Inferior vena cava (IVC) thrombosis associated with filters is not uncommon in clinical practice. However, suprarenal extension of the thrombosis to the hepatic segment of the IVC is very rare, particularly in absence of renal or hepatic tumors (1). Benign intrinsic IVC stenosis is a rare anomaly that has been associated with IVC thrombosis (2). We describe a case of extensive IVC thrombosis secondary to perihepatic IVC stenosis (documented by intravascular ultrasound [IVUS]) and a permanent IVC filter, which was successfully treated by endovascular techniques.

A 38-year-old male smoker presented with back pain and left leg swelling for 1 week. He had no past medical or surgical history except for a motor vehicle accident 18 years ago, which led to left hip surgery and a prophylactic IVC filter placement. He was not taking any medications and had no family history of venous thromboembolism or hypercoagulable state. On physical examination, he...
had a markedly swollen and tender left leg and thigh with moderate skin erythema. He had normal pulses and no evidence of skin breakdown or ulceration. Duplex ultrasound showed acute deep vein thrombosis (DVT) of the left common femoral vein, and no evidence of DVT in the right leg. A computed tomography venogram showed massive IVC filter–associated thrombosis with totally occluded infrarenal IVC and suprarenal extension of the thrombus all the way up to hepatic segment. It also suggested a possible stenosis of the perihepatic IVC (Figure 1).

In view of the severe and disabling symptoms in this young man, we decide to proceed with catheter-directed thrombolysis (CDT). A 50-cm EKOS catheter (EKOS, Bothell, Washington) was positioned from the hepatic segment of the IVC to the left femoral vein, and tissue plasminogen activator was infused over 16 h. While undergoing CDT, he noted complete resolution of his back pain. Post-CDT venography showed complete lysis of the thrombus above the IVC filter and a severe stenosis of the IVC just below the hepatic veins (Figure 2, Online Video 1). The latter was confirmed by IVUS, as well as pressure gradient measurement (Figure 2, Online Video 1). Minimal clot lysis was noted below the filter, and was treated with mechanical thrombectomy followed by balloon angioplasty and stenting. In view of the IVUS findings and a pressure gradient of 18 mm Hg across the lesion in the hepatic IVC, we went on to treat it with a self-expanding (24 × 70 mm) wall stent with excellent angiographic and hemodynamic results (Figure 2). On 1-week follow-up, he had complete resolution of his lower extremity swelling and pain. At 6 months, he continued to be asymptomatic with patent stents and no evidence of recurrent DVT (Figure 3).

In the majority of cases, IVC filter–related thrombosis is seen below or a few centimeters above the
filter (1). Extension of clot all the way up to the hepatic segment is usually seen in patients with hepatic or nephric tumors, which either invade or compress the IVC (1,2). Benign intrinsic IVC stenosis is very rare (<0.1%) but is known to be associated with massive caval thrombosis (2). The most common location for this stenosis is around the perihepatic region of the IVC. The exact pathogenesis of such lesions is not well understood. Some authors suggest that it is a congenital anomaly related to abnormal fusion of the vitelline and the subcardinal veins in utero (3), whereas others propose that it is an acquired condition caused by healed thrombosis producing intravascular membrane or web formations (4).

This case suggests that patients with IVC stenosis who are undergoing IVC filter placement may be at particularly high risk for extensive IVC thrombosis including very high suprarenal extension. Hence, there is a need for the very judicious use of IVC filters in these patients and timely retrieval if they are absolutely indicated. In addition, this case highlights the feasibility of catheter-based treatment of this rare but debilitating complication.

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APPENDIX For accompanying videos, please see the online version of this article.