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Beyond the Obvious Provoking Factor: May-Thurner Syndrome Presenting With Extensive Venous Thromboembolism in an 18-Year-Old Male

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Abstract

May-Thurner syndrome (MTS), also known as iliac vein compression syndrome or Cockett syndrome, is an anatomical vascular disorder characterized by chronic extrinsic compression of the left common iliac vein between the overlying right common iliac artery and the fifth lumbar vertebral body. Although MTS is a well-established cause of left-sided iliofemoral venous thrombosis, it is classically described in women between the second and fourth decades of life and remains underdiagnosed outside this population.

We present the case of an otherwise healthy 18-year-old male student with no history of surgery, no prior medical comorbidities, recent hospitalizations, surgical procedures, lower-extremity trauma, or long-distance travel, and no personal or family history of thromboembolic disease. However, he presented with extensive left lower-extremity deep venous thrombosis extending into the inferior vena cava, accompanied by bilateral pulmonary emboli. Comprehensive evaluation revealed severe compression of the left common iliac vein consistent with MTS as the primary underlying etiology.

Categories: Radiology, Internal Medicine, Cardiac/Thoracic/Vascular Surgery
Keywords: iliofemoral deep vein thrombosis, may-thurner's syndrome, pulmonary embolism (pe), venous compression syndromes, young adults

Introduction

May-Thurner syndrome (MTS), also known as iliac vein compression syndrome or Cockett syndrome, is an underrecognized vascular disorder resulting from chronic extrinsic compression of the left common iliac vein between the overlying right common iliac artery and the fifth lumbar vertebral body. Repetitive arterial pulsations exert chronic mechanical stress on the adjacent venous wall, leading to endothelial injury, intimal hyperplasia, collagen deposition, and the formation of intraluminal fibrous spurs. Over time, these structural changes progressively narrow the venous lumen, impair venous return from the left lower extremity, and create a prothrombotic environment characterized by venous stasis and endothelial dysfunction [1,2].

Despite these pathological changes, anatomical compression of the left common iliac vein is relatively common, occurring in up to 20-30% of the general population [1,3]. However, the vast majority of cases remain asymptomatic, as compression alone is often insufficient to produce clinically significant venous outflow obstruction or venous thromboembolism (VTE) without secondary prothrombotic factors [1,3].

The pathogenesis of symptomatic MTS is therefore best understood through a "two-hit" model. The first hit is the underlying anatomical predisposition created by chronic iliac vein compression. The second hit consists of an acquired prothrombotic trigger (infection, inflammation, dehydration, prolonged immobilization, trauma, surgery, pregnancy, estrogen exposure, or inherited thrombophilia) that shifts the hemostatic balance toward overt thrombosis [1,2,4]. When these factors converge, patients may present with acute iliofemoral deep venous thrombosis (DVT), chronic venous insufficiency, recurrent VTE, or, less commonly, life-threatening pulmonary embolism [1,2,4].

Although MTS is traditionally described in women between the second and fourth decades of life, increasing use of advanced venous imaging has revealed that clinically significant disease can occur across a broader demographic spectrum [1,5]. Nevertheless, extensive thromboembolic presentations in young males without established thrombophilic risk factors remain distinctly uncommon and may delay recognition of the underlying anatomical abnormality.

Because the left common iliac vein serves as the primary conduit for venous drainage from the entire left lower extremity to the inferior vena cava (IVC) and heart, its focal obstruction provides a direct mechanical

setup for extensive left-sided thrombus propagation and subsequent pulmonary embolism [1,2]. We report the case of an otherwise healthy 18-year-old male presenting with extensive left-sided iliofemoral and IVC thrombosis complicated by bilateral pulmonary emboli following a viral respiratory infection. Despite lacking conventional VTE risk factors, such as recent surgery, prolonged immobility, trauma, or active malignancy, the patient harbored underlying May-Thurner anatomy that served as the primary structural substrate for his severe presentation.

Case Presentation

An 18-year-old previously healthy male student presented with a one-day history of severe retrosternal chest pain and progressive shortness of breath. The pain was non-radiating and was not related to exertion or position. He denied hemoptysis, syncope, orthopnea, paroxysmal nocturnal dyspnea, preceding lower-extremity swelling, recent surgery, immobilization, long-distance travel, trauma, smoking, recreational drug use, or a personal or family history of thrombosis.

Two weeks prior to presentation, he was managed at an outside facility for presumed community-acquired pneumonia. He also recalled an isolated episode of chest and back pain several months earlier that had been similarly treated as a respiratory infection. However, because these episodes occurred at an external institution, primary clinical records, diagnostic imaging, and discharge summaries were unavailable for review. Consequently, whether these prior symptoms represented antecedent thromboembolic events or unrelated respiratory illnesses remains speculative.

On presentation, he appeared acutely unwell but was alert and oriented (GCS 15/15). His temperature was 37.6°C, heart rate was 140 beats/minute, respiratory rate was 23 breaths/minute, blood pressure was 121/78 mmHg, and oxygen saturation was 95% on room air. Cardiovascular examination revealed sinus tachycardia without murmurs, while respiratory examination demonstrated normal bilateral air entry without wheeze or crackles. There was no peripheral edema, clinical evidence of DVT, focal neurological deficit, or meningeal signs.

Laboratory investigations demonstrated leukocytosis with neutrophilia, markedly elevated inflammatory markers, and an elevated D-dimer level. Hemoglobin was preserved, while the platelet count was mildly reduced. The patient's laboratory findings and corresponding reference intervals are summarized in Table *1*.

Investigation	Patient result	Reference range
White blood cell count (WBC)	15.06 × 10^9/L	4.00-10.00 × 10^9/L
Red blood cell count (RBC)	4.53 × 10^{12}/L	4.50-5.50 × 10^{12}/L
Hemoglobin	13.40 g/dL	13.00-17.00 g/dL
Hematocrit	39.70%	40.00-50.00%
Mean corpuscular volume (MCV)	87.60 fL	83.00-101.00 fL
Mean corpuscular hemoglobin (MCH)	29.60 pg	27.00-32.00 pg
Mean corpuscular hemoglobin concentration (MCHC)	33.80 g/dL	31.50-34.50 g/dL
Red cell distribution width (RDW)	12.20%	11.60-14.00%
Platelet count	144 × 10^9/L	150-410 × 10^9/L
Neutrophils	74.50%	40.00-70.00%
Lymphocytes	12.20%	20.00-40.00%
Monocytes	12.00%	2.00-10.00%
Eosinophils	1.00%	1.00-6.00%
Basophils	0.30%	0.00-2.00%
Sodium	135.90 mmol/L	136.00-145.00 mmol/L
Potassium	3.69 mmol/L	3.50-5.10 mmol/L
Chloride	98.90 mmol/L	98.00-107.00 mmol/L
Bicarbonate	23.60 mmol/L	22.00-29.00 mmol/L
Blood urea nitrogen (BUN)	6.62 mmol/L	3.20-8.20 mmol/L
Creatinine	109 µmol/L	62.00-115.00 µmol/L
Estimated creatinine clearance	101 mL/min	90-130 ml/min
Estimated GFR	87 mL/min/1.73 m^2	≥90 mL/min/1.73 m^2
C-reactive protein (CRP)	187.00 mg/L	0.00-10.00 mg/L
D-dimer	4.09 µg/mL	<0.50 µg/mL FEU
Malaria parasites	Absent	
Blood culture	No growth	
Urine culture	No growth	
Coronavirus HKU1	Detected	

TABLE 1: Laboratory and microbiological investigations

WBC: white blood cell count; RBC: red blood cell count; MCV: mean corpuscular volume; MCH: mean corpuscular hemoglobin; MCHC: mean corpuscular hemoglobin concentration; RDW: red cell distribution width; BUN: blood urea nitrogen; eGFR: estimated glomerular filtration rate (CKD-EPI formula); eCC: estimated creatinine clearance (Cockcroft-Gault formula); CRP: C-reactive protein; FEU: fibrinogen equivalent units; PCR: polymerase chain reaction.

Although the positive viral PCR initially suggested an infectious etiology, the severity of symptoms, persistent tachycardia, and markedly elevated D-dimer prompted further evaluation for VTE. A chest radiograph demonstrated no evidence of pneumonia. Contrast-enhanced CT of the chest, abdomen, and pelvis revealed acute bilateral pulmonary emboli involving the left main pulmonary artery (Figure *1*), with extension into the lower lobe segmental and subsegmental branches, as well as segmental and subsegmental emboli within the right lower lobe pulmonary arteries (Figure *2*).

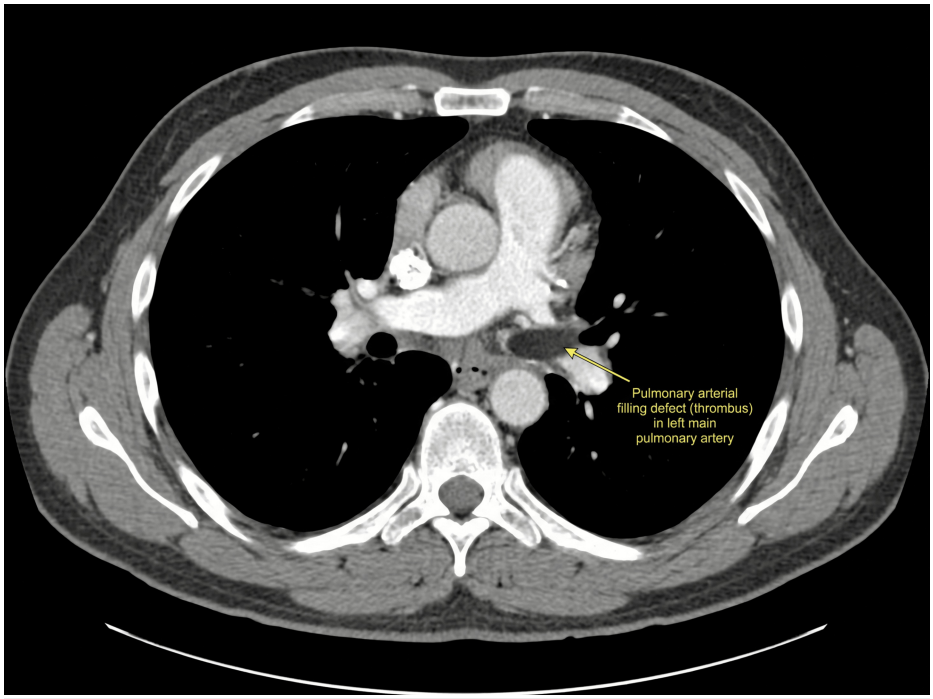


FIGURE 1: Axial contrast-enhanced CT pulmonary angiogram at the level of the main pulmonary artery bifurcation. Directly visualized findings demonstrate a distinct hypodense, non-enhancing intravascular filling defect within the lumen of the left main pulmonary artery (yellow arrow), extending into its lower lobe branch vessels. These findings confirm acute pulmonary arterial thrombosis (pulmonary embolism) complicating extensive lower-extremity deep venous thrombosis

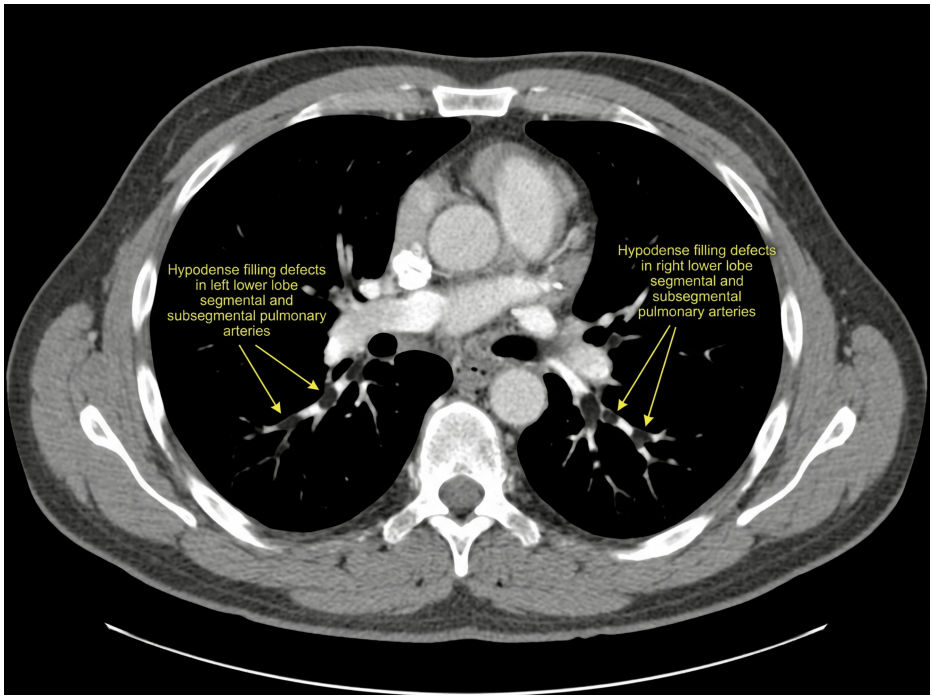


FIGURE 2: Axial contrast-enhanced CT pulmonary angiogram demonstrating bilateral lower lobe segmental and subsegmental pulmonary emboli. Directly visualized findings demonstrate hypodense, non-enhancing intravascular filling defects within the distal lower lobe branches bilaterally. Yellow arrows denote pulmonary arterial filling defects (thrombi) involving the left lower lobe segmental and subsegmental pulmonary arteries, as well as the right lower lobe segmental and subsegmental pulmonary arteries, confirming acute bilateral lower lobe pulmonary thromboembolism

Imaging demonstrated compression of the left common iliac vein between the overlying right common iliac artery and the lumbar vertebral body, consistent with MTS (Figure *3*).

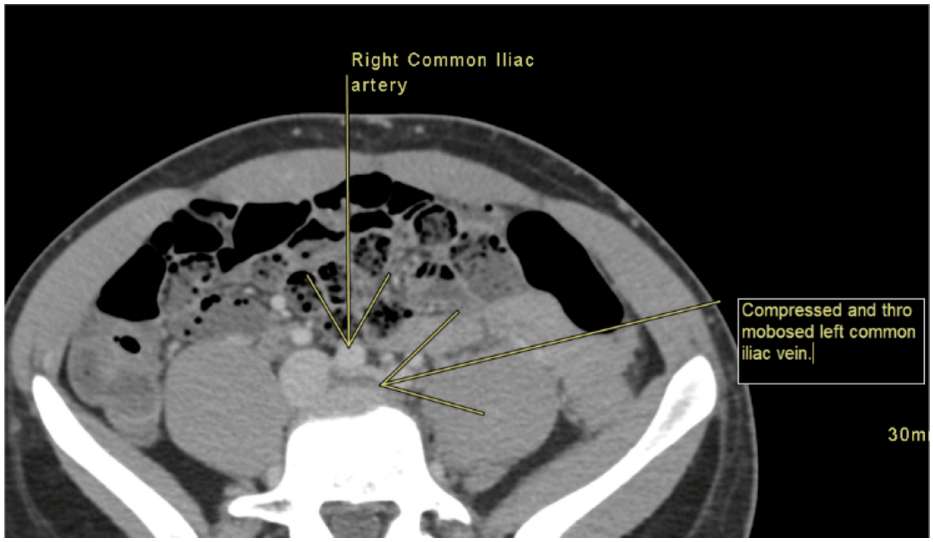


FIGURE 3: Axial contrast-enhanced CT venogram of the abdomen at the level of the lower lumbar vertebrae, demonstrating extrinsic compression of the left common iliac vein between the overlying right common iliac artery and the underlying lumbar vertebral body, with associated thrombosis of the compressed left common iliac vein. Appearances are in keeping with May-Thurner syndrome (MTS)

The sagittal reconstructed image showed the compressed and thrombosed left common iliac vein and the traversing right common iliac artery (Figure *4*).

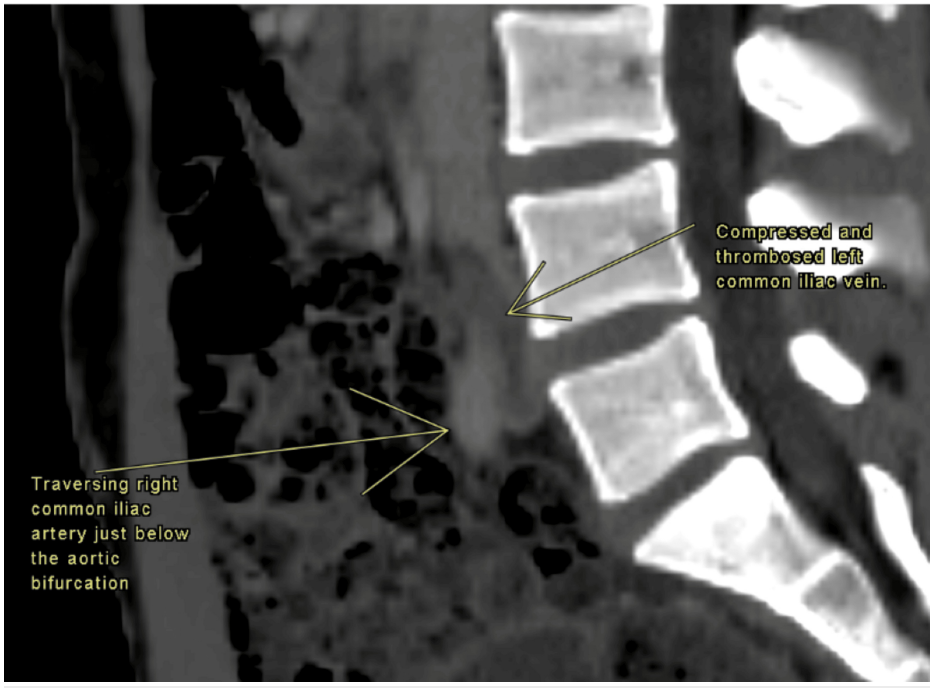


FIGURE 4: Sagittal reconstructed image just below the aortic bifurcation, demonstrating the compressed, thrombosed left common iliac vein and the traversing right common iliac artery

Extensive venous thrombosis extended from the distal IVC into the left external iliac vein (Figure *5*).



FIGURE 5: Axial contrast-enhanced CT through the mid-pelvis showing thrombus extending into the left external iliac vein, which appears expanded and hypodense compared to the normally opacified contralateral vessel (arrows)

Imaging also demonstrated venous thrombosis extending to the deep and superficial femoral veins (Figure *6*).

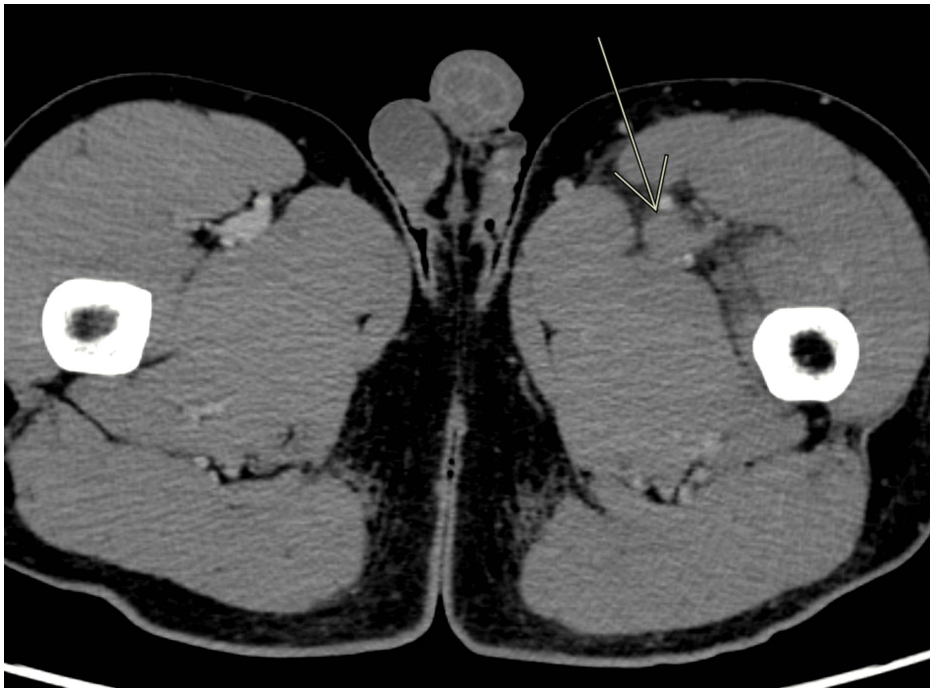


FIGURE 6: Axial contrast-enhanced CT of the proximal thighs showing hypodense, non-enhancing filling defects within the visualized left deep and superficial left femoral veins (arrow), consistent with distal extension of the thrombus

There was no evidence of right ventricular strain, and cardiac size was normal. No significant thoracic or abdominal lymphadenopathy was identified. Transthoracic echocardiography demonstrated preserved biventricular systolic function with mild pulmonary hypertension. Given the acute presentation and extensive thrombotic burden, thrombophilia testing was deferred until completion of the acute treatment phase.

Acute therapeutic anticoagulation was initiated with subcutaneous enoxaparin 1 mg/kg twice daily. Acute hypercoagulability testing, including lupus anticoagulant, protein C, protein S, and Factor V Leiden, was ordered but cancelled during admission due to active low-molecular-weight heparin administration interfering with assay validity. Outpatient autoimmune screening (ANA, anti-cardiolipin, and anti-beta-2-glycoprotein I antibodies) was ordered upon discharge. Vascular surgery evaluation recommended suction thrombectomy and advanced endovascular intervention abroad; however, this was financially unattainable for the family. Definitive iliac vein stenting was considered premature while extensive acute thrombus remained in situ due to the high risks of procedural failure and stent thrombosis. The patient was transitioned to oral apixaban 10 mg twice daily as a loading dose for seven days, followed by 5 mg twice daily for an intended duration of 3-6 months, with outpatient magnetic resonance (MR) venography planned to re-evaluate luminal patency prior to considering stenting. At a two-week post-discharge clinical evaluation, the patient demonstrated complete clinical symptom resolution; however, repeat imaging (venous duplex or CT) was not performed at that time.

Discussion

MTS is an underrecognized but clinically important cause of VTE resulting from chronic extrinsic compression of the left common iliac vein by the overlying right common iliac artery against the lumbar vertebral body. Although this anatomical variant has been reported in up to 20-30% of the general population, only a minority of affected individuals develop symptomatic disease. Consequently, the diagnosis of MTS should not be based solely on radiographic evidence of iliac vein compression but rather on the demonstration of clinically significant venous outflow obstruction, thrombosis, or manifestations of venous hypertension occurring in the appropriate anatomical context [1-3].

The pathophysiology of MTS extends beyond simple mechanical compression. Chronic arterial pulsations transmitted to the compressed venous wall induce repetitive endothelial injury, promoting intimal hyperplasia, collagen deposition, and the development of intraluminal fibrous spurs. These structural changes progressively narrow the venous lumen, impair venous return, and establish a local prothrombotic milieu characterized by venous stasis and endothelial dysfunction [1,2]. This mechanism provides a

biologically plausible explanation for the occurrence of extensive iliofemoral thrombosis in otherwise healthy individuals who lack traditional thromboembolic risk factors. Our patient exemplifies this phenomenon, developing extensive left-sided iliofemoral and IVC thrombosis with bilateral pulmonary emboli despite a low baseline thrombotic risk profile [1,2].

An important teaching point from this case is the interplay between anatomical predisposition and transient prothrombotic triggers. The detection of Coronavirus HKU1 via respiratory PCR provides a temporal link to an acute viral respiratory illness preceding the thrombotic presentation. While respiratory viral infections can induce transient systemic inflammation and hypercoagulability, a positive PCR result alone does not establish direct causality. Within the two-hit model of MTS, acute viral infection and inflammatory stress (evidenced by a markedly elevated CRP of 187 mg/L) are best understood as potential transient triggers that unmasked a pre-existing, hemodynamically silent iliac venous compression rather than acting as sole drivers of extensive thrombosis. This observation supports the two-hit model of MTS pathogenesis, in which chronic anatomical obstruction remains clinically silent until an acquired prothrombotic stimulus, such as infection, inflammation, dehydration, trauma, surgery, immobilization, or hormonal exposure, precipitates overt thrombosis [1,2,4]. Importantly, the identification of an apparent provoking factor should not prematurely terminate diagnostic evaluation when the thrombotic burden is unusually severe, anatomically localized, or disproportionate to the patient's overall risk profile [1,2,4].

The demographic characteristics of this patient further underscore the unusual nature of the presentation. MTS has traditionally been described in women between the second and fourth decades of life and is frequently associated with hormonal or pregnancy-related risk factors [1,5,6]. In contrast, our patient was an otherwise healthy 18-year-old male with no history of smoking, immobilization, recent surgery, malignancy, or known thrombophilia. Although increasing use of advanced venous imaging has broadened recognition of MTS across diverse populations, life-threatening VTE secondary to MTS in adolescent and young adult males remains uncommon [5]. This case therefore expands the clinical spectrum of MTS and highlights the need to maintain diagnostic suspicion regardless of age or sex when extensive left-sided proximal thrombosis is encountered.

Notably, the patient presented without lower-extremity edema despite extensive thrombosis extending from the popliteal vein to the IVC. Lower-extremity Doppler ultrasound explicitly confirmed the absence of subcutaneous edema. The absence of edema in severe proximal DVT can occur if thrombus propagation is acute without complete venous occlusion, or if non-visualized collateral venous pathways (such as ascending lumbar, transpubic, or presacral plexuses) provide sufficient temporary drainage. However, because cross-sectional imaging did not definitively delineate collateral networks, the precise hemodynamic mechanism remains unconfirmed. Additionally, while unilateral leg swelling is often considered a hallmark of proximal DVT, its absence does not exclude significant iliocaval venous obstruction. Clinical manifestations of MTS are heterogeneous and may range from asymptomatic compression to chronic venous insufficiency, acute iliofemoral DVT, or pulmonary embolism [1-3]. In some patients, particularly those presenting with pulmonary embolism, respiratory symptoms may dominate the clinical picture and divert attention from the underlying venous pathology. This case serves as a reminder that the absence of classic lower-extremity findings should not reduce suspicion for proximal venous thrombosis when other clinical features suggest VTE [2,3].

The diagnostic evaluation of suspected MTS requires an appreciation of the limitations of conventional imaging modalities. Duplex ultrasonography remains the first-line investigation for lower-extremity DVT but has reduced sensitivity for assessing the iliac veins and pelvic venous circulation because of their deep anatomical location and interference from overlying structures [2,3,7]. As a result, iliac vein compression may be overlooked if evaluation is limited to standard lower-limb ultrasonography. Cross-sectional imaging with CT venography or MR venography is often required to visualize the iliocaval segment, characterize the degree of venous compression, and assess thrombus burden [2,3,7]. When intervention is being considered, catheter venography and intravascular ultrasound (IVUS) provide detailed anatomical assessment. IVUS, in particular, has an important role in defining luminal compromise, identifying intraluminal abnormalities, and guiding endovascular therapy [2,3,7]. In our patient, cross-sectional imaging demonstrated the characteristic anatomical relationship underlying MTS, while planned MR venography represented an appropriate strategy for further anatomical characterization and treatment planning [3,7].

Given the patient's young age and extensive iliocaval thrombotic burden, evaluation for underlying thrombophilia was clinically indicated. Antiphospholipid syndrome (APS) was considered as part of the differential diagnosis for unprovoked or extensive proximal thrombosis in young adults. However, comprehensive antibody panels were deferred to the outpatient setting, and acute testing was cancelled to avoid false-positive results during active anticoagulation. Importantly, the identification of an anatomical cause such as MTS should not preclude consideration of concurrent thrombophilic disorders, particularly in young patients presenting with unusually extensive or recurrent VTE [5].

Management of thrombotic MTS requires treatment of both the acute thromboembolic event and the underlying mechanical obstruction. Therapeutic anticoagulation remains the cornerstone of initial management for acute DVT and pulmonary embolism [6,8]. However, anticoagulation alone does not address the anatomical lesion responsible for venous stasis and may therefore leave patients at risk of

recurrent thrombosis and post-thrombotic syndrome [6,8]. In selected patients with extensive iliofemoral thrombosis, severe symptoms, threatened venous outflow, or a low bleeding risk profile, catheter-directed thrombolysis, pharmacomechanical thrombectomy, or mechanical thrombectomy may be considered to reduce thrombus burden and preserve venous function [2,6,8]. Endovascular stenting of the compressed iliac vein has become an established definitive treatment for persistent hemodynamically significant obstruction and is associated with favorable long-term patency and symptom relief [6,8]. The choice of intervention should be individualized according to thrombus chronicity, symptom severity, anatomical findings, bleeding risk, and available expertise [2,6,8].

Definitive management of thrombotic MTS typically combines acute thrombus removal with catheter-directed therapy and endovascular stenting of the compressed iliac vein to restore long-term patency. In our patient, surgical thrombectomy abroad was recommended but remained financially unattainable, a common challenge in resource-limited healthcare environments. Consequently, management relied on oral anticoagulation alone. Stenting was deferred in the acute setting due to the high risk of stent thrombosis while an unlysed clot occupied the lumen. Furthermore, because follow-up thrombophilia panels were not completed post-discharge and repeat venous imaging was not performed, the persistence of the thrombus and the patient's long-term thrombotic risk remain unquantified, representing clear study limitations. Persistent iliac vein obstruction may increase the risk of recurrent VTE, chronic venous hypertension, and post-thrombotic syndrome [6,8]. Accordingly, continued clinical follow-up, reassessment of the venous anatomy when feasible, and individualized decisions regarding the duration of anticoagulation and potential endovascular intervention remain essential [6,8].

Finally, this case raises the possibility that the patient's earlier episode of chest and back pain, previously attributed to pneumonia, may have represented an antecedent thromboembolic event. Although the available records do not definitively establish that prior symptoms were caused by pulmonary embolism, the subsequent discovery of extensive VTE and underlying MTS makes this possibility plausible. Rather than constituting a confirmed missed diagnosis, this observation highlights the diagnostic challenges associated with MTS and reinforces the importance of considering occult venous anatomical abnormalities in young patients with recurrent, unexplained, or disproportionately severe thromboembolic presentations [1-3].

Conclusions

In conclusion, MTS should be considered in young patients presenting with disproportionately severe or secondary-triggered left-sided iliofemoral venous thrombosis. While CT findings demonstrated left common iliac vein compression consistent with May-Thurner anatomy, the absence of quantitative stenosis measurements, invasive venography, IVUS, and completed outpatient thrombophilia testing limits definitive causal attribution. In settings where immediate endovascular intervention is constrained by financial or logistical barriers, long-term anticoagulation and structured clinical follow-up are critical to reduce the risks of recurrent VTE and post-thrombotic syndrome.

Additional Information

Author Contributions

All authors have reviewed the final version to be published and agreed to be accountable for all aspects of the work.

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Supervision: Sayed K. Ali, Consolata Gitonga, Hanika Patel

Disclosures

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submitted work.

References

1. Brazeau NF, Harvey HB, Pinto EG, Deipolyi A, Hesketh RL, Oklu R: May-Thurner syndrome: diagnosis and management. Vasa. 2013, 42:96-105. 10.1024/0301-1526/a000252
2. Knuttinen MG, Naidu S, Oklu R, Kriegshauser S, Eversman W, Rotellini L, Thorpe PE: May-Thurner: diagnosis and endovascular management. Cardiovasc Diagn Ther. 2017, 7:S159-64. 10.21037/cdt.2017.10.14
3. Dwivedi A, Singh SN, Sharma A, Sharma R, Mishra T: A systematic review of radiological diagnosis and management of May-Thurner syndrome. J Pharm Bioallied Sci. 2024, 16:S1012-6. 10.4103/jpbs.jpbs_1135_23
4. Shi B, Huang L, Wei S, Zhu J, Li Z, Ren J, Chen D: Hemodynamic characteristics and clinical treatment of patients with iliac vein compression syndrome. Front Surg. 2025, 12:1542894. 10.3389/fsurg.2025.1542894
5. Cuntz F, Gebauer B, Greiner A, et al.: Current diagnostic and therapeutic approaches in May-Thurner syndrome in children, adolescents, and young adults: a survey among thrombosis experts of the German Society of Thrombosis and Haemostasis. Hamostaseologie. 2026, 46:127-35. 10.1055/a-2282-4565
6. Fereydooni A, Stern JR: Contemporary treatment of May-Thurner syndrome. J Cardiovasc Surg (Torino). 2021, 62:447-55. 10.23736/S0021-9509.21.11889-0
7. Arnoldussen CW: Imaging of deep venous pathology. Cardiovasc Intervent Radiol. 2024, 47:1580-94. 10.1007/s00270-024-03785-y
8. Jayaraj A, Rossi FH, Lurie F, Muck P: Diagnosis of chronic iliac venous obstruction . J Vasc Surg Venous Lymphat Disord. 2024, 12:101744. 10.1016/j.jvsv.2023.101744