The treatment of iliofemoral thrombophlebitis has changed little since the initiation of heparin use in the 1930s. Whereas current management strategies focus on the outpatient use of low-molecular-weight heparin, treatment relies on the prevention of clot propagation rather than any effort to resolve the clot burden present. As a result, preservation of valve function is unusual, with most patients resolved to lifelong postphlebitic syndrome, often leading to chronic debilitating swelling and ulceration in the leg.

If practitioners are to make a difference in the chronic debilitating outcomes associated with thrombophlebitis and valve destruction, they must obviously develop a treatment strategy that prevents venous reflux and obstruction. To this end, if the patient presents with a first-time venous thrombosis in the proximal lower-extremity veins, more aggressive treatment in an effort to preserve valve function may be warranted. Modern therapy with percutaneous mechanical thrombectomy and thrombolysis may allow preservation of valve function and avoid the sequelae of chronic venous insufficiency.\(^2\)\(^-\)\(^7\) Although this hypothesis is not yet definitively proven, several studies suggest improved outcomes associated with thrombolysis in deep venous thrombosis (DVT).\(^2\)\(^-\)\(^7\)

Left-leg DVT is nearly twice as common as DVT in the right leg. The etiology of this is related to the anatomic compression of the left common iliac vein by the right common iliac artery, known as May-Thurner syndrome.\(^8\) Therefore, particularly in the proximal venous segments, DVT may be secondary to the anatomic compression that is readily treatable with modern endovascular techniques.\(^9\)\(^-\)\(^17\) For this reason, left iliofemoral DVT warrants aggressive early treatment to prevent valve destruction and possibly relieve anatomic venous compression.\(^18\)

In this case report, we review the treatment of iliofemoral thrombophlebitis in a young male physician with a second episode of left lower-extremity DVT with pulmonary embolus.

**HISTORY**

A 38-year-old male physician presented with a second episode of shortness of breath associated on this occasion with significant swelling of his left leg. Three years earlier, he had suffered a pulmonary embolus with hemodynamic compromise treated by systemic thrombolytic therapy. A CT scan at that time revealed a saddle embolus in his main pulmonary artery (Figure 1). Subsequent workup revealed left calf and popliteal vein DVT without proximal extension. He was treated with low-molecular-weight heparin followed by warfarin for...
6 months. Workup for hypercoagulable disorders was negative, and a family history for clotting disorders was absent. Warfarin was continued for 6 months and then discontinued, and he was placed on 325 mg of aspirin daily.

The patient did well for the intervening period of 3 years, with a convalescent CT scan demonstrating complete resolution of his pulmonary emboli. No further episodes of shortness of breath occurred, and his exercise tolerance was normal. He did notice mild intermittent swelling of the left lower extremity, which resolved with elevation.

On the morning of presentation, the patient noted increased shortness of breath on arising, associated with significant swelling of the left lower extremity. Recognizing the symptoms of recurrent pulmonary embolus, he was sent for an emergency CT scan that once again demonstrated pulmonary emboli (Figure 2), smaller than the previous episode. Duplex ultrasound of the left leg revealed complete occlusion of his left leg venous system, including the left iliac veins. His inferior vena cava appeared to be patent. The patient was systemically placed on heparin.

After careful discussion with the patient concerning the short- and long-term risks of catheter-directed thrombolytic therapy and rheolytic mechanical thrombectomy, he asked to proceed secondary to the debilitating swelling and pain with ambulation associated with his left-leg phlegmasia. This procedure was scheduled for the next morning.

On the second hospital day, the patient was taken to the angiography suite for his procedure. He was placed in a prone position under conscious seda-
tion, and access to his thrombosed popliteal vein was achieved using ultrasound guidance (Figure 3). A 6-F sheath was placed, and a Berenstein catheter (AngioDynamics, Inc., Queensbury, NY) was passed over a Bentson wire. Venography confirmed occlusive DVT from the popliteal to the iliac veins (Figures 4 through 6). A 6-F AngioJet DVX catheter (Possis Medical) was prepared, and thrombolysis was initiated using the Power-Pulse spray technique. A total of 5 mg of tissue plasminogen activator (tPA) was mixed in 50 mL of saline and infused into the clot with the suction port of the AngioJet turned off. An 8-mm X 4-cm angioplasty balloon was inflated repeatedly during a 30-minute dwell period to mix the tPA thoroughly with the clot. No attempt was made to cross the proximal occlusion because this natural cap served to prevent proximal embolization and avoided the need for an inferior vena cava filter.

After 30 minutes, the AngioJet catheter was reinserted, and suction was reactivated. Multiple passes were made in the area of thrombosis with dramatic improvement (Figures 7 and 8). Nonetheless, proximal chronic clot remained, and because we felt this was likely due to May-Thurner compression chronically, the decision to perform extended catheter-directed thrombolysis was made. A 90-cm UniFuse infusion catheter with 50 cm of side holes (AngioDynamics, Inc.) was placed from the popliteal vein to the common iliac vein, and an infusion of tenecteplase at .5 mg/h was begun. During this infusion, the heparin dose was decreased to 300 U/h and was continued overnight.

The next morning, repeat venography was performed (Figures 9 through 12). An inferior vena cavogram showed a widely patent IVC and proximal common iliac vein. Surprisingly, a bifid external iliac vein was seen, which created a flow abnormality in the left leg and appeared to be the cause of the left-leg recurrent DVT. Residual chronic thrombus was seen in this bifid vein, and a decision was made to stent the segment. Two 10-mm X 40-mm Smart stents (Cordis Corporation, Warren, NJ) were placed in a kissing configuration with excellent results (Figures 13

Figure 5. Catheter and wire hanging up in the left iliac vein (reversed due to prone position).

Figure 6. Venogram of the left iliac vein shows occlusive DVT.

Figure 7. Venogram of the popliteal vein after AngioJet percutaneous thrombectomy and Power-Pulse spray infusion.

Figure 8. Iliac vein after AngioJet percutaneous thrombectomy and Power-Pulse Spray infusion.

Figure 9. Popliteal access site after overnight thrombolytic infusion.
and 14). The patient was recommended for lifelong warfarin given his anatomic variation. Follow-up duplex scan at 2 months demonstrated normal venous flow patterns, normal valve closure times in the iliofemoral segment, and widely patent iliac veins.

**DISCUSSION**

This is the first report of a bifid external iliac vein as a cause for recurrent iliofemoral DVT and pulmonary embolus. Although compression of the left iliac vein by the right common iliac artery (May-Thurner syndrome) is common even in a normal population,9,15,19 its association with DVT has been well described. Presumably, this flow abnormality increases blood stagnation and leads to a left-sided predilection for all lower-extremity DVT. In this patient, a flow abnormality was created by a bifid external iliac vein. This anatomic variant has not been previously described in association with DVT or pulmonary embolus. Nonetheless, a flow abnormality from this anatomy clearly would predispose the individual to an increased risk of ipsilateral DVT.

Perhaps the bigger question is why to treat this patient with thrombolysis and rheolytic mechanical thrombectomy in the first place. Clearly, this is a young patient with a
lead a normal healthy life without the chronic complications of lower-extremity venous insufficiency.

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Figure 14. Completion left iliac venogram shows stents in place in the bifid left iliac vein.

long life expectancy. Although anticoagulation alone is likely to prevent further pulmonary emboli, the left-sided veins will likely be obstructed to flow because recanalization is less common in the proximal veins.15,20 Similarly, the valves in the left-leg veins are unlikely to be functional, leading to chronic reflux and further venous hypertension in the lower leg. Postphlebitic syndrome would be expected in the majority, resulting in a chronic debilitating outcome for this patient.

With thrombolysis, valve function can be preserved13,14,22 although long-term prospective data are unavailable. The addition of percutaneous mechanical thrombectomy speeds the clot resolution, hopefully decreasing the time to restoration of normal flow and preventing irreversible valve damage. This adjunct also lowers the dose of thrombolytic necessary to restore flow and decreases the duration of treatment, both hopefully limiting overall bleeding risk. In this way, risk of thrombolysis seems small compared to the benefit of valve preservation, especially in young patients with first‐time episodes of DVT.

Many interventionists believe that the time is right for a new treatment paradigm in proximal DVT.1,4,10,12,23-25 This case exemplifies the results that can be achieved and supports a more aggressive approach to DVT management, particularly in first-time iliofemoral thrombosis. If we can preserve valve function, then the chronic sequelae of DVT can hopefully be avoided. With this more aggressive management, these patients should be able to