic iliac venous obstruction. *J Vasc Surg Venous Lymphat Disord*. 2024;12:101744.
14. Kibbe MR, Ujiki M, Goodwin AL, Eskandari M, Yao J, Matsumura J. Iliac vein compression in an asymptomatic patient population. *J Vasc Surg*. 2004;39:937–943.
15. Steinberg JB, Jacocks MA. May-Thurner syndrome: a previously unreported variant. *Ann Vasc Surg*. 1993;7:577–581.
16. D'Oria M, Zlatanovic P, Anthony A, et al. International union of angiology consensus document on vascular compression syndromes. *Int Angiol*. 2023;42:282–309.

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