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*Case report*

# Inferior Vena Cava Agenesis Presenting with Recurrent Deep Vein Thrombosis in a 54-Year-Old Woman: A Rare Case Report

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*Inferior Vena Cava Agenesis; Congenital IVC Anomaly; Deep Vein Thrombosis; Recurrent DVT; Venous Stasis; Collateral Circulation; Warfarin.*

**Abstract**

Congenital anomalies of the inferior vena cava (IVC) are uncommon vascular abnormalities that are frequently asymptomatic. When clinically significant, they may predispose to venous stasis and recurrent deep vein thrombosis (DVT). Case presentation: A 54-year-old woman with a history of recurrent right lower-limb DVT (a first episode five years earlier in the posterior tibial vein and a second episode three years earlier in the femoral vein) presented with chronic recurrent lower abdominal pain, loss of appetite, and bilateral lower-limb pain. Physical examination was unremarkable. A thrombophilia work-up, including inherited and acquired thrombophilia investigations, was reported as negative. She also had a history of three recurrent abortions. Contrast-enhanced computed tomography of the abdomen and pelvis demonstrated a major congenital venous anomaly characterised by agenesis/hypoplasia of the infrahepatic segment of the inferior vena cava with extensive, dilated collateral venous channels. The hepatic veins and left renal vein were visualised and remained intact. The patient was admitted to the hospital and initially treated with subcutaneous enoxaparin (8,000 IU twice daily) for one week and was started on oral warfarin (5 mg once daily) four days after initiation of enoxaparin, once the diagnosis was confirmed. Her INR increased from 1.2 before anticoagulation to 2.5 after one month of treatment. Complete resolution of the abdominal and bilateral lower-limb pain was reported after approximately two weeks of anticoagulant therapy. Congenital IVC agenesis should be considered in patients with recurrent or otherwise unexplained DVT, particularly when routine thrombophilia investigations are negative. Contrast-enhanced CT can demonstrate the venous anomaly and the associated collateral circulation. Recognition of this anatomical abnormality is important for long-term thromboembolic risk assessment and management.

## Introduction

Congenital anomalies of the inferior vena cava (IVC) include agenesis, hypoplasia, interruption, duplication, and other developmental variants [9,10]. Most IVC anomalies are clinically silent and may be detected incidentally on abdominal imaging. However, abnormalities that impair venous return from the lower extremities can promote venous stasis and increase the risk of deep vein thrombosis (DVT). IVC agenesis is an uncommon but clinically important cause of venous thromboembolism.

Published case reports and reviews have described recurrent or apparently unprovoked DVT in association with absent or severely hypoplastic segments of the IVC, often with extensive collateral venous pathways [1–8]. The diagnosis may be overlooked because symptoms can be nonspecific and the anomaly may not be recognised until abdominal or pelvic imaging is performed. We report a 54-year-old woman with recurrent right lower-limb DVT, recurrent lower abdominal and bilateral lower-limb pain, a history of three abortions, negative thrombophilia investigations, and contrast-enhanced CT findings consistent with congenital agenesis/hypoplasia of the infrahepatic IVC with extensive collateral venous circulation.

## Case Presentation

A 54-year-old woman presented with chronic recurrent lower abdominal pain associated with loss of appetite and bilateral lower-limb pain. She had a previous history of two episodes of deep vein thrombosis involving the right lower limb: a first episode five years earlier affecting the posterior tibial vein, and a second episode three years earlier affecting the femoral vein. No previous imaging documenting these DVT episodes was available for review. Her past obstetric history was notable for three recurrent abortions. A comprehensive thrombophilia evaluation, including protein C, protein S, antithrombin, antiphospholipid antibodies, Factor V Leiden, and prothrombin gene mutation, was reported as negative. On physical examination, the patient was clinically stable. Pulse and blood pressure were within normal limits, and examinations of the chest, cardiovascular system, abdomen, and central nervous system were unremarkable.


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### Laboratory Investigations

Laboratory evaluation showed a random blood glucose level of 89 mg/dL. Serum electrolytes were within the laboratory reference ranges: sodium 137.8 mmol/L, potassium 4.41 mmol/L, and chloride 107.3 mEq/L. Amylase was 25 U/L, and lipase was 21.4 U/L, without biochemical evidence suggestive of pancreatitis. Inflammatory markers were elevated, with a C-reactive protein (CRP) level of 47.8 mg/L and an erythrocyte sedimentation rate (ESR) of 120 mm/hour. The stool examination showed loose stool; 2–3 white blood cells/high-power field and 1–2 red blood cells/high-power field; and no trophozoites, cysts, ova, larvae, or other parasitological findings detected. The thrombophilia work-up included testing for protein C deficiency, protein S deficiency, antithrombin deficiency, antiphospholipid antibodies, Factor V Leiden mutation, and prothrombin (Factor II) gene mutation; all results were reported as negative. Specific numerical laboratory values for each test should be inserted into the final manuscript once the original reports are retrieved.

### Imaging Findings

Contrast-enhanced computed tomography (CT) of the abdomen and pelvis performed in August 2026 demonstrated a major systemic venous abnormality. The inferior vena cava was not confidently identified along its expected course, and the expected bilateral external and common iliac veins were not confidently visualised. Multiple prominent collateral venous channels were identified within the retroperitoneal and paravertebral regions. The left renal vein appeared to drain through collateral venous pathways, with an apparent abrupt termination near its expected caval confluence. The imaging report also described dilated and tortuous collateral vessels communicating with paravertebral and anterior abdominal wall venous pathways and additional pelvic collateral veins. The hepatic veins and left renal vein were visualised and described as intact. The liver, spleen, pancreas, kidneys, abdominal aorta, urinary bladder, and pelvic organs were otherwise reported as having no significant focal abnormality. The radiological impression was congenital agenesis/hypoplasia of the infrahepatic segment of the inferior vena cava with prominent collateral venous circulation. A second radiology report considered the findings highly suspicious for a major congenital systemic venous abnormality, including IVC agenesis, interruption, or severe hypoplasia. Although dedicated CT venography or MR venography was recommended, it was not performed. Therefore, the final manuscript should describe the diagnosis as congenital infrahepatic IVC agenesis based on the available contrast-enhanced CT interpretation, while acknowledging that dedicated venography was not undertaken.

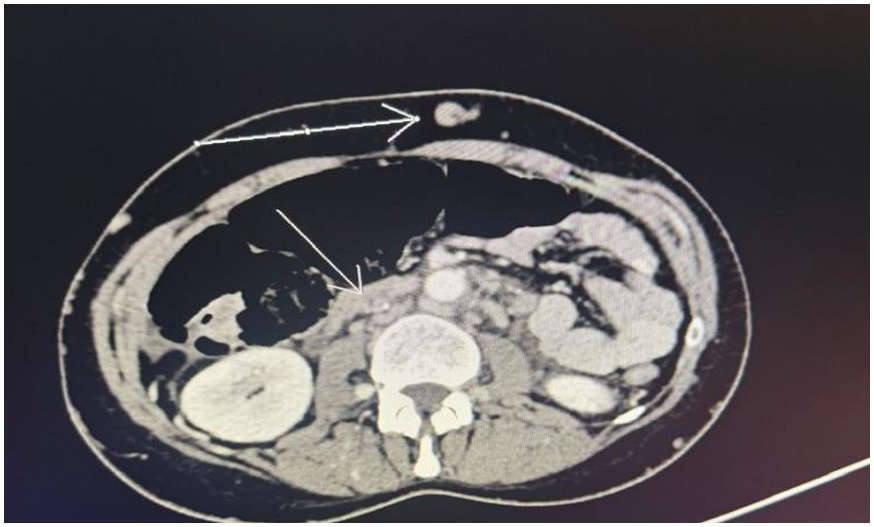

***Figure 1. Axial contrast-enhanced CT of the abdomen in the present patient, obtained at the level of the retroperitoneum, showing dilated and tortuous paravertebral and retroperitoneal collateral venous channels (arrows) in the absence of a normal-calibre infrahepatic inferior vena cava, consistent with congenital IVC agenesis/hypoplasia and collateral venous drainage.***


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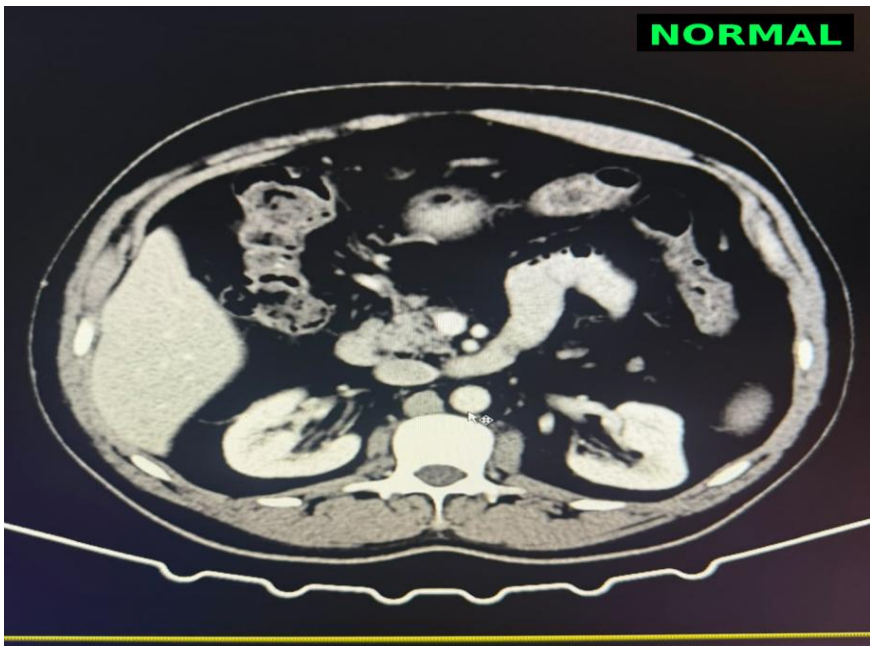


***Figure 2. Axial contrast-enhanced CT from a comparison subject with normal venous anatomy.***

## Treatment and Clinical Outcome

The patient was admitted to the hospital and started on subcutaneous enoxaparin (Clexane) at a dose of 8,000 IU twice daily for one week. Oral warfarin (5 mg once daily) was started four days after initiation of enoxaparin, once the diagnosis of congenital IVC agenesis was confirmed, with enoxaparin and warfarin overlapping until a therapeutic INR was achieved. The INR before anticoagulation was 1.2. On the most recent measurement, obtained after one month of anticoagulant treatment, the INR was 2.5. Following anticoagulant treatment, the patient reported complete resolution of the chronic lower abdominal pain and bilateral lower-limb pain within approximately two weeks.

## Discussion

The present case highlights congenital IVC agenesis as a potential anatomical risk factor for recurrent venous thromboembolism. The IVC is the principal venous conduit returning blood from the lower extremities and abdomen to the right atrium. Congenital absence or severe hypoplasia of one or more IVC segments results in diversion of venous blood through collateral pathways. Although these collateral channels may maintain venous return, the altered anatomy can produce venous hypertension, reduced flow, and venous stasis, thereby increasing susceptibility to thrombosis [9,10]. The association between IVC agenesis and DVT has been reported in multiple case reports and systematic reviews. In many reported patients, thrombophilia investigations are negative, and the DVT is described as recurrent or apparently unprovoked [1–8]. This pattern is relevant to the present patient, who had two previous right lower-limb DVT episodes (posterior tibial vein five years earlier and femoral vein three years earlier) and a negative thrombophilia work-up.

An important feature of this case is the patient's age of 54 years. IVC anomalies associated with DVT are often discussed in younger adults, and the diagnosis may therefore be overlooked in older patients. The current case emphasises that congenital IVC anomalies should remain in the differential diagnosis of recurrent or unexplained DVT irrespective of age when conventional thrombotic risk factors are not identified. The patient's history of three recurrent abortions is noteworthy. However, a causal relationship between IVC agenesis and recurrent pregnancy loss cannot be established from this single case. The negative thrombophilia and antiphospholipid investigations reduce support for a recognised acquired or inherited thrombophilic explanation, but other obstetric causes of recurrent pregnancy loss should be considered. Accordingly, the abortions should be presented as an important part of the patient's history rather than as a proven manifestation of IVC agenesis.

The diagnosis in this patient was made by contrast-enhanced CT of the abdomen and pelvis, which demonstrated the absent/hypoplastic IVC segment and extensive collateral venous pathways. Dedicated CT venography or MR venography can provide further anatomical characterisation when required, particularly when the distinction between agenesis, interruption, severe hypoplasia, or chronic occlusion is uncertain. Anticoagulation is the mainstay of treatment when IVC anomalies are associated with DVT. There is no universally established anticoagulation regimen or duration specific to IVC agenesis because of the rarity of the condition and the absence of randomised clinical trials. Recent literature has also evaluated direct oral anticoagulants in this setting [5], while traditional anticoagulation with warfarin remains an


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established treatment option. In the present case, the patient received initial enoxaparin followed by warfarin, with an INR of 2.5 after one month of treatment and complete clinical improvement over approximately two weeks.

## Conclusion

Congenital agenesis of the inferior vena cava is a rare but clinically relevant anatomical abnormality that may predispose patients to recurrent deep vein thrombosis through impaired venous drainage and collateral-dependent circulation. It should be considered in patients with recurrent or unexplained DVT, particularly when routine thrombophilia testing is negative. Contrast-enhanced CT can reveal the characteristic absence or severe hypoplasia of the IVC and associated collateral vessels. Early recognition may improve long-term thromboembolic risk assessment and guide appropriate anticoagulant management.

***Conflict of interest.*** Nil

## References

1. Prado VE, Rey-Mendoza JP, Wakefield CJ, Ba Aqeel S, Kumssa A. Infrarenal inferior vena cava agenesis and recurrent deep vein thrombosis: a case report and literature review. Oxf Med Case Rep. 2021;2021(1):omaa104. doi:10.1093/omcr/omaa104.
2. Gerges P, Mian A, Singh G, Aziz M, Guirguis S, Koteish A. Agenesis of the intrahepatic inferior vena cava: a case report and literature review. Cureus. 2023;15(2):e35589. doi:10.7759/cureus.35589.
3. Williams GJ, Chowdhury P, Packham S. Inferior vena cava agenesis: a rare cause of deep vein thrombosis. BMJ Case Rep. 2022;15(12):e250511. doi:10.1136/bcr-2022-250511.
4. Alende Castro V, Díaz-Peromingo JA. Vena cava atresia and deep vein thrombosis: a case report and systematic review. Int J Angiol. 2021;31(2):88-91. doi:10.1055/s-0041-1732434.
5. Osti N, Racanelli V, Susca N, Martinelli N, Maino A. Direct oral anticoagulants in deep vein thrombosis associated with inferior vena cava agenesis: a report of three cases and a systematic review. Int J Cardiol Heart Vasc. 2025;56:101565. doi:10.1016/j.ijcha.2024.101565.
6. Salgado Ordóñez F, Gavilán Carrasco JC, Bermúdez Recio F, Aguilar Cuevas R, Fuentes López T, González Santos P. Absence of the inferior vena cava causing repeated deep venous thrombosis in an adult—a case report. Angiology. 1998;49(11):951-956. doi:10.1177/000331979804901113.
7. Kondo Y, Koizumi J, Nishibe M, Muto A, Dardik A, Nishibe T. Deep venous thrombosis caused by congenital absence of the inferior vena cava: report of a case. Surg Today. 2009;39(3):231-234. doi:10.1007/s00595-008-3830-7.
8. Reslan OM, et al. Congenital absence of inferior vena cava in a young patient with iliofemoral deep venous thrombosis treated with ultrasound-accelerated catheter-directed thrombolysis: case report and review of the literature. Ann Vasc Surg. 2015;29(8):1657.e9-1657.e15. doi:10.1016/j.avsg.2015.05.018.
9. Li SJ, Lee J, Hall J, Sutherland TR. The inferior vena cava: anatomical variants and acquired pathologies. Insights Imaging. 2021;12:123. doi:10.1186/s13244-021-01066-7.
10. Spentzouris G, Zandian A, Cesmebasi A, et al. The clinical anatomy of the inferior vena cava: a review of common congenital anomalies and considerations for clinicians. Clin Anat. 2014;27:1234-1243. doi:10.1002/ca.22445.


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