

# Inferior vena cava occlusion causing syncope during upper extremity exertion treated with ilio caval venous revascularization

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Inferior vena cava (IVC) thrombosis is rare, but its incidence is increased in those with IVC filters or inflammatory bowel disease. Once the IVC is thrombosed, venous return is via collateral channels on the torso and retroperitoneum. Limitations in this collateral venous return can result in symptoms, usually in the lower extremities. Syncope and dyspnea are rare. We report a patient with a 1-year history of worsening syncope when working with his upper extremities. Iliocaval venous occlusion with lack of accommodation of venous return at the thoracic outlet was diagnosed. Treatment with ilio caval stenting resolved his symptoms. (*J Vasc Surg Cases* 2015;1:208-10.)

Inferior vena cava (IVC) thrombosis occurs in 4% to 15% of patients with confirmed deep venous thrombosis (DVT).<sup>1</sup> However, the exact incidence of IVC thrombosis is difficult to elucidate due to the variable presentation. Patients may be asymptomatic or present with complications of DVT, such as pulmonary embolism (PE). The more common presenting symptoms can be bilateral lower extremity swelling, varicosities, manifestations of venous hypertension, cardiovascular collapse, and dilated abdominal wall veins. We report the first case of syncope due to decreased cardiac preload resulting from lack of thoracic outlet accommodation of venous return. The patient consented to publish the data in this report.

## CASE REPORT

A 79-year-old man presented with vague abdominal pain, dyspnea on exertion, hypotension, and syncope, especially when working with his upper extremities. He had experienced two unprovoked left leg DVTs (1985 and 1987) and was prescribed warfarin for 6 months after the first event. The second DVT occurred off warfarin, and he was prescribed lifelong warfarin. He developed a third (2004) venous thromboembolic event, a PE and another DVT, during a time that his warfarin was poorly monitored. After a negative hypercoagulable workup, his recurrent thrombosis was due to his history of ulcerative colitis (UC). A

Günther Tulip (Cook Medical, Bloomington, Ind) IVC filter was placed due to failure of warfarin.

He had been monitored more closely and had experienced no new events until 9 months before he presented when he noticed left-sided abdominal varicosities. His left-sided abdominal pain worsened throughout the day, especially after meals and during any activity, but subsided at night after lying down. He also had dyspnea when bending over or when lifting his arms above his head, with several episodes of syncope and near-syncope with standing and working in his yard.

His hypotension worsened for 1 to 1.5 years, which led to a cardiac evaluation before his evaluation at our facility. An attempt at a right heart catheterization failed due to the IVC thrombosis, and his cardiologist did not think that his symptoms were related to his heart. Results of Holter monitoring, echocardiography, and tilt table testing were normal. We did not obtain a carotid duplex study before his intervention.

The physical examination demonstrated large left-sided abdominal wall varices (Fig 1). Leg swelling was not observed, but he did have hemosiderin deposition and venous telangiectasias. Arm elevation appeared to trigger near-syncope. When performing the elevated arm stress test, his arms became weak and heavy, he had to lie down, and he could not perform the test for the full 3 minutes. We did not have arterial duplex studies or pulse volume recordings. A computed tomography (CT) scan did not demonstrate stenosis of the arch vessels. He had equal axillary, brachial, and radial pulses.

Ultrasound imaging confirmed IVC occlusion with remote nonobstructive DVT in bilateral iliac and femoral veins. The IVC occlusion resulted in significant abdominal wall collaterals. Although the patient experienced lightheadedness with his arms elevated, nearly passing out, bilateral upper extremity ultrasound imaging did not demonstrate DVT and was negative for venous thoracic outlet syndrome with maneuvers at 90° and 180° abduction. Mesenteric ultrasound imaging demonstrated normal hemodynamics with 0% to 69% stenosis of the celiac, superior mesenteric artery, and internal mammary artery.

Venous return was dependent on return via subclavian veins and anterior abdominal wall collaterals noted on CT scan. The syncopal events might have been subject to the thoracic outlet

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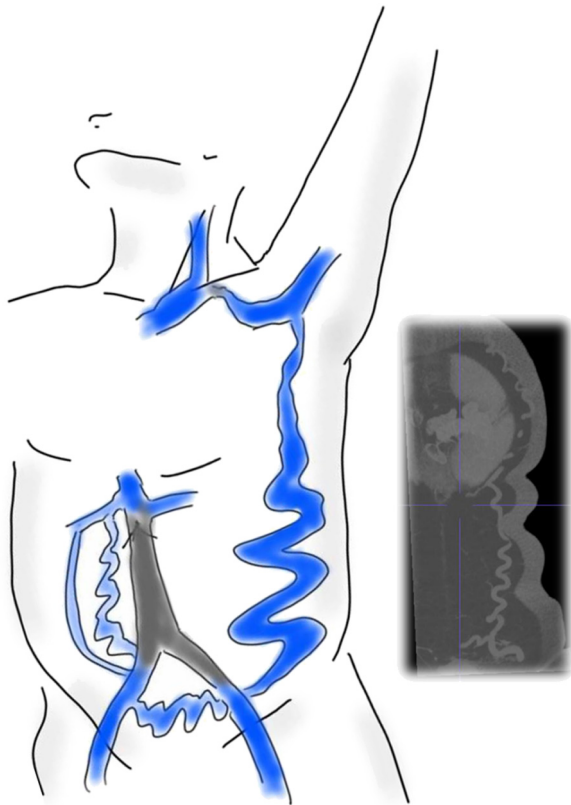
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**Fig 1.** **Left,** Illustration of venous drainage through the patient's axillosubclavian veins. These vessels were prone to occlusion with thoracic outlet compression. **Right,** A computed tomography (CT) three-dimensional reconstruction corroborates the illustration.

not being able to accommodate the venous return from the upper and lower extremities, leading to worsening cardiac preload and ultimately resulting in hypotension. We did not try to reproduce the symptoms with compression of the abdominal wall collaterals. The surgeon used the physical examination to create the illustration in Fig 1, and CT three-dimensional reconstruction corroborated this illustration.

Venacavography showed occlusion of the ilio caval system to the IVC filter, with outflow of the lower extremity veins via pelvic and abdominal wall collaterals and retroperitoneal collaterals. These collaterals were prominent and tortuous, consistent with a chronic obstruction. Wires were passed into the IVC above the filter, and larger sheaths were placed to accommodate the balloons and stents. The IVC was then serially dilated from 14 to 18 mm. The filter was crushed over as well. A 22-mm × 45-mm Wallstent (Boston Scientific, Marlborough, Mass) was deployed across the IVC filter and postdilated to 20 mm. Kissing stents (14-mm × 50-mm Wallstents) were placed and postdilated to 14 mm with adequate overlap into the main IVC stent. These were extended using a 14-mm × 75-mm Wallstent into the external iliac veins and postdilated to 14 mm. These were further extended into the groins with 14-mm × 80-mm S.M.A.R.T. CONTROL stents (Cordis Corp, Bridgewater, NJ). Intravascular ultrasound imaging showed patency of the stents from the common femoral

venous confluence up to the renal confluence. Completion venacavography confirmed good flow once Valsalva was released (Fig 2).

Follow-up 5 months later revealed no further dyspnea, and he was walking without his cane. He denied leg heaviness, hypotension, dyspnea, or near-syncope and was able to work without restrictions. He only complained of abdominal bloating that was more consistent with his UC. On examination, the abdominal wall collaterals were no longer visible.

No postoperative CT scan was available to view the status of the filter, but postoperative visceral venous duplex imaging showed a widely-patent IVC stent. Flow was respirophasic and spontaneous proximal to the stent within the IVC. Bilateral iliac vein and IVC stents were widely patent. We have no plans during his follow-up to repeat venography because the patient is currently asymptomatic.

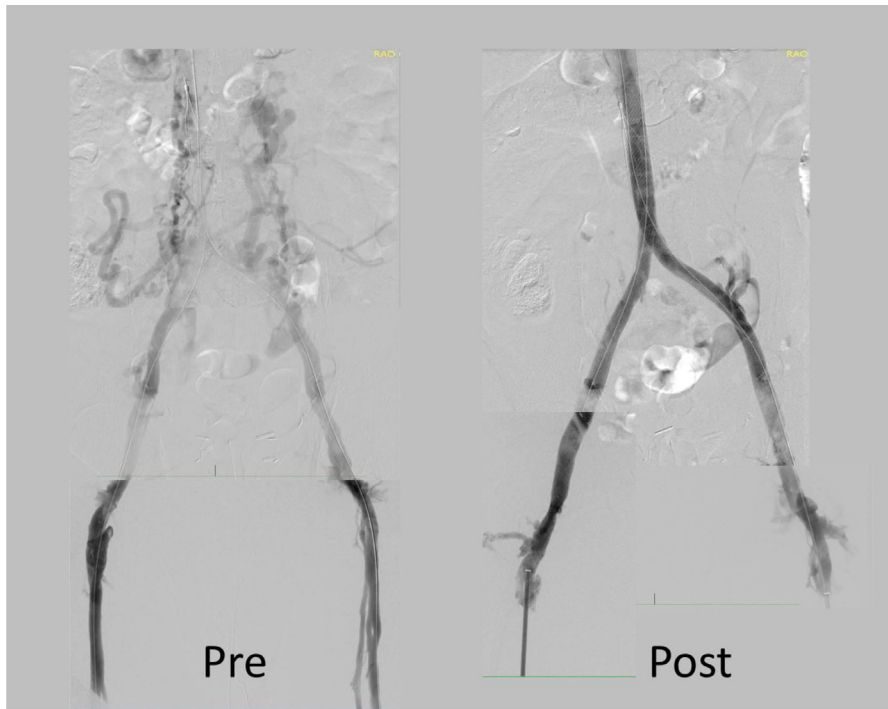
## DISCUSSION

In 1856, Dr Rudolf Virchow suggested the causes of venous thrombosis to include stasis, endothelial injury/dysfunction, and hypercoagulable states.<sup>2</sup> Our patient had an extensive hypercoagulable workup, which did not demonstrate any known clotting disorders. However, thromboembolic event in conjunction with inflammatory bowel disease has been reported over a wide range, from 0.3% to 41%.<sup>3-5</sup> Thus, we believe the patient was hypercoagulable as a result of his inflammatory bowel disease.

IVC filter occlusion has been reported as occurring in 2% to 20% of patients.<sup>6-9</sup> Consideration should be given to removing these filters if lifelong anticoagulation is not indicated and if the filter was placed for prophylaxis. In this patient, there was a clear indication for the filter and lifelong anticoagulation because of PE due to recurrent DVT despite anticoagulation. Subsequent IVC filter thrombosis despite anticoagulation raises the question of whether he will need a filter in the future. For now, he will be maintained on lifelong warfarin anticoagulation with close follow-up. If he becomes symptomatic again, he will need further venography for planning and possible treatment.

## CONCLUSIONS

Patients who have symptomatic ilio caval obstruction should be considered for percutaneous stenting. Our patient had atypical symptoms that have not been described to the best of our knowledge. The preprocedural findings suggested that ilio caval occlusion and venous return were mostly dependent on the left subclavian vein and were the cause of his syncope. Despite an extensive workup of his symptoms, we cannot explain his consistent, repetitive abdominal pain and near-syncope when eating, bending over, or using his upper extremities. His UC may explain the abdominal pain findings. However, treatment of his ilio caval occlusion resolved his symptoms during follow-up. His abdominal wall collaterals are no longer visualized during the physical examination, and he no longer has the abdominal pain or syncope when working with his upper extremities.



**Fig 2.** Left, Preoperative inferior vena cava (IVC) occlusion with significant collaterals compared with (right) postoperative images after recanalization, angioplasty, and stenting of the IVC and iliac veins.

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