

RIGHT-SIDED MAY–TURNER SYNDROME CAUSED BY AN ABERRANT RIGHT COMMON ILIAC ARTERY, MANAGED BY VENOPLASTY AND OVERLAPPING STENT PLACEMENT: A CASE REPORT

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ABSTRACT

Background: May–Turner syndrome (MTS) classically denotes compression of the left common iliac vein (LCIV) between the overlying right common iliac artery (RCIA) and the fifth lumbar vertebral body, producing left-sided venous outflow obstruction, deep vein thrombosis (DVT) and chronic venous insufficiency [1,2]. Compression of the right common iliac vein (RCIV) is uncommon, and the published experience is confined to isolated reports and small series [3,4].

Case presentation: A 37-year-old man presented in July 2025 with mild swelling and pain of the right lower limb and dilated abdominal wall veins, two years after an ipsilateral iliofemoral DVT that had been treated with retrievable inferior vena cava (IVC) filter placement and mechanical thromboaspiration, followed by six months of anticoagulation. Thrombophilia screening had been negative. He was CEAP clinical class C3 at presentation. Duplex ultrasound demonstrated a severely stenotic right external iliac vein, echogenic webs and strands within a partially recanalised superficial femoral vein, and extensive abdominal wall collaterals. Computed tomography venography (CTV) showed the right common iliac vein flattened to an anteroposterior diameter of 2 mm against a 12 mm reference segment, with a preserved transverse diameter of 13 mm — an estimated 82% cross-sectional area reduction — where an aberrantly coursing RCIA crossed anterior to it and compressed it against the vertebral body. The IVC was normally positioned. Catheter venography via right popliteal access confirmed an occluded right iliac venous segment with cross-pelvic collateral drainage. The occlusion was crossed with a coaxial hydrophilic 0.035-inch guidewire and 4 Fr angled catheter, dilated with 6 × 150 mm and 10 × 100 mm balloons, and treated with overlapping 14 × 100 mm and 14 × 80 mm self-expanding nitinol stents. Completion venography showed brisk in-line flow into the IVC. The procedure was uncomplicated.

Outcome: Duplex ultrasound on the first postoperative day and at eight weeks confirmed stent patency with continuous low-velocity flow, and the presenting symptoms resolved on clinical assessment.

Conclusions: Right-sided May–Turner syndrome may arise from an aberrant right common iliac artery in the presence of an otherwise normal infrarenal venous anatomy, without a left-sided IVC. Iliac vein compression should be considered in unexplained or recurrent right iliofemoral venous obstruction, and endovascular recanalisation with stenting is an effective treatment. This report is presented in accordance with the CARE guidelines [5].

KEYWORDS: May–Turner syndrome; iliac vein compression syndrome; right common iliac vein; venous stenting; deep vein thrombosis; non-thrombotic iliac vein lesion; interventional radiology

INTRODUCTION

In 1957, May and Turner examined 430 cadavers and described a characteristic intraluminal “spur” at the point where the right common iliac artery crosses the left common iliac vein, present in approximately 22% of specimens [1]. They attributed the lesion to chronic pulsatile arterial trauma producing intimal proliferation, and proposed it as the explanation for the observed left-sided predominance of iliofemoral thrombosis. Cockett and Thomas subsequently described the corresponding clinical entity, the iliac compression syndrome [2].

Iliac vein compression is common as an anatomical finding and uncommon as a disease. In an asymptomatic population undergoing abdominal and pelvic computed tomography for unrelated indications, Kibbe et al. found greater than 25% compression of the LCIV in 66% of subjects and greater than 50% compression in 24% [6]. Radiographic compression therefore cannot be equated with symptomatic obstruction, and the true population prevalence of clinically significant MTS remains undefined [7]. Consistent with this, a case–control study of patients who had undergone pelvic imaging before developing DVT found no significant association between increasing degrees of iliac vein compression and left-sided thrombosis [8]. Among patients presenting with iliofemoral DVT at a large referral centre, MTS was confirmed in 31% of those with left-sided or bilateral thrombosis [9].

The corresponding right-sided lesion is decidedly rarer. In a series of 173 patients treated for symptomatic MTS,

Park et al. identified atypical compression in 10 patients (5.8%), of whom only 5 (2.9% of the whole cohort) had compression of a vein other than the LCIV — four by the RCIA and one by an L5 osteophyte [3]. Chen et al. subsequently proposed a working classification of non-thrombotic right iliac vein compression into three patterns: compression of the RCIV between the right common iliac artery and the lumbar spine (type I); compression of the right external iliac vein within the angle formed by the bifurcating right external and internal iliac arteries (type II); and compression by the right external iliac artery alone (type III) [4].

Right-sided compression has been reported in association with a left-sided IVC, in which the mirror-image relationship places the left common iliac artery across the right common iliac vein [10,11]; with a high aortic bifurcation, in which the right common iliac artery compresses the IVC itself [12]; and with an overlying right internal iliac artery, either alone [13,14] or as an arterial “sandwich” between the right internal and external iliac arteries [15]. A left-sided IVC, arising from persistence of the left supracardinal vein, is one of a spectrum of infrarenal caval variants that are well characterised on cross-sectional imaging [16]. It is frequently stated that right-sided MTS requires such an anomaly; the case described here demonstrates that it does not.

We report a case of right-sided May–Thurner syndrome in a young man with a normally positioned IVC, in whom an aberrantly coursing right common iliac artery compressed the right common iliac vein against the vertebral body, and describe its successful endovascular management.

CASE PRESENTATION

Patient information

A 37-year-old man presented to the interventional radiology outpatient clinic in July 2025 with a several-month history of mild swelling and aching pain of the right lower limb, together with prominent dilated veins over the lower abdominal wall. Two years earlier, in 2023, he had sustained a right lower limb DVT that was managed elsewhere with placement of a retrievable IVC filter and mechanical thromboaspiration; the filter was subsequently retrieved. He completed six months of anticoagulation after that episode and had been off anticoagulant therapy since. Thrombophilia screening performed at that time was negative. There was no history of trauma, pelvic surgery, malignancy, prolonged immobilisation or family history of venous thromboembolism.

Clinical findings

Examination showed mild oedema of the right lower limb and multiple dilated, non-tender collateral veins over the right lower abdominal wall. There was no ulceration, induration or skin pigmentation, and the patient was classified as CEAP clinical class C3. Peripheral arterial pulses were normal and the limb was neurovascularly intact.

Diagnostic assessment

Screening duplex ultrasonography of the right lower limb demonstrated a severely stenotic right external iliac vein, a patent right common femoral vein, and echogenic strands and webs within the superficial femoral vein consistent with partial recanalisation after previous thrombosis. Multiple venous collaterals were identified in the region of the superficial femoral vein, and the popliteal vein was patent. Numerous dilated collateral channels were seen over the right side of the lower abdomen communicating with the right common femoral vein (Figure 1).

Contrast-enhanced CT venography confirmed severe stenosis of the right common and external iliac veins. At the point of compression the right common iliac vein was flattened to an anteroposterior diameter of 2 mm, with a preserved transverse diameter of 13 mm, against a reference vein diameter of 12 mm in the adjacent normal segment (Figure 2). Assuming an elliptical cross-section at the compression site and a circular reference vein, this corresponds to a cross-sectional area of approximately 20 mm² against a reference of approximately 113 mm² — an estimated 82% area reduction, with an 83% reduction in anteroposterior diameter. The preservation of the transverse dimension in the face of near-complete anteroposterior collapse is the morphological signature of extrinsic arterial compression rather than intrinsic stenosis. Both values exceed the thresholds of greater than 50% area reduction and greater than 61% diameter reduction above which intervention is considered appropriate [17]. The right common iliac vein was compressed between the right common iliac artery and the adjacent vertebral body, with the artery following an aberrant course crossing anterior to the vein. The IVC was normally positioned on the right, and no left-sided or duplicated IVC, retroaortic renal vein or other caval variant was present. The appearances were those of right-sided May–Thurner syndrome, corresponding to a type I pattern of right iliac vein compression [4].

Diagnostic catheter venography was performed through right popliteal venous access. This demonstrated a partially recanalised superficial femoral vein with multiple outflow channels, an occluded right iliac venous segment through which only a narrow stream of contrast passed, and prominent cross-pelvic collateral channels draining the right common femoral vein into the left common femoral vein (Figure 3).

Therapeutic intervention

The occluded iliac venous segment was crossed using a coaxial combination of a hydrophilic 0.035-inch guidewire and a 4 Fr angled (Vert) catheter (Figure 4A, 4B). Serial venoplasty was performed with 6 × 150 mm and 10 × 100 mm balloons (Figure 4C). Because recanalisation of the diseased segment remained inadequate after angioplasty alone, the right common and external iliac veins were stented with two overlapping self-expanding nitinol stents measuring 14 × 100 mm and 14 × 80 mm (E-Luminexx; BD/Bard Peripheral Vascular, Tempe, AZ, USA) (Figure 5A). Completion venography demonstrated good recanalisation of the right common and external

iliac veins with free antegrade flow of contrast into the IVC and disappearance of collateral filling (Figure 5B). The procedure was uncomplicated, with no access-site or periprocedural adverse events.

Follow-up and outcomes

Duplex ultrasonography performed on the first postoperative day confirmed a patent iliac stent. The patient was discharged on apixaban 5 mg twice daily together with aspirin 75 mg once daily, a combination of therapeutic anticoagulation with a single antiplatelet agent consistent with reported practice after stenting of post-thrombotic iliofemoral obstruction [18]. At eight weeks, the stents remained patent on duplex ultrasound, with continuous low-velocity flow demonstrated within the stented segment on spectral Doppler (Figure 6), and the presenting limb swelling, pain and abdominal wall collateral prominence had resolved on clinical assessment.

DISCUSSION

Anatomical basis of right-sided compression

The classic May–Thurner lesion depends on a fixed anatomical relationship: the right common iliac artery crosses the left common iliac vein and pins it against the lumbosacral spine, so that chronic pulsatile arterial trauma drives intimal hyperplasia and spur formation [1,2]. Venous stasis and turbulent flow follow, completing Virchow’s triad and predisposing to thrombosis.

For the right common iliac vein to be compressed, that relationship must be altered. The commonly cited mechanism is a left-sided IVC, which produces a mirror-image configuration in which the left common iliac artery crosses the right common iliac vein [10,11]. Other described mechanisms include a high aortic bifurcation with caval compression by the right common iliac artery [12], compression by an overlying right internal iliac artery [13,14], entrapment of the right external iliac vein between the right internal and external iliac arteries [15], and compression by a lumbosacral osteophyte [3].

The present case does not fit the assumption that anomalous venous anatomy is a prerequisite. The IVC was normally positioned, and compression arose instead from an aberrant course of the right common iliac artery anterior to the right common iliac vein, corresponding to the type I pattern in the classification proposed by Chen et al. [4]. Statements in the literature that right-sided MTS “always” requires a left-sided IVC should therefore be read as a description of the commonest reported association rather than as a necessary condition.

The morphology observed on CT venography supports an extrinsic mechanism. The vein was not concentrically narrowed but flattened, its anteroposterior diameter reduced to 2 mm while the transverse diameter was maintained at 13 mm — slightly wider than the 12 mm reference segment. This pattern of anteroposterior collapse with preserved or increased transverse dimension is characteristic of arterial compression and distinguishes it from intrinsic post-thrombotic stenosis, in which luminal loss is more uniform.

Epidemiology

Robust epidemiological data for right-sided MTS do not exist, and the figures quoted for MTS in general should be treated with caution [7]. The best available estimate of the relative frequency of non-left-sided compression comes from Park et al., in whose single-centre series of 173 symptomatic patients only 5 (2.9%) had compression of a vein other than the LCIV [3]. Reported sex distributions relate almost entirely to left-sided disease: a systematic review of 1,569 patients with MTS found a female-to-male ratio of approximately 2:1 with younger age at presentation in women [19]. No comparable data exist for right-sided disease, and the published right-sided cases include both men and women; assertions of a marked female predominance in right-sided MTS are not supported by the current literature. The present patient was a young man, consistent with several other reported right-sided cases [13,15].

The traditional teaching of a marked left-sided predominance of iliofemoral DVT also warrants qualification. In a consecutive series of 496 patients with iliofemoral or thigh DVT, thrombosis was left-sided in 53%, right-sided in 42% and bilateral in 5% [9] — a ratio far closer to unity than the figures often quoted, and a reminder that right iliac venous obstruction is not so rare a diagnosis that it can be excluded on laterality alone.

Clinical presentation and differential diagnosis

Presentations range from asymptomatic radiographic compression through chronic limb heaviness, aching and swelling exacerbated by dependency, to acute iliofemoral DVT [20]. Chronic obstruction may be accompanied by venous hypertension with telangiectasia, hyperpigmentation, induration, varicosities and, in advanced disease, ulceration. Extensive collateral formation — as in the present case, where suprapubic and abdominal wall collaterals were prominent — may partially compensate for the obstruction and mask symptoms until a further prothrombotic insult supervenes.

Right-sided disease is particularly liable to delayed diagnosis, because unilateral right limb swelling is readily attributed to cellulitis, lymphoedema or post-traumatic swelling, and because the clinician’s index of suspicion for iliac compression is anchored to the left side [13]. The differential diagnosis of unilateral limb swelling also includes lymphatic obstruction, extrinsic pelvic mass and, in the post-thrombotic patient, recurrent thrombosis. Persistent or progressive swelling after an adequately treated DVT, as here, should prompt dedicated imaging of the ilio caval segment rather than repeated assessment of the infrainguinal veins alone.

Diagnostic evaluation

Duplex ultrasonography is the appropriate first-line investigation and is sensitive for femoropopliteal thrombosis, but is limited in the iliac segment by depth, bowel gas and body habitus; indirect signs such as loss of respiratory phasicity, absent flow augmentation and the presence of abdominal wall collaterals are often the only clues. In

the present case duplex correctly raised the suspicion of iliac obstruction (Figure 1).

CT venography and MR venography define the level and mechanism of compression, delineate collateral pathways and, critically, identify the anatomical variants that underlie right-sided disease — caval variants, a high aortic bifurcation, or an aberrant arterial course as here [16]. MR venography is preferred in pregnancy and in renal impairment. Cross-sectional imaging also permits the quantification that a purely descriptive report cannot supply, and both anteroposterior and transverse dimensions should be recorded, since a flattened vein reported by a single diameter will be misrepresented.

Catheter venography permits dynamic assessment and simultaneous intervention but systematically underestimates the severity of obstruction. In the VIDIO trial, three-view venography detected stenotic lesions in 51 of 100 patients compared with 81 by intravascular ultrasound (IVUS), underestimated diameter reduction by a mean of 11% ($P < 0.001$), and IVUS altered the treatment plan in 57 of 100 patients [21]. Current consensus recommends IVUS, including dynamic assessment, for evaluating non-thrombotic iliac vein lesions (NIVL), and identifies thresholds of greater than 50% area reduction or greater than 61% diameter stenosis as those correlating with symptomatic benefit after stenting [17].

Management

Asymptomatic or minimally symptomatic compression does not require intervention. Conservative management comprises graduated compression hosiery, limb elevation, exercise and clinical surveillance, with anticoagulation determined by the patient's thrombotic risk rather than by the presence of compression per se. In treated patients with a non-thrombotic iliac vein lesion and no history or imaging evidence of prior venous thromboembolism, the 2024 VIVA Foundation / American Venous Forum / American Vein and Lymphatic Society consensus statement reached no agreement that anticoagulant or antiplatelet therapy is necessary, and recommends instead an individualised assessment of thrombotic risk [17].

Endovascular therapy is the treatment of choice for symptomatic obstruction. In acute iliofemoral DVT, anticoagulation is the foundation of treatment; the role of adjunctive catheter-directed or pharmacomechanical thrombolysis is more limited than was once assumed, and is addressed below. In chronic non-thrombotic stenosis and in post-thrombotic obstruction such as the present case, treatment consists of crossing the lesion, serial balloon dilatation and placement of self-expanding stents sized to the reference vein, with stent landing zones ideally selected on IVUS. Long-segment or multifocal disease may require overlapping stents, as here.

Open surgery is now rarely required and is reserved for failed endovascular treatment, refractory in-stent restenosis, or unusual anatomical circumstances such as extrinsic compression by a pelvic mass. The Palma crossover femoro-femoral venous bypass remains a salvage option but has inferior durability compared with endovascular reconstruction.

Antithrombotic management after venous stenting is not standardised. The European Society for Vascular Surgery advises that anticoagulation after early thrombus removal, with or without stenting, should be at least as long as anticoagulation alone would have been, with the final duration at the discretion of the treating clinician [22]. The addition of a single antiplatelet agent to anticoagulation is common practice after recanalisation of post-thrombotic obstruction, and was the approach taken here, although the supporting evidence is retrospective [18]. A systematic review of 56 studies and 5,153 patients concluded that the impact of post-interventional antithrombotic therapy on stent patency remains unknown because the available data are insufficient [23].

Outcomes and surveillance

Reported outcomes after iliac venous stenting are good but should be quoted accurately. In the largest single-centre series, comprising 982 stented limbs, primary patency at 72 months was 79% for non-thrombotic disease and 57% for post-thrombotic disease, with secondary patency of 100% and 86% respectively; severe in-stent restenosis exceeding 50% occurred in 1% of non-thrombotic and 10% of post-thrombotic limbs [24]. A systematic review reported mean primary patency of 82.3% at one year and 73.3% at two years [23]. Clinical benefit is substantial but incomplete: in the same large series, complete relief of pain was achieved in 62% and complete relief of swelling in 32% at five years, with ulcer healing in 58% [24].

An important correction is warranted to the widely repeated claim that early intervention substantially reduces post-thrombotic syndrome. In the randomised ATTRACT trial, pharmacomechanical catheter-directed thrombolysis did not reduce the incidence of post-thrombotic syndrome compared with anticoagulation alone (47% versus 48% between 6 and 24 months; $P = 0.56$), and was associated with more major bleeding within 10 days (1.7% versus 0.3%; $P = 0.049$). Moderate-to-severe post-thrombotic syndrome was less frequent in the intervention arm (18% versus 24%; $P = 0.04$) [25]. Claims of a large relative risk reduction in post-thrombotic syndrome from timely intervention are not supported by randomised evidence. Complex clinical cases may benefit from interdisciplinary collaboration to support comprehensive assessment and coordinated treatment planning [26].

Surveillance duplex ultrasonography at 1, 3, 6, 12 and 24 months and annually thereafter is a reasonable schedule, allowing early detection and re-intervention for in-stent restenosis. Adherence to prescribed anticoagulation and to follow-up remains the principal modifiable determinant of stent thrombosis.

Future research may investigate whether validated artificial intelligence approaches can assist clinicians in interpreting vascular imaging and identifying uncommon anatomical patterns. Any such application would require dedicated clinical validation before it could be used to guide diagnosis or treatment. Broader applications of artificial intelligence have been discussed in other applied research fields [27].

LIMITATIONS

This report describes a single patient followed for eight weeks, which is insufficient to draw conclusions about durability; longer-term patency and symptom data are required and will be reported. Intravascular ultrasound was not performed, so the degree of stenosis was quantified on CT venography rather than to the intravascular standard on which current consensus thresholds are based [17], and the estimate of area reduction assumes an elliptical lumen at the compression site and a circular reference vein. Clinical outcome was assessed by symptom resolution rather than by a validated venous severity score. The aberrant arterial course was established on cross-sectional and catheter venography rather than by operative confirmation.

CONCLUSION

Right-sided May–Thurner syndrome is uncommon but should not be regarded as requiring anomalous venous anatomy. In this patient an aberrantly coursing right common iliac artery compressed the right common iliac vein against the vertebral body in the presence of a normally positioned inferior vena cava, producing chronic right iliofemoral venous obstruction that progressed after a completed course of anticoagulation for an earlier deep vein thrombosis. Cross-sectional venography identified the mechanism and quantified the lesion, and endovascular recanalisation with serial venoplasty and overlapping self-expanding stents restored in-line flow with resolution of symptoms at eight weeks. Iliac vein compression merits consideration in any patient with unexplained, recurrent or progressive unilateral limb swelling, irrespective of side, particularly when conventional thrombotic risk factors are absent.

ABBREVIATIONS

CEAP, Clinical–Etiological–Anatomical–Pathophysiological classification; CTV, Computed tomography venography; DVT, Deep vein thrombosis; IVC, Inferior vena cava; IVUS, Intravascular ultrasound; LCIV, Left common iliac vein; MRV, Magnetic resonance venography; MTS, May–Thurner syndrome; NIVL, Non-thrombotic iliac vein lesion; PTS, Post-thrombotic syndrome; RCIA, Right common iliac artery; RCIV, Right common iliac vein; VTE, Venous thromboembolism.

DECLARATIONS

Patient consent. Written informed consent was obtained from the patient for publication of this case report and the accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

Ethics approval. Institutional ethics committee approval is not required for single-patient case reports at Jawaharlal Nehru Medical College, Belagavi. The report was prepared in accordance with the Declaration of Helsinki.

Funding. This research received no specific grant from any funding agency in the public, commercial or not-for-profit sectors.

Conflicts of interest. The authors declare that they have no competing interests.

Author contributions. IMH and NMM performed the procedure; PSV, SB and AR acquired and interpreted the imaging; PSV drafted the manuscript; BB, AR and IMH revised it critically for important intellectual content. All authors read and approved the final manuscript and agree to be accountable for all aspects of the work.

Data availability. All data generated or analysed during this study are included in this published article.

Reporting guideline. This case report was prepared in accordance with the CARE (CAse REport) guidelines [5].

REFERENCES

1. May R, Thurner J. The cause of the predominantly sinistral occurrence of thrombosis of the pelvic veins. *Angiology*. 1957;8(5):419–427. doi:10.1177/000331975700800505
2. Cockett FB, Thomas ML. The iliac compression syndrome. *Br J Surg*. 1965;52(10):816–821. doi:10.1002/bjs.1800521028. PMID: 5828716
3. Park JY, Park KM, Cho SG, Hong KC, Jeon YS. Atypical iliac vein compression in patients with symptomatic May–Thurner syndrome. *Diagn Interv Radiol*. 2021;27(3):372–377. doi:10.5152/dir.2021.20183. PMID: 34003124
4. Chen ZL, Zhang XC, Sun Y, Xu M. Diagnosis and treatment of nonthrombotic right iliac vein compression syndrome. *Ann Vasc Surg*. 2019;61:363–370. doi:10.1016/j.avsg.2019.05.033
5. Gagnier JJ, Kienle G, Altman DG, Moher D, Sox H, Riley D; CARE Group. The CARE guidelines: consensus-based clinical case reporting guideline development. *BMJ Case Rep*. 2013;2013:bcr2013201554. doi:10.1136/bcr-2013-201554. PMID: 24155002
6. 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(5):937–943. doi:10.1016/j.jvs.2003.12.032. PMID: 15111841
7. Harbin MM, Lutsey PL. May–Thurner syndrome: history of understanding and need for defining population prevalence. *J Thromb Haemost*. 2020;18(3):534–542. doi:10.1111/jth.14707. PMID: 31821707
8. Narayan A, Eng J, Carmi L, McGrane S, Ahmed M, Sharrett AR, et al. Iliac vein compression as risk factor

- for left- versus right-sided deep venous thrombosis: case-control study. *Radiology*. 2012;265(3):949–957. doi:10.1148/radiol.12111580. PMID: 23175547
9. Heller T, Teichert C, Hafer J, Weber MA, Kröger JC, Meinel FG. Prevalence of May–Thurner syndrome in patients with deep vein thrombosis at a large medical referral center. *Rofo*. 2019;191(12):1107–1117. doi:10.1055/a-0959-6230. PMID: 31272108
 10. Burke RM, Rayan SS, Kasirajan K, Chaikof EL, Milner R. Unusual case of right-sided May–Thurner syndrome and review of its management. *Vascular*. 2006;14(1):47–50. doi:10.2310/6670.2006.00012
 11. Abboud G, Midulla M, Lions C, El Ngheoui Z, Gengler L, Martinelli T, et al. “Right-sided” May–Thurner syndrome. *Cardiovasc Intervent Radiol*. 2010;33(5):1056–1059. doi:10.1007/s00270-009-9654-z. PMID: 19629587
 12. Fretz V, Binkert CA. Compression of the inferior vena cava by the right iliac artery: a rare variant of May–Thurner syndrome. *Cardiovasc Intervent Radiol*. 2010;33(5):1060–1063. doi:10.1007/s00270-009-9671-y. PMID: 19680718
 13. Nasif A, Ahmed AM, Al-Embideen S, Nazzal M, Osman M, Ahmed A. Unusual presentation of right-sided May–Thurner syndrome. *J Vasc Surg Cases Innov Tech*. 2021;7(4):768–771. doi:10.1016/j.jvscit.2021.10.005. PMID: 34816070
 14. 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(3):291–292. doi:10.1177/096721090201000319. PMID: 12044441
 15. Yamanaka T, Ishihara T, Hara T, Ichinohe Y, Fukatsu T. Right-sided iliac vein compression syndrome: when the vein is compressed between the internal and external iliac arteries — a case report. *Eur Heart J Case Rep*. 2024;8(1):ytae011. doi:10.1093/ehjcr/ytae011. PMID: 38239308
 16. Bass JE, Redwine MD, Kramer LA, Huynh PT, Harris JH Jr. Spectrum of congenital anomalies of the inferior vena cava: cross-sectional imaging findings. *RadioGraphics*. 2000;20(3):639–652. doi:10.1148/radiographics.20.3.g00ma09639. PMID: 10835118
 17. Desai KR, Sabri SS, Elias S, Gagne PJ, Garcia MJ, Gibson K, et al. Consensus statement on the management of nonthrombotic iliac vein lesions from the VIVA Foundation, the American Venous Forum, and the American Vein and Lymphatic Society. *Circ Cardiovasc Interv*. 2024;17(8):e014160. doi:10.1161/CIRCINTERVENTIONS.124.014160. PMID: 39034930
 18. Xiao N, Genet M, Khaja MS, Desai KR. Antithrombotic therapy after deep venous intervention. *Semin Intervent Radiol*. 2022;39(4):357–363. doi:10.1055/s-0042-1757320
 19. Kaltenmeier CT, Erben Y, Indes J, Lee A, Dardik A, Sarac T, et al. Systematic review of May–Thurner syndrome with emphasis on gender differences. *J Vasc Surg Venous Lymphat Disord*. 2018;6(3):399–407.e4. doi:10.1016/j.jvsv.2017.11.006. PMID: 29290600
 20. Birn J, Vedantham S. May–Thurner syndrome and other obstructive iliac vein lesions: meaning, myth, and mystery. *Vasc Med*. 2015;20(1):74–83. doi:10.1177/1358863X14560429. PMID: 25502563
 21. Gagne PJ, Tahara RW, Fastabend CP, Dzieciuchowicz L, Marston W, Vedantham S, et al. Venography versus intravascular ultrasound for diagnosing and treating iliofemoral vein obstruction. *J Vasc Surg Venous Lymphat Disord*. 2017;5(5):678–687. doi:10.1016/j.jvsv.2017.04.007. PMID: 28818221
 22. Kakkos SK, Gohel M, Baekgaard N, Bauersachs R, Bellmunt-Montoya S, Black SA, et al. Editor’s Choice — European Society for Vascular Surgery (ESVS) 2021 clinical practice guidelines on the management of venous thrombosis. *Eur J Vasc Endovasc Surg*. 2021;61(1):9–82. doi:10.1016/j.ejvs.2020.09.023. PMID: 33334670
 23. Notten P, ten Cate H, ten Cate-Hoek AJ. Postinterventional antithrombotic management after venous stenting of the iliofemoral tract in acute and chronic thrombosis: a systematic review. *J Thromb Haemost*. 2021;19(3):753–796. doi:10.1111/jth.15197. PMID: 33249698
 24. Neglén P, Hollis KC, Olivier J, Raju S. Stenting of the venous outflow in chronic venous disease: long-term stent-related outcome, clinical, and hemodynamic result. *J Vasc Surg*. 2007;46(5):979–990. doi:10.1016/j.jvs.2007.06.046. PMID: 17980284
 25. Vedantham S, Goldhaber SZ, Julian JA, Kahn SR, Jaff MR, Cohen DJ, et al. Pharmacomechanical catheter-directed thrombolysis for deep-vein thrombosis. *N Engl J Med*. 2017;377(23):2240–2252. doi:10.1056/NEJMoal615066. PMID: 29211671
 26. Kabra P. Interdisciplinary dentistry as the key player of successful treatment outcomes in dentistry: a narrative review. *J Dent Clin Sci*. 2025;1(1):1–8. <https://agrpjournals.com/journals/journal/index.php/jdacs/article/view/8>
 27. The evolution of marketing strategy in the age of artificial intelligence: from mass targeting to hyper-personalization. *J Artif Intell Agric Appl Res*. 2025;1(1):20–24. <https://agrpjournals.com/journals/journal/index.php/jaiaar/article/view/5>

FIGURE LEGENDS

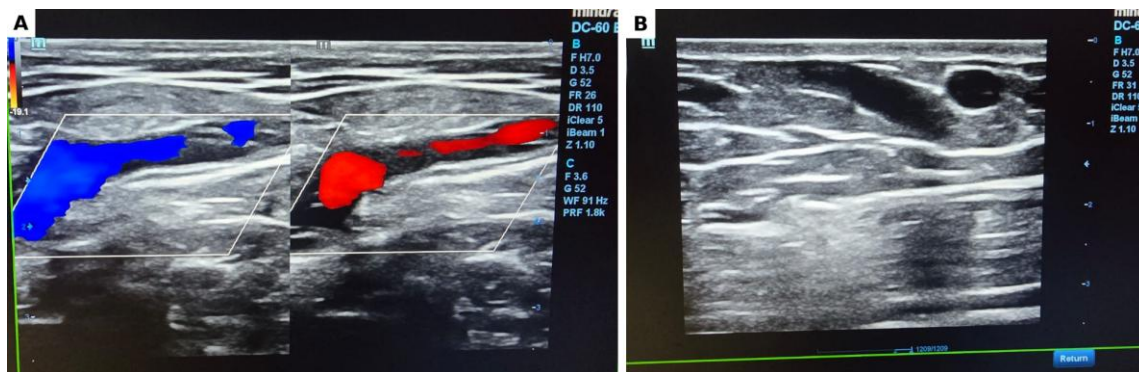


Figure 1. Baseline duplex ultrasonography of the right lower limb. (A) Dual-panel colour Doppler images of the right saphenofemoral region, showing colour reversal between the two acquisitions consistent with bidirectional flow on augmentation. (B) Greyscale image of the right iliac region of the abdominal wall, demonstrating numerous dilated collateral channels communicating with the right common femoral vein.

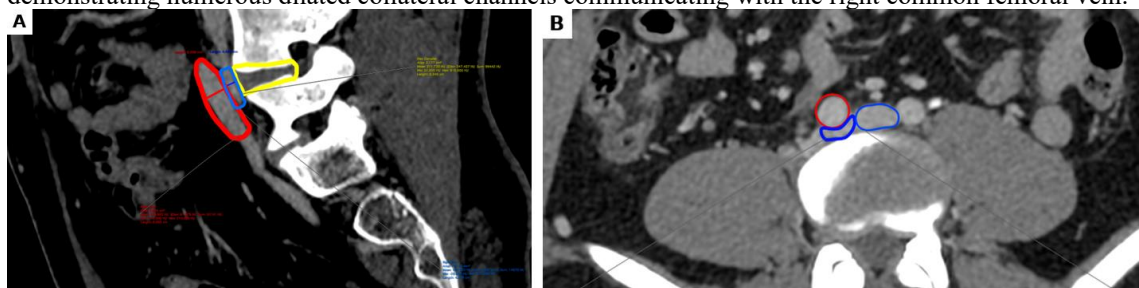


Figure 2. Computed tomography venography demonstrating right-sided iliac vein compression. (A) Oblique sagittal multiplanar reformat and (B) axial image at the level of the iliac vessels. The right common iliac vein (blue outline) is flattened to an anteroposterior diameter of 2 mm, with a preserved transverse diameter of 13 mm, where it is crossed by the aberrantly coursing right common iliac artery (red outline) and compressed against the adjacent vertebral body; the reference vein diameter in the adjacent normal segment was 12 mm. The inferior vena cava is normally positioned on the right, with no caval variant. Regions of interest and measurement annotations are those generated at the diagnostic workstation.



Figure 3. Initial diagnostic catheter venography via right popliteal access. (A) Femoral phase demonstrating a partially recanalised right superficial femoral vein with multiple parallel outflow channels. (B) and (C) Pelvic digital subtraction venograms showing absence of opacification of the right iliac venous segment, with extensive tortuous ascending pelvic and abdominal wall collaterals and cross-pelvic collateral drainage towards the left common femoral vein. Only a narrow stream of contrast traversed the occluded iliac segment.

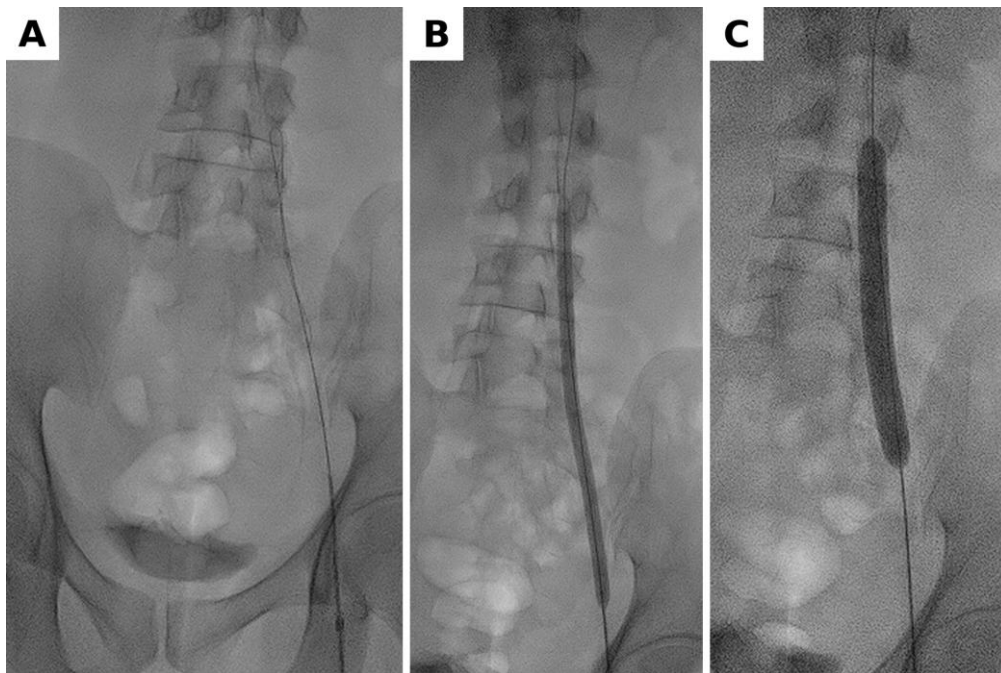


Figure 4. Recanalisation and balloon venoplasty of the occluded right iliac venous segment. (A) and (B) Unsubtracted fluoroscopic images showing the coaxial hydrophilic 0.035-inch guidewire and 4 Fr angled catheter traversing the occluded right common and external iliac veins into the inferior vena cava. (C) Serial balloon venoplasty of the diseased iliac segment.

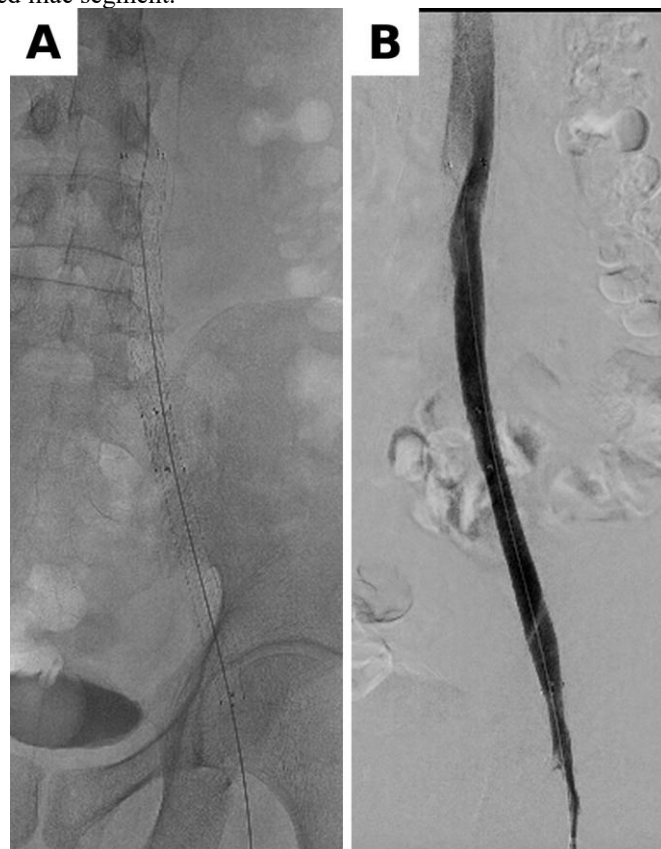


Figure 5. Stent placement and completion venography. (A) Unsubtracted fluoroscopic image showing the two overlapping self-expanding nitinol stents (14 × 100 mm and 14 × 80 mm) deployed across the right common and external iliac veins. (B) Completion digital subtraction venogram demonstrating a widely patent stented segment with brisk antegrade flow of contrast into the inferior vena cava and disappearance of collateral filling.

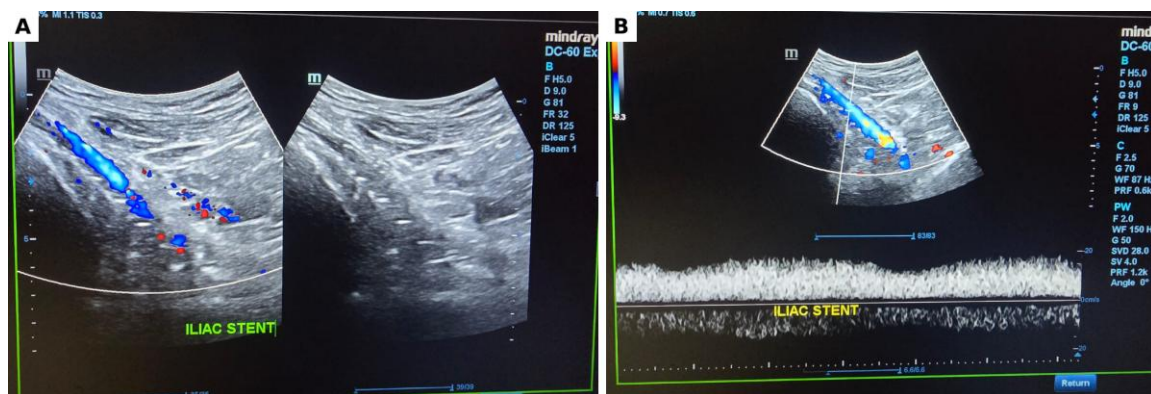


Figure 6. Duplex ultrasonography at eight-week follow-up. (A) Colour Doppler and paired greyscale images demonstrating flow within the patent right iliac stent. (B) Colour Doppler with spectral tracing from within the stented segment, showing continuous low-velocity venous flow and no evidence of in-stent stenosis or thrombosis.