**Absence at Birth**

**An Unusual Case of Deep Vein Thrombosis**

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**Foreword**

Information about a real patient is presented in stages (bold-face type) to expert clinicians (Drs. Beckman and Sobieszczyk), who respond to the information and share their reasoning with the reader (regular type). A discussion by the authors follows.

**Patient presentation:** An otherwise healthy 29-year-old white woman presented to our emergency department with leg pain and edema. She was well until 2 weeks before, when she developed lower back pain following a 3-hour flight. Her pain progressed despite treatment with ice packs, nonsteroidal anti-inflammatory drugs, and stretching, and it became so severe that she was unable to sit comfortably at work. Five days before presentation, she began noticing tightness in both thighs and subtle swelling of both legs, as well. Over the subsequent 48 hours, she experienced rapidly worsening bilateral lower extremity swelling, primarily in her thighs, accompanied by pain in her legs. She denied dyspnea, palpitations, chest pain, abdominal distension, or lightheadedness. Her symptoms worsened to the point where ambulation was difficult, thus prompting her visit to the emergency department.

Additional medical history was notable for no previous illnesses or surgeries. Medications included ethinyl estradiol and levonorgestrel (a combination oral contraceptive). She had no known drug allergies. She denied tobacco or illicit drug use and consumed alcohol in moderation. There was no family history of venous thromboembolism (VTE), stroke, or sudden death.

**Dr. Beckman:** Lower extremity edema is commonly seen in both right- and left-sided heart failure, and in individuals with advanced liver and kidney disease, as well. However, this case is notable for the rapid progression of her symptoms, and the lack of dyspnea or other symptoms suggestive of cardiopulmonary disease. Her description of severe pain, tension, and swelling in her extremities indicates significant impairment to venous drainage, which is most likely caused by deep vein thrombosis (DVT). Although she denies a personal or family history of VTE, her use of a combination oral contraceptive1 and her recent air travel,2 increase her risk of DVT. In severe cases, DVT can lead to the development of phlegmasia cerulea dolens, whereby obstruction of venous outflow causes edema and a rise in interstitial pressure to such a degree that it impairs arterial perfusion of the extremity. Therefore, physical examination of the lower extremities should include a thorough pulse and neurological assessment to determine whether there is evidence of neurovascular compromise, and the diagnosis of DVT should be confirmed by noninvasive duplex ultrasonography. In addition, I would look for evidence of acute right heart dysfunction, such as a right ventricular heave or murmur consistent with tricuspid regurgitation, in case there is a concomitant pulmonary embolism (PE).

**Patient presentation (continued):** On arrival, her vital signs included a temperature of 98.8°F, heart rate of 118 beats per minute, blood pressure of 118/67 mm Hg, respiratory rate of 16 breaths per minute, and an oxygen saturation of 99% on room air. She was healthy appearing but in obvious pain. Physical examination revealed a jugular venous pressure of 6 cm H2O. Her lungs were clear without rales or wheezes. Cardiac examination showed a regular, tachycardic rhythm with a normal S1 and a physiologically split S2. There were no murmurs or gallops, and her point of maximal impulse was nondisplaced. Her abdomen was soft and nontender with no evidence of hepatosplenomegaly or ascites. Examination of her lower extremities was notable for bilateral pitting edema, primarily of the thighs, that was worse on the left and extended proximally to her hips. Both legs were notably warmer than other portions of her body with no palpable venous cords. Femoral, popliteal, dorsalis pedis, and posterior tibial arterial pulses were 2+ bilaterally. Skin examination was notable for livedo reticularis on her left thigh with no vascospasm or spider veins. Neurological examination showed intact sensation to light touch in her legs, although further assessment was limited by severe pain. She was unable to ambulate without assistance because of pain.

Laboratory studies were notable for a sodium concentration of 133 mmol/L (normal, 136–145) with otherwise normal electrolytes and renal function. Hematocrit was 31.2% (normal, 36%–48%) with a partial thromboplastin time of 25.6 seconds (normal, 23.8–36.6), and an international normalized ratio of 1.1 (normal, 0.9–1.1). ECG showed normal sinus rhythm at 84 beats per minute with sinus arrhythmia and diffuse T-wave flattening, as well. A lower extremity venous duplex ultrasound study was...
performed to evaluate for DVT. This showed extensive thrombus with a lack of venous compressibility (Figure 1), and absent respiratory variability (Figure 2), during Doppler ultrasonographic assessment of the bilateral external iliac and common femoral veins. These findings are consistent with obstructing DVTs in these segments. Contrast-enhanced chest computed tomography (CT) showed no evidence of PE.

**Dr Beckman:** Her examination is notable for sinus tachycardia, although she is hemodynamically stable at present. Although sinus tachycardia is frequently associated with PE,3 the normal chest CT essentially excludes this as a significant possibility, and her severe pain could readily explain the tachycardia. Her examination reveals no other signs suggestive of cardiopulmonary or hepatic disease, and her ECG changes are nonspecific. The fact that her lower extremity edema is bilateral and extends to her thighs is suggestive of proximal venous obstruction. Despite the livedo pattern apparent on her thigh, her warm skin, normal peripheral pulses, and intact sensation argue against neurovascular compromise of the limb that would warrant emergent intervention to prevent tissue loss. She has no evidence for C1 (telangiectasias or reticular veins) or C2 level venous insufficiency (varicose veins) of the Clinical-Etiology-Anatomy-Pathophysiology classification system. Indeed, there are no stigmata of longstanding venous hypertension on examination, although this could be masked by her significant lower extremity edema at present. Her laboratory analysis is notable for mild hyponatremia, which is likely secondary to either dehydration in the setting of acute illness or the syndrome of inappropriate antidiuretic hormone from severe pain. Her mild anemia is not unusual in young women and could be related to menstrual blood loss and iron deficiency; however, this warrants additional confirmatory testing.

As expected, ultrasound imaging confirmed the presence of DVT. Although we have identified her VTE risk factors of recent travel and combination oral contraceptive use, it is unusual for individuals with this risk profile alone to present with such an extensive, proximal DVT burden without distal extension. Moreover, air travel of this short duration is not commonly associated with VTE in otherwise healthy persons.4 A congenital or acquired thrombophilia would certainly put her at additional risk for DVT. However, the proximal, bilateral clot distribution raises concern for common iliac vein or inferior vena cava (IVC) occlusion, compression, or obstruction. I would proceed with axial imaging, either CT or magnetic resonance (MR) imaging, to define her pelvic and abdominal venous anatomy, because this will help determine the optimal management strategy. In addition to providing adequate analgesia, I would initiate anticoagulation with intravenous unfractionated heparin. This will permit rapid reversibility in case a procedure is ultimately required, and it may also provide a therapeutic benefit over other forms of anticoagulation because of its anti-inflammatory effects.5 In addition to leg elevation, she should also have either compression stockings or tight elastic wraps placed on both extremities to help alleviate pain and swelling.

**Patient presentation (continued):** The patient received a bolus and continuous infusion of unfractionated heparin with a goal partial thromboplastin time of 60 to 80. Thigh-high compression stockings were placed on both legs, and...
her extremities were kept elevated while in bed. In addition, she required frequent administration of intravenous hydromorphone for pain control. The patient underwent abdominal and pelvic CT imaging with contrast to evaluate for proximal venous obstruction. The study showed complete agenesis of the IVC with an extensive system of serpiginous venous collaterals providing drainage from the abdomen (Figure 3). In addition, there was marked dilatation of the azygos, hemiazygos, and lumbar veins, which provided collateral lower extremity, pelvic, and abdominal venous drainage (Figure 4A and 4B).

Dr Beckman: Despite the presence of venous collaterals, the lack of a normal IVC would increase the venous pressure in her extremities and create an anatomic risk factor for DVT. She may have developed small DVTs over time that affected some of these collateral channels, but her oral contraceptive use and immobility during travel may have served as additional triggers for this acute DVT event. The absence of a PE is not surprising, because the small caliber and serpiginous nature of these channels likely prevented embolization of distal thrombus. This anatomic finding is similar to the effect of the Adams-DeWeese IVC clip, which was developed as a surgical means of interrupting the IVC to prevent PE in patients at high risk.6

Given her extensive iliofemoral thrombus burden and marked venous hypertension, she is at risk for developing postthrombotic syndrome (PTS).7 This entity is characterized by ongoing pain, edema, leg fatigue, and heaviness following DVT, and it can develop despite appropriate anticoagulation. In addition, with anticoagulation alone, she would likely require many more days of hospitalization before her pain has improved to permit independent ambulation, and a significant period of limited activity because of the pain before she would be able to return to her previous functional status. Therefore, I would discuss the role of catheter-delivered thrombolysis (CDT) with the patient and consult an interventionalist.

Patient presentation (continued): The patient was brought to the catheterization suite and underwent invasive venography via the popliteal veins. This revealed thrombotic occlusion of the right common femoral vein at the inferior aspect of the femoral head (Figure 5A), and thrombotic occlusion of the left femoral vein within the proximal segment, as well (Figure 5B). A wire was passed beyond the thrombus, and venography at this level showed numerous venous collaterals draining the occluded right and left femoral and iliac system (Figure 5C). Venography also revealed bilateral lumbar veins draining the lower extremities that were also thrombosed. The venous pressure was elevated at 18 mm Hg on the right and 27 mm Hg on the left.

Dr Sobieszczyk: The venogram confirms the findings from her CT imaging. She has impressive iliofemoral DVT burden with thrombus extending into an extensive collateral system.
including large lumbar veins substituting for the IVC with respect to lower extremity drainage. In addition, the venous pressure distal to the thrombosed segments is markedly elevated. It is difficult to determine what degree of her thrombus burden is acute versus chronic. Over time, thrombus becomes more organized and less amenable to thrombolysis. However, she clearly developed rapidly progressive symptoms over a short time period, which indicates that at least a portion of the thrombus is acute. Given her ongoing pain and edema despite maximal medical therapy, and her risk of PTS, as well, I recommend proceeding with CDT (Figure 6).

During lower extremity CDT, a catheter is inserted into the venous system and advanced to the thrombosed venous segment. In some cases, a thrombolytic agent (typically alteplase) can be passively infused in the hopes of restoring venous patency. However, depending on the thrombus burden and chronicity, passive infusion may not be sufficient. Numerous devices have been developed to facilitate thrombus removal through mechanical means in conjunction with pharmacological thrombolysis. This can be achieved by vacuum aspiration, by high-pressure saline jets directed at the thrombus (rheolyis) in conjunction with aspiration, or by devices that use acoustic energy to disrupt thrombus. In general, CDT uses a lower dose of thrombolytic agent relative to systemic thrombolysis. Additionally, depending on the final results, CDT permits venous stenting if necessary in the hopes of maintaining long-term patency.

**Patient presentation (continued):** Pulse spray thrombectomy was performed in each lower extremity. As part of the procedure, the device was advanced through the common femoral veins into the common iliac veins bilaterally by using 10 mg of alteplase divided between both legs. Infusion catheters were then advanced to the level of the lumbar vein on each side. These were left in place with

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**Figure 5.** A, Invasive venography showed contrast staining and visualized thrombus (arrow) beginning in the right common femoral vein. B, Venography of the left extremity revealed a long segment of occlusion (arrow) in the femoral vein with proximal reconstitution (arrowhead). C, Proximally on the left, venography showed thrombus within the common femoral and iliac veins (arrow) with drainage extending through a lumbar vein (arrowhead) rather than the IVC. IVC indicates inferior vena cava.

**Figure 6.** Clinical algorithm for management of deep vein thrombosis. DVT indicates deep vein thrombosis.
concomitant infusion of alteplase at a rate of 0.75 mg/h in each catheter. After 20 hours, the patient underwent follow-up venography. This showed patent right-sided femoral, common femoral, and iliac veins (Figure 7A and 7B) terminating in a system of collaterals with residual thrombus in the lumbar vein. On the left side, there was improved patency in the common femoral and iliac veins (Figure 7C) with a denser network of pelvic collaterals than seen before thrombolysis (Figure 7D).

**Dr Sobieszczyk:** Follow-up venography after thrombolysis is routinely performed to both assess the efficacy of the progress and to determine whether additional interventions, such as venous stenting, are necessary. Not surprisingly, in this case, there remains some residual thrombus, which points to the chronicity of her illness. Catheter-directed thrombolysis is most effective when the thrombolytic eluting catheter spans the thrombosed segment and connects the patent distal and proximal veins. In this case, congenital absence of the IVC did not allow advancement of the catheter to a proximal, thrombus-free vein. However, there is clearly an overall reduction in thrombus burden with improvement in venous collateralization, which also likely improved her local venous hypertension and her symptoms of pain and swelling. Given the persistence of her underlying anatomic anomaly, and her residual thrombus, as well, she will likely require indefinite anticoagulation to reduce her risk of recurrence.

**Patient presentation (continued):** Following the procedure, the patient was transitioned to rivaroxaban 20 mg daily with plans for indefinite anticoagulation. Testing for heritable and acquired thrombophilias was deferred, because the results would not alter management in this case. Her pain improved dramatically, and she was quickly weaned from narcotics. She returned home 48 hours later with instructions to continue wearing thigh-high compression stockings rated at 30 to 40 mmHg. At follow-up 2 months later, she denied any lower extremity pain and noted only mild thigh swelling with strenuous exercise that resolved quickly with leg elevation.

**Dr Beckman:** In this case of an otherwise healthy young woman with severe, symptomatic DVT, detailed history taking did not reveal an obvious explanation for her significant thrombus burden. Abdominal imaging was critical in defining an anatomic variant that places her at increased lifelong risk for DVT. Careful attention to her unusual presentation not only yielded an explanation for her illness but also helped guide her ultimate therapy. Hopefully, with lifelong anticoagulation, ongoing use of compression stockings, and the judicious use of catheter-directed thrombolysis, she will not develop more severe symptoms of PTS and will not experience a recurrent DVT.

**Discussion**

The annual global incidence of lower extremity DVT is ≈1.6 per 1000 individuals with a greater risk among men and the elderly.8,9 Risk factors include hospitalization, immobility (particularly following surgery or trauma), malignancy, congenital or acquired thrombophilias, pregnancy, obesity, and estrogen therapy.10,11 DVTs most commonly originate in the calf veins, although they can subsequently extend into more proximal segments.12 By the time symptoms develop, thrombus has typically progressed to also involve the popliteal, femoral, and iliac beds.13

Iliofemoral DVT has additional long-term implications for patients. Over 3 months of follow-up, iliofemoral DVT is associated with a 2.4-fold increased risk of recurrence in comparison with patients without iliac involvement.14 Similarly, patients with iliac involvement have a higher rate of PTS at 2 years despite therapy.7 Isolated iliofemoral DVT is uncommon and warrants special consideration. In a previous series of 542 patients with symptomatic DVT, proximal thrombus was associated with contiguous extension into the calf vessels in 99% of cases, and there were no instances of isolated iliac or common femoral DVT.13 Anatomic abnormalities are frequently associated with iliofemoral DVT. A single-center study of 56 cases of...
iliofemoral DVT used CT venography to identify a predisposing anatomic variant in 80% of these individuals. These variants led to decreased lower extremity venous return through a variety of mechanisms, some of which include left common iliac vein compression by the right common iliac artery (May-Thurner syndrome), tumor, venous compression by vertebral body bone spurs, and congenital venous anomalies (Table 1).

Nearly 60 distinct congenital IVC anomalies have been described in the literature. Although some are associated with various congenital heart defects, many exist in isolation and are clinically silent because of collateral drainage through the azygos, hemiazygos, and paravertebral systems. Some variants, such as a left-sided IVC or a duplicated IVC, are relatively common and present in up to 0.5% and 3% of the general population, respectively. However, agenesis of the IVC, as in this patient, is exceedingly rare with an estimated prevalence of 0.0005% to 0.001%. It occurs because of failed embryological development of a connection between the subcardinal and hepatic venous systems, although there is

| Table 1. Anatomic Variants Associated With Increased Risk of Iliofemoral DVT |
|--------------------------------|---------------------------------|---------------------------------|-----------------|------------------|-----------------|
| May-Thurner syndrome | Tumor compression | Vertebral body bone spurs | Congenital IVC/femoral vein anomalies | Retroperitoneal fibrosis | Postpartum uterus |
| Radiation changes |

DVT indicates deep vein thrombosis; and IVC, inferior vena cava.

Table 2. Anticoagulation Options for Deep Vein Thrombosis

<table>
<thead>
<tr>
<th>Agent</th>
<th>Mechanism of Action</th>
<th>Administration Route</th>
<th>Benefits</th>
<th>Limitations</th>
<th>Dosage</th>
</tr>
</thead>
<tbody>
<tr>
<td>Unfractionated heparin&lt;sup&gt;23&lt;/sup&gt;</td>
<td>Activation of antithrombin III; inactivation of thrombin and factor Xa</td>
<td>Intravenous; subcutaneous</td>
<td>Rapid onset; cost; widely available; no dose adjustment for renal insufficiency; can be used during pregnancy</td>
<td>Off-label use; intravenous form requires frequent laboratory monitoring; injection site reaction; risk of HIT</td>
<td>80 U/kg bolus followed by infusion at 10 U·kg&lt;sup&gt;-1&lt;/sup&gt;·h&lt;sup&gt;-1&lt;/sup&gt; with titration to PTT of 60–80 s; 333 U/kg subcutaneously followed by 250 U/kg every 12 h</td>
</tr>
<tr>
<td>Low-molecular-weight heparin&lt;sup&gt;24&lt;/sup&gt;</td>
<td>Activation of antithrombin III and inactivation of factor Xa</td>
<td>Subcutaneous</td>
<td>Rapid onset; no laboratory monitoring required</td>
<td>Dose adjustment required for renal insufficiency; injection site reaction; limited data in obese patients; HIT</td>
<td>Varies with agent; dosage based on weight and CrCl</td>
</tr>
<tr>
<td>Warfarin&lt;sup&gt;25&lt;/sup&gt;</td>
<td>Vitamin K antagonist</td>
<td>Oral</td>
<td>Decades of clinical experience; once daily dosing; provides some coverage in setting of noncompliance</td>
<td>Requires laboratory monitoring; multiple food and medication interactions; narrow therapeutic window</td>
<td>Dose varies; titrate to INR 2–3</td>
</tr>
<tr>
<td>Fondaparinux&lt;sup&gt;26&lt;/sup&gt;</td>
<td>Factor Xa inhibitor</td>
<td>Subcutaneous</td>
<td>Rapid onset; no laboratory monitoring required; safe in patients with HIT</td>
<td>Dose adjustment required for renal insufficiency; injection site reaction; cost</td>
<td>5–10 mg daily depending on weight</td>
</tr>
<tr>
<td>Dabigatran&lt;sup&gt;27&lt;/sup&gt;</td>
<td>Direct thrombin inhibitor</td>
<td>Oral</td>
<td>No laboratory monitoring required</td>
<td>Twice daily dosing; dyspepsia; requires initial treatment with parenteral anticoagulation; not studied in patients with CrCl &lt;30 mL/min</td>
<td>150 mg bid after 5–10 days of parenteral anticoagulation</td>
</tr>
<tr>
<td>Rivaroxaban&lt;sup&gt;28&lt;/sup&gt;</td>
<td>Factor Xa inhibitor</td>
<td>Oral</td>
<td>No laboratory monitoring required; once daily maintenance dosing</td>
<td>Not recommended with CrCl &lt;30 mL/min</td>
<td>15 mg bid for 21 days, then 20 mg daily with food</td>
</tr>
<tr>
<td>Apixaban&lt;sup&gt;29&lt;/sup&gt;</td>
<td>Factor Xa inhibitor</td>
<td>Oral</td>
<td>No laboratory monitoring required; less bleeding than warfarin; approved for patients on HD</td>
<td>Twice daily dosing</td>
<td>5 mg bid; 2.5 mg bid if patient has 2 of the following: age ≥80, weight ≤60 kg, serum creatinine ≥1.5 mg/dL</td>
</tr>
<tr>
<td>Edoxaban&lt;sup&gt;30&lt;/sup&gt;</td>
<td>Factor Xa inhibitor</td>
<td>Oral</td>
<td>No laboratory monitoring required; less bleeding compared to warfarin; once daily dosing</td>
<td>Cost; not studied in patients with CrCl &lt;30 mL/min</td>
<td>60 mg daily; 30 mg daily if CrCl 30–50 mL/min or body weight &lt;60 kg</td>
</tr>
<tr>
<td>Sulodexide&lt;sup&gt;31&lt;/sup&gt;</td>
<td>Interaction with antithrombin and heparin cofactor II</td>
<td>Oral</td>
<td>No laboratory monitoring required; no renal adjustment required</td>
<td>Limited data in comparison with other agents</td>
<td>500 lipasemic units bid</td>
</tr>
</tbody>
</table>

*bid indicates twice daily; CrCl, creatinine clearance; HD, hemodialysis; HIT, heparin-induced thrombocytopenia; INR, international normalized ratio; and PTT, partial thromboplastin time.*
some evidence that perinatal or intrauterine thrombosis of the developing venous system may also result in this anomaly.\textsuperscript{20}

Although the prevalence of IVC anomalies among patients with idiopathic DVT is unclear, these vascular anomalies seem to increase the risk of DVT, because they are frequently discovered as part of the workup for idiopathic iliofemoral DVT. In 1 study of 31 patients with idiopathic iliac vein DVT, 5 patients were found to have an IVC anomaly by MR venography.\textsuperscript{21} Within this cohort, the age of presentation of those with IVC abnormalities was significantly younger (25±6 years versus 53±19 years, \( P = 0.002 \)). Similarly, another center identified 10 patients with iliofemoral DVT and IVC agenesis confirmed by MR venography.\textsuperscript{20} The median age of presentation was 25. Fifty percent of these individuals reported a family history of VTE, and additional risk factors within this group included prolonged travel, immobility, trauma, and pregnancy. Interestingly, 8 of these individuals reported intense physical activity followed by back pain just before developing symptoms more typical of DVT.

The mainstay of therapy in iliofemoral DVT is anticoagulation regardless of the underlying precipitant.\textsuperscript{22} There are no data showing superiority of 1 agent over another in iliofemoral DVT or those with IVC anomalies, and the specific agent should be chosen based on the presence of other comorbidities, and patient preference, as well (Table 2).\textsuperscript{23–31}

The duration of therapy must be individualized, but data from randomized, controlled trials show a reduced rate of recurrent VTE with extended-duration anticoagulation.\textsuperscript{32,33} In patients with IVC anomalies and DVT, we recommend indefinite anticoagulation given the persistence of the underlying anatomic risk factor. This strategy is consistent with that of other centers.\textsuperscript{20}

Compression stockings have long been used to reduce the risk of PTS following DVT. Previous trial data have shown a 50% reduction in the development of PTS in patients using thigh-high compression stockings rated at 30 to 40 mm Hg for 2 years following the development of DVT.\textsuperscript{34,35} More recently, however, a placebo-controlled trial comparing therapeutic compression stockings with sham stockings found no reduction in the rate of PTS development at 2 years.\textsuperscript{36} Importantly, only 55.6% of trial participants used compression stockings for \( \geq 23 \) days per week in this study, which could account for the different results in this intention-to-treat analysis. Particularly in patients with extensive iliofemoral DVT and an underlying anatomic impairment to normal venous return, we routinely recommend the use of compression stockings.

Despite aggressive therapy, >40% of patients with DVT will develop PTS symptoms within 2 years of diagnosis.\textsuperscript{7} As a result, investigators have begun examining whether active removal of thrombus and restoration of vein patency can decrease the development of PTS. In the Catheter-Directed Venous Thrombolysis in Acute Iliofemoral Vein Thrombosis (CaVenT) study, 209 patients with acute iliofemoral DVT were randomly assigned to anticoagulation alone or anticoagulation in addition to CDT.\textsuperscript{37} At 24 months, there was a significant reduction in the presence of PTS in individuals who underwent anticoagulation in addition to CDT in comparison with individuals who underwent anticoagulation alone (41.1% versus 55.6%, \( P = 0.047 \)) with an absolute risk reduction of 14.4%. CDT resulted in 3 episodes of major bleeding. The upcoming Acute Venous Thrombosis: Thrombus Removal with Adjunctive Catheter-Directed Thrombolysis (ATTRACT) study will examine the role of CDT in a larger cohort of 692 patients, and this study protocol uses pharmacomechanical thrombolysis rather than the passive infusion catheters predominantly used in previous studies.\textsuperscript{38}

Conclusion

This case demonstrates the importance of recognizing unusual features in a young, otherwise healthy patient presenting with isolated iliofemoral DVT. Careful history taking did not reveal a clear precipitating cause for her dramatic presentation, and the thoughtful evaluation by the clinicians involved in her care led to the diagnosis of a rare IVC anomaly that contributed to her DVT. By identifying this anatomic variant, the clinicians were able to recommend both acute and long-term therapies to reduce her risk of recurrent VTE and PTS. She experienced a rapid improvement in symptoms and was able to return to her previous level of activity in a matter of days.

Disclosures

None.

References


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