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CASE REPORT

Extensive inferior vena cava thrombosis extending to the right atrium: a case report

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ABSTRACT

BACKGROUND: Inferior vena cava (IVC) thrombosis is an uncommon manifestation of venous thromboembolism, occurring in 2.6% to 4.0% of patients with lower-extremity deep vein thrombosis. Hyperhomocysteinemia has been proposed as a risk factor for venous thromboembolism, though its independent contribution remains controversial.

CASE REPORT: We report the case of a 24-year-old male with a history of vitiligo and Graves' disease who developed extensive IVC thrombosis extending into the right atrium during hospitalization for community-acquired pneumonia. The initial presentation included new-onset left lower-extremity edema on hospital day 7. Imaging revealed bilateral iliac and femoral deep vein thrombosis, complete occlusion of the left lower-extremity deep venous system, partially occlusive IVC thrombosis extending to the right atrium, and an acute pulmonary embolism. Transthoracic echocardiography confirmed a floating right atrial thrombus. A thrombophilia workup identified elevated plasma homocysteine (40 µmol/L; upper limit of normal 18.5 µmol/L) with normal vitamin B12 and folate levels. Testing for hereditary thrombophilias, malignancy, and autoimmune conditions was negative. The patient was treated conservatively with therapeutic anticoagulation (unfractionated heparin followed by low-molecular-weight heparin for 3 months, then acenocoumarol) and elastic compression stockings.

After 5 years of follow-up, he remains asymptomatic without residual edema. Serial imaging demonstrated resolution of thrombus in the suprahepatic IVC and right atrium, with persistent residual thrombosis and endoluminal synechiae in IVC segments, and progressive development of venous collateral circulation.

CONCLUSION: This case demonstrates that extensive IVC thrombosis with right atrial extension can be successfully managed with anticoagulation alone in hemodynamically stable patients. Moderate hyperhomocysteinemia was the only identified risk factor, underscoring the importance of comprehensive thrombophilia evaluation in young patients with extensive venous thromboembolism. Although the role of hyperhomocysteinemia as an independent risk factor remains debated, it may contribute to thrombotic risk in young patients without other identifiable causes.

Keywords: Deep vein thrombosis; Inferior vena cava thrombosis; Thrombosis;

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BACKGROUND

Inferior vena cava (IVC) thrombosis is part of the clinical and pathological spectrum of deep vein thrombosis (DVT). IVC thrombosis remains underrecognized, partly because of low clinical suspicion and limited diagnostic confirmation. The lifetime incidence of DVT is approximately 0.1%, and IVC thrombosis occurs in 2.6% to 4.0% of patients with confirmed lower-extremity DVT. However, the true incidence may be underestimated because of the lack of standardized detection methods.[1]

The etiological factors of IVC thrombosis are similar to those of DVT. They can be divided into congenital and acquired causes, encompassing all elements of Virchow's triad: venous stasis, endothelial injury, and hypercoagulability. Hyperhomocysteinemia has been established as a risk factor for venous thromboembolism, particularly in young adults.[2,3] We present a case of a young male with moderate hyperhomocysteinemia and extensive IVC thrombosis extending into the right atrium.

CASE REPORT

A 24-year-old male with a past medical history of vitiligo and Graves' disease was admitted to a peripheral hospital with community-acquired pneumonia. On the seventh day of hospitalization, he developed new-onset left lower-extremity edema and was transferred to our vascular surgery service for suspected DVT.

Upon admission to the emergency department, venous duplex ultrasound of the lower extremities revealed bilateral iliac and femoral DVT, with complete occlusion of the deep venous system in the left lower extremity. To assess cranial extension of the thrombus, thoraco-abdomino-pelvic computed tomography angiography was performed, demonstrating partially occlusive IVC thrombosis extending to the right atrium, areas of pulmonary infarction, and acute pulmonary embolism (Figure 1). Transthoracic echocardiography confirmed a floating thrombus in the right atrium.

The patient was hemodynamically stable but febrile (39°C) and had mild inflammatory marker elevation (leukocytosis and elevated C-reactive protein). Therapeutic anticoagulation with unfractionated heparin was initiated, along with elastic compression stockings (23-32 mmHg) on both lower extremities.

Thrombophilia workup revealed elevated plasma homocysteine (40 µmol/L; upper limit of normal 18.5 µmol/L), with normal vitamin B12 and folate levels. No other hereditary or acquired causes of venous thrombosis were identified. Immunological studies were negative, malignancy screening was unrevealing, and thrombophilia testing was negative, including testing for the factor V Leiden mutation, the prothrombin G20210A mutation, and coagulation factor levels. Organic acid analysis was consistent with moderate hyperhomocysteinemia.

The patient was discharged on low-molecular-weight heparin for three months and then bridged to acenocoumarol (target INR 2-3). He was instructed to continue wearing elastic compression stockings, which he wore for 2 years.

Figure 1. Computed tomography angiography demonstrating partially occlusive IVC thrombosis extending to the right atrium.

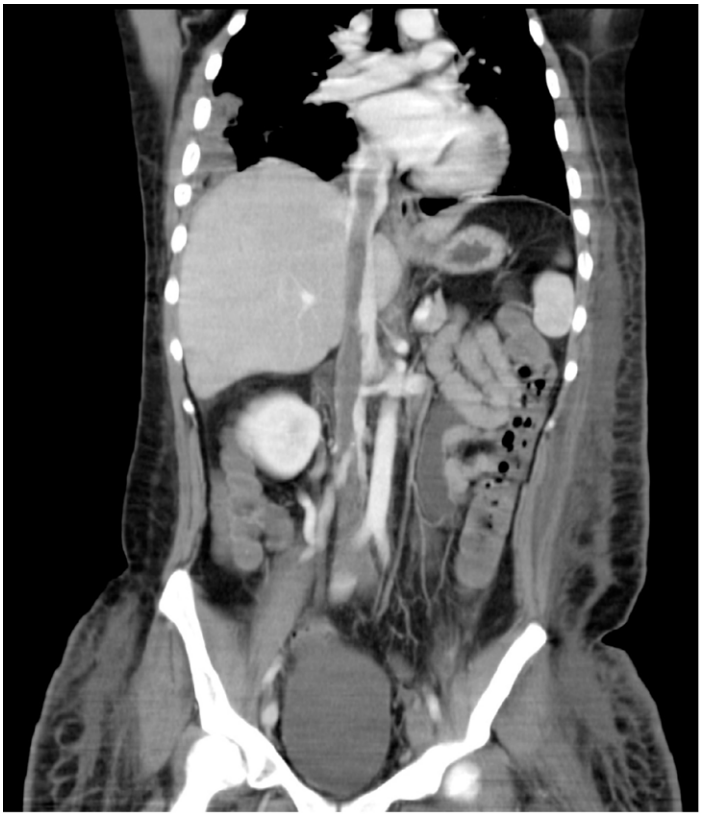
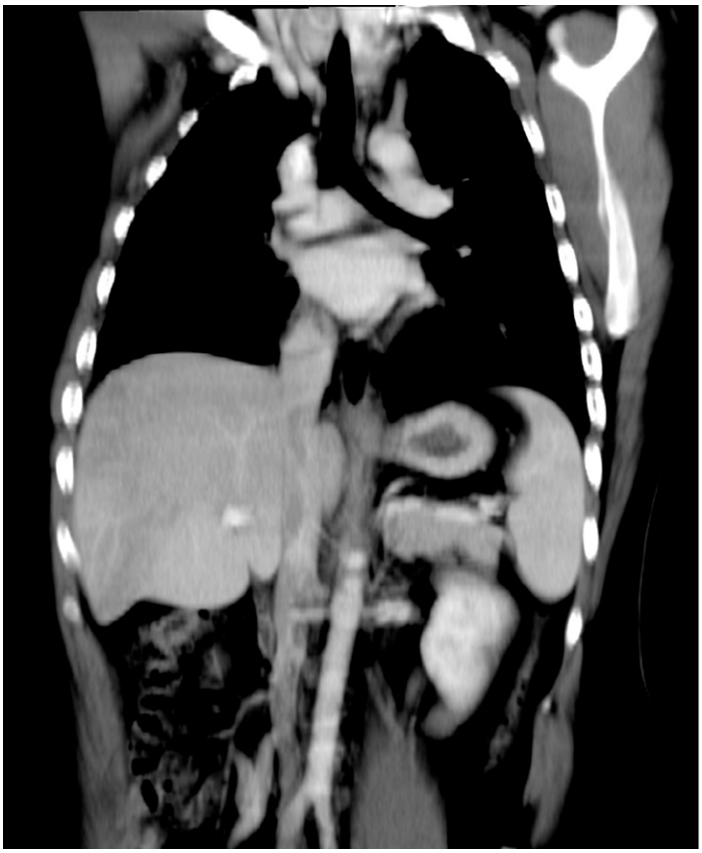


Figure 2. Computed tomography angiography at 5-year follow-up, showing thrombus dissolution in the suprahepatic portion of the IVC and the right atrium, with persistent residual thrombosis.



During follow-up, he showed significant improvement in left lower-extremity edema. After 5 years of follow-up, he remains asymptomatic without residual edema. Serial imaging studies showed thrombus dissolution in the suprahepatic portion of the IVC and the right atrium, with persistent residual thrombosis and endoluminal synechiae in the remaining IVC segments (Figure 2). Progressive increases in both the caliber and extent of venous collateral circulation were evident on successive imaging studies.

The pathogenesis of venous thrombosis encompasses the factors described in Virchow's triad: venous stasis, endothelial damage, and hypercoagulability. IVC thrombosis shares the etiological factors of DVT and can be divided into congenital and acquired causes.[2]

Congenital IVC anomalies are rare, occurring in 0.5% to 1% of the general population, and most cases remain asymptomatic because of well-developed collaterals. These anomalies result from the failure of paired embryonic veins to fuse into a unilateral right-sided venous system during the sixth to eighth week of gestation. IVC thrombosis is prevalent in 60% to 80% of patients with congenital IVC anomalies.[1]

Acquired causes of IVC thrombosis include malignancy (present in 37.5% to 39% of cases), iatrogenic factors (IVC filters, catheters, surgical procedures), trauma, external compression, and hypercoagulable states.[2,4,5] Inherited thrombophilias, including factor V Leiden, prothrombin gene mutation, and deficiencies of protein C, protein S, or antithrombin, increase the risk of venous thromboembolism.[4]

Hyperhomocysteinemia is characterized by elevated plasma homocysteine levels. Homocysteine, an intermediate amino acid formed during methionine-to-cysteine conversion, is metabolized via transsulfuration (requiring vitamin B6) or remethylation (requiring vitamin B12 and folate). Approximately 5% to 7% of the general population exhibits mild elevations in plasma homocysteine concentration.[2] The most common genetic alteration leading to hyperhomocysteinemia is the thermolabile variant of methylenetetrahydrofolate reductase (MTHFR), which results in decreased enzymatic activity.[6]

The association between moderate hyperhomocysteinemia and venous thrombosis was first described in 1995. A landmark study by Ospina-Romero et al. found that hyperhomocysteinemia (defined as plasma homocysteine >18.5 µmol/L) was associated with a 2.5-fold increased risk of deep vein thrombosis (95% CI, 1.2-5.2).[5] However, more recent studies that adjusted for confounding factors have questioned this association, and randomized trials of homocysteine-lowering therapy have shown no benefit in reducing VTE risk.[7]

The clinical presentation of IVC thrombosis varies by the level, acuity, and degree of venous obstruction. More than half of patients present with bilateral lower-extremity edema and superficial abdominal vein engorgement. Associated signs and symptoms may include lower back pain, nephrotic syndrome, hematuria, hepatic congestion, heart failure, pulmonary embolism, fever, and elevated inflammatory markers. When IVC thrombosis is extensive and involves the pelvic and femoral veins, the risk of phlegmasia cerulea dolens and venous gangrene increases.[2]

Diagnostic imaging has advanced significantly. Although venous duplex ultrasound remains the first-line modality for diagnosing DVT, computed tomography venography and magnetic resonance venography provide superior assessment of the extent of IVC thrombosis, anatomical relationships, and thrombus age. Contrast venography remains the gold standard but is invasive and carries a 2% to 10% risk of post-procedural DVT.[2]

Treatment of acute IVC thrombosis aims to remove the thrombus, prevent propagation with anticoagulation, and reduce the risk of pulmonary embolism. Anticoagulation is the mainstay of treatment. Unfractionated heparin or low-molecular-weight heparin is typically initiated at diagnosis to reduce the risk of embolization and thrombus propagation. Patients with acute (within 14 days) or subacute (15-28 days) presentations who are not at high bleeding risk may benefit from catheter-directed thrombolysis or pharmacomechanical catheter-directed thrombolysis, with or without percutaneous transluminal angioplasty and stenting. Direct oral anticoagulants have emerged as preferred agents for long-term VTE treatment, demonstrating improved safety compared with conventional anticoagulation (relative risk of major bleeding 0.61; 95% CI, 0.45-0.83) and similar recurrence risk.[9]

In our patient, the decision to pursue anticoagulation alone rather than surgical or interventional approaches was based on hemodynamic stability, the absence of limb-threatening ischemia, and the extensive thrombosis. The excellent long-term outcome, with complete symptom resolution and the development of robust collateral circulation, supports this conservative approach in selected patients.

The relationship between this patient's vitiligo, Graves' disease, and his thrombotic event remains unclear. Although autoimmune conditions can be associated with hypercoagulable states, particularly antiphospholipid syndrome, our patient's immunological workup was negative. Moderate hyperhomocysteinemia with normal B12 and folate levels suggests a possible genetic variant affecting homocysteine metabolism, though MTHFR testing was not performed.

CONCLUSION

This case illustrates IVC thrombosis in a young adult with moderate hyperhomocysteinemia as the sole identified risk factor. Despite extensive thrombosis extending into the right atrium, conservative management with anticoagulation and compression therapy resulted in excellent long-term outcomes.

This case underscores the importance of considering IVC thrombosis in young patients with bilateral lower-extremity DVT and the need for a comprehensive thrombophilia evaluation. Although the role of hyperhomocysteinemia as an independent VTE risk factor remains debated, it may contribute to thrombotic risk in young patients without other identifiable causes. Long-term anticoagulation with appropriate monitoring can yield favorable outcomes even in extensive IVC thrombosis.

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