Venovenous bypass using continuous renal replacement therapy and endovascular inferior vena cava reconstruction to treat bilateral massive iliocaval deep venous thrombosis

Thomas Heafner, MD,^a Alexander P. Nissen, MD,^a David Schechtman, MD,^a Joseph F. Alderete, MD,^b Zachary M. Arthurs, MD,^c and Brandon W. Propper, MD,^c Fort Sam Houston, Tex; and Bethesda, Md

ABSTRACT

Many inferior vena cava (IVC) anomalies remain asymptomatic because of collateral circulation, but thrombosis of these channels can cause acute deep venous thrombosis with serious sequelae. For those with threatened limbs, anticoagulation is the mainstay of treatment, with endovascular pharmacomechanical thrombolysis replacing open surgical thrombectomy. Described is a severe case of massive iliocaval deep venous thrombosis with bilateral lower extremity Rutherford IIb acute limb ischemia in a patient with congenital IVC atresia. After initial thrombolysis, endovascular IVC reconstruction was accomplished to decompress the lower extremities. The patient ultimately required a right through-knee amputation but remains ambulatory with a prosthetic. (J Vasc Surg Cases and Innovative Techniques 2019;5:438-42.)

Keywords: Inferior vena cava atresia; Iliocaval thrombosis; Deep venous thrombosis; Iliocaval reconstruction; Continuous renal replacement therapy; Venovenous bypass

Many inferior vena cava (IVC) anomalies remain clinically silent because of well-developed collateral circulation; however, thrombosis of these channels can have serious sequelae, including phlegmasia cerulea dolens, renal vein thrombosis, and venous ulceration.^{1,2} Documented is the use of endovascular pharmacomechanical thrombolysis, infradiaphragmatic to supradiaphragmatic continuous renal replacement therapy (CRRT), and endovascular IVC reconstruction to treat a case of massive iliocaval deep venous thrombosis (DVT).

CASE REPORT

A 47-year-old man presented to the emergency department with bilateral lower extremity Rutherford IIb acute limb ischemia. He was unable to move his ankles and toes bilaterally, with sensory loss through the thigh, loss of arterial signals below the femoral arteries, and tight lower extremity compartments. He had no known hypercoagulability and normal results of laboratory evaluation on presentation. Computed tomography (CT) angiography of the chest, abdomen, and pelvis with lower

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extremity runoff demonstrated no acute aortoiliac arterial disease, but there was no contrast opacification below the superficial femoral arteries bilaterally, concerning for occlusion. Although there was no delayed phase, extensive thrombus was seen in the IVC and iliac veins with an associated suprarenal IVC atresia and enlarged collaterals (azygos and lumbar veins; Fig 1). Therapeutic anticoagulation was initiated before proceeding emergently to the operating room.

Given the absence of arterial signals bilaterally, lack of leg swelling, and nonopacification of the infrageniculate vessels on CT, our initial objective was to rule out arterial occlusion. Bilateral groin cutdowns were performed to facilitate open arterial thrombectomy; however, antegrade bilateral lower extremity angiograms confirmed no intra-arterial thrombus. Standard four-compartment bilateral lower extremity fasciotomies were performed with bulging of all compartments and viableappearing muscle.

With arterial disease excluded, the patient's lower extremity symptoms were a consequence of venous hypertension from the iliocaval DVT. Open Fogarty catheter and Esmarch venous thrombectomy were discussed, but an endovascular approach with thrombolysis was chosen primarily to re-establish the patient's collateral outflow. Venous access was obtained in bilateral great saphenous veins (to facilitate groin closure), with venography confirming thrombus extending from the iliofemoral systems into the left renal vein with stagnation of contrast material in the IVC (Fig 2). Transduced pressures obtained from a catheter in the infrarenal IVC compared with the left radial artery line differed by only 20 mm Hg, 100 mm Hg, and 120 mm Hg, respectively. Blood drawn from above the diaphragm vs the IVC demonstrated significant differences in pH (7.35 vs 7.15) and potassium concentration (4.7 mg/dL vs 5.6 mg/dL). Pharmacomechanical thrombolysis with AngioJet (Boston Scientific, Marlborough, Mass) using 20 mg of tissuetype plasminogen activator (tPA) on power-pulse mode

From the San Antonio Military Medical Center,^a and Center for the Intrepid,^b Fort Sam Houston; and the Department of Surgery, Uniformed Services University of the Health Sciences, Bethesda.^c

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Correspondence: Brandon W. Propper, MD, Department of Vascular Surgery, San Antonio Military Medical Center, 3551 Roger Brooke Dr, Fort Sam Houston, TX 78234 (e-mail: brandon.w.propper.mil@mail.mil).

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Fig 1. A, Illustration of the preoperative anatomy with extensive iliocaval thrombus in the setting of a congenital suprarenal inferior vena cava (IVC) atresia that reconstitutes at the level of the hepatic veins. **B**, Coronal image from initial computed tomography (CT) angiography on presentation of the patient.

followed by mechanical thrombectomy mode minimally decreased the thrombus burden. We performed overnight lytic therapy through EKOS catheters (EKOS Corporation, Bothell, Wash) positioned in the azygos vein from right basilic vein access and another spanning the left renal vein and IVC from the left great saphenous vein. Dialysis catheters were placed in the right great saphenous and right internal jugular veins for CRRT, acting as temporary venovenous bypass to offload the lower extremity venous hypertension and to filter and return blood to the heart through the jugular catheter. Pedal signals were unable to be obtained bilaterally before leaving the operating room.

During the first 12 hours in the intensive care unit, bilateral posterior tibial signals returned. There was a drop of 50 mm Hg in venous pressure, assessed through the side arm of the great saphenous vein sheath. The tPA was started at 1 mg/h, and dosing was adjusted to maintain a fibrinogen level >100 mg/dL; serial hemoglobin and platelet levels remained stable. His legs were wrapped and elevated.

Venography at 24-hour follow-up demonstrated persistent thrombus but improved outflow through paraspinal and

azygos collaterals, corresponding to collateral pathways previously visualized on CT. No changes were made to lytic catheter placement. CRRT was continued while venous pressures remained elevated at 30 to 50 mm Hg. On 48-hour follow-up, a channel in the atretic suprarenal IVC was crossed with a stiff Glidewire (Terumo Interventional Systems, Somerset, NJ) and snared from the right basilic vein, achieving through-and-through wire access. This segment was predilated with a 10-mm balloon (Fig 3), and intravascular ultrasound was used to mark the proximal and distal landing zones. Four Wallstents (Boston Scientific) were deployed from the hepatic veins through the left external iliac vein to create a neo-IVC and to cover residual thrombus. Repeated intravascular ultrasound measured a 14-mm lumen in the previously atretic portion and >16 mm throughout the infrarenal IVC and iliac veins (Fig 4). Completion venography confirmed a patent reconstruction with in-line outflow from the lower extremities to the heart (Fig 5).

The only complication during hospitalization was development of an infection in the right fasciotomy wounds requiring



Fig 2. Initial venogram demonstrating significant iliocaval thrombus.

débridement of significant portions of multiple compartments. After discussion with the orthopedic surgery service, the patient elected to proceed with a through-knee amputation, favoring improved mobility with a prosthetic over retaining a nonfunctional limb. He was transitioned off CRRT and hemodialysis with full renal recovery. He was discharged to a Veterans Affairs rehabilitation center and prescribed rivaroxaban. At 6- and 12month follow-up appointments, he ambulated using his prosthetic. Duplex ultrasound of the Wallstents showed no evidence of thrombosis or stenosis.

Consent has been obtained from the patient for this publication.

DISCUSSION

Historically, treatment of an iliocaval DVT focused on reducing angiospasm, preventing propagation of thrombus, and treating underlying conditions.³ Open thrombectomy offered the first paradigm shift, and since the 1990s, treatment has focused on resuscitation, anticoagulation, thrombectomy, and thrombolysis.⁴⁻¹⁰ Whereas contemporary series of open and endovascular venous treatments have demonstrated similar success when used in combination with iliocaval stenting.^{9,10} open thrombectomy is most effective when there is already established outflow through the IVC. In this



Fig 3. Venogram after initial balloon dilation of the atretic inferior vena cava (IVC) segment now with in-line flow back to the heart.

case, open thrombectomy would not address the thrombosed collateral veins needed to re-establish baseline anatomy. Using endovascular thrombolysis, our followup venogram demonstrated flow in several collaterals, which helped to further offload the venous hypertension.

One downside to endovascular treatment is the prolonged time needed for tPA to take effect. To quickly



Fig 4. A, Intravascular ultrasound image in atretic inferior vena cava (IVC) measuring <10 mm in greatest dimension. **B**, Intravascular ultrasound image of the Wallstent in the same location now measures 14 mm.

decrease the lower extremity venous hypertension in our patient, we used a novel technique for jugularfemoral venovenous bypass using a CRRT machine as a pump with infradiaphragmatic outflow and supradiaphragmatic return. A decrease in the IVC pressure from 100 mm Hg to 50 mm Hg was seen during the first 24 hours. This technique also allowed filtration of the lower extremity blood in anticipation of rhabdomyolysis, hyperkalemia, and lactic acidosis. This technique for venovenous bypass was beneficial in our patient but may not be necessary in those who already have in-line outflow from the lower extremities through a patent IVC.

Few recent series discuss the safety and efficacy of IVC recanalization in patients with chronic occlusions. The durability of this technique rivals iliac vein stenting with

4-year follow-up primary, primary assisted, and secondary patency of 52% to 78%, 85% to 87%, and 91% to 93%.^{11,12} When encroaching on the renal or hepatic veins, other groups prefer the Gianturco Z stents (Cook Medical, Bloomington, Ind) as their wide interstices allow better cross-flow, but these were unavailable at our institution. Although we jailed the renal veins, we maximized the interstices of the Wallstent by ballooning them to profile. However, the risk remains for future renal vein obstruction due to pseudointima formation within the stent.

CONCLUSIONS

Aggressive venous thrombolytics and endovascular IVC reconstruction were successful in this unusual case of massive iliocaval DVT and associated IVC atresia. The



Fig 5. Completion venogram demonstrating a patent neo-inferior vena cava (IVC) reconstruction as well as flow from the right common iliac vein.

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technique of venovenous bypass with CRRT was a novel adjunct to decrease the venous pressure while also filtering the metabolic toxins.

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