Treating Common but Complex VTE Scenarios

Drs. Thomas M. Tu, Thomas S. Maldonado, and Suman Annambhotla present case reports using the FlowTriever and ClotTriever Systems for thrombolytic-free, single-session treatments of complex VTE presentations.

WITH THOMAS M. TU, MD, FACC, FSCAI; THOMAS S. MALDONADO, MD, FACS; AND SUMAN ANNAMBHOTLA, MD

Safe and Effective Debulking of Large Thrombus in an Intermediate-Low–Risk PE Patient

Thomas M. Tu, MD, FACC, FSCAI
Cath Lab Director
Baptist Health Louisville
Louisville, Kentucky
ttu@bellsouth.net
Disclosures: None.

Advanced interventional approaches are often not considered when treating patients with intermediate- to low-risk pulmonary embolism (PE). In line with current guidelines, anticoagulation is usually the therapy of choice.[1,2] The low rates of mortality during index hospitalization are often the justification for this conservative strategy. However, adverse events and mortality might be underestimated—possibly due to limited longitudinal follow-up.[3] Recent reports have highlighted surprisingly high 30- and 90-day mortality rates: > 10% in intermediate-risk PE.[4,5] The latest analysis of patients cared for by eight United States PE response teams from the National PERT Consortium registry reported a 30-day mortality rate of 15% in intermediate-low–risk PE, 14% in intermediate-high–risk PE, and 11% even in low-risk PE.[4]

These rates are remarkably similar to those predating catheter-directed PE therapy. A subanalysis of the landmark ICOPER registry, for instance, reported a 90-day mortality rate of 14.7% in patients with nonmassive PE.[6] Although comorbidities might influence mortality rates, a high rate of death during early follow-up among those with intermediate-risk PE can be directly attributed to PE (31.6% in a recent study[5]). This high mortality in supposedly lower-risk patients supports the idea that mortality prediction isn't optimized yet and that currently applied risk stratification tools have significant limitations.

A 2016 meta-analysis indeed demonstrated that updating or shifting cut-off points within scoring systems, especially for normotensive PE, could improve stratification.[7] Additional factors—such as the integration of biomarkers, degree of right ventricular (RV) dysfunction,[8] and presence of concurrent deep vein thrombosis (DVT)[7]—are currently considered in many treatment algorithms. Interestingly, additional studies have also shown that patients left with varying levels of residual pulmonary vascular obstruction (RPVO), as measured by ventilation-perfusion scintigraphy, have a higher risk of death.[9,10] The presence of RPVO (ie, clot remaining after PE treatment) has furthermore been linked to another often overlooked aspect of stratification and treatment: the risk of seriously debilitating long-term complications, including recurrence, readmissions, and adverse events such as heart failure, functional limitation, and chronic thromboembolic pulmonary hypertension (Table 1).[9-20] Unfortunately, RPVO is not a rare occurrence (Figure 1),[10,12,16-19,21,22] and its high prevalence allows for the hypothesis that although patients may not initially die from PE, many might not fully recover.

Supporting this hypothesis, the 2016 INFORM study found that 87% of 7,068 analyzed patients reported at least one pulmonary hypertension–related symptom during 2 years of follow-up.[3] Given that a wide range of severe long-term complications has been linked to RPVO posttreatment (Table 1), rapid and extensive debulking of thrombus in patients with large clot burden may be an important area of future study and consideration in acute PE, even in patients without significant hemodynamic compromise. Clot burden, or initial obstruction severity, has been shown

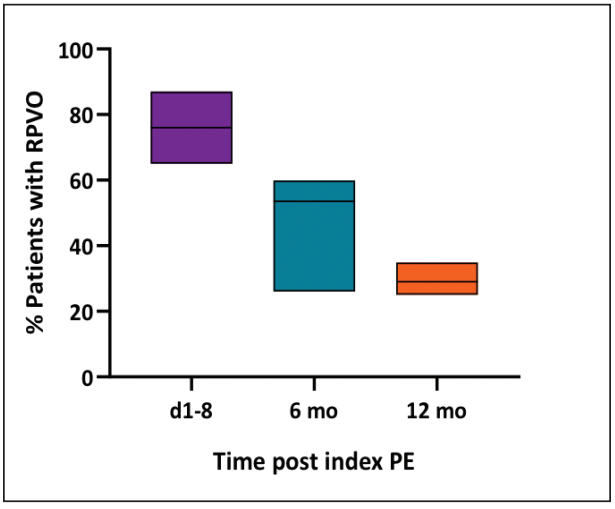

Figure 1. Reported rates of persistent RPVO in the literature at different time points postindex PE. From discharge up to 8 days postindex PE, 65% to 87% of patients are left with relevant RPVO.[17,21] Even 6 months later, high rates of RPVO have been reported in the literature, ranging from 26% to 59.9%.[10,12,19,22] At 1-year postindex PE, RPVO is prevalent in 25% to 35% of patients.[16,18,19]

to be predictive of RPVO and perfusion restoration.[11,14,16] Hence, in addition to biomarkers, RV dysfunction, and DVT presence, the initial obstruction severity could be another valuable factor in PE risk stratification.

PATIENT PRESENTATION

Contrary to the paradigm that treatment escalation is indicated only for patients with clinically tenuous PE, this case demonstrates the potential benefit of effective clot removal in a stable patient with large clot burden. The patient was a 72-year-old man with an extensive saddle PE but stable hemodynamics. He presented with progressive shortness of breath, especially when climbing stairs, but no syncope, collapse, or rest symptoms. His medical history included coronary artery disease, specifically previous myocardial infarction and non–ST-segment elevation myocardial infarction, as well as hypertension, hyperlipidemia, diabetes, and obstructive sleep apnea.

PROCEDURAL OVERVIEW

CTA of the chest confirmed an acute saddle PE with extensive clot burden (Figure 2A), and duplex ultrasound (DUS) showed a concomitant DVT of the left leg. In addition, the patient also had a mildly dilated right ventricle with a right ventricle/left ventricle ratio of 1 (Figure 2B). His troponin was normal and his N-terminal pro b-type natriuretic peptide (NT-proBNP) was mildly elevated (753 pg/mL). Treatment of the PE via aspiration and mechanical thrombectomy using the FlowTriever System (Inari Medical) was planned to reduce his massive clot burden in both pulmonary arteries (PAs; Figure 2C and 2D). After informed consent was obtained, the patient was prepped and draped in the usual sterile manner, and

TABLE 1. SELECTED LITERATURE ON THE INFLUENCE OF RPVO ON OUTCOMES IN PE

Study	Patients (N)	Treatment	RPVO-Associated Clinical Outcomes
Meneveau et al[9] (2013)	416	AC/TT	RPVO was identified as an independent predictor for a combined endpoint of death, recurrent symptomatic PE, development of clinical signs of congestive heart failure associated with elevated brain natriuretic peptide, and change of New York Heart Association functional class to class III or IV
Meneveau et al[10] (2003)	249	TT	RPVO was an independent predictor of long-term mortality
Pesavento et al[12] (2017)	647	AC	RPVO was found to be an independent predictor of recurrent VTE and/or CTEPH
Planquette et al[13] (2016)	310	AC	RPVO was an independent predictor of recurrent VTE
Sanchez et al[16] (2010)	254	AC/TT	RPVO was associated with an increase in PAP and functional limitation; the initial severity of obstruction was an independent risk factor for persistent perfusion defects
Chopard et al[17] (2017)	241	AC/TT	RPVO was identified as an independent predictor for a composite of adverse outcomes (death, heart failure, recurrent VTE, CTEPH, cardiac-caused rehospitalization, worsening dyspnea)
Miniati et al[18] (2006)	320 (subgroup)	–	RPVO was found to be an independent predictor for short-term mortality
Mrozek et al[19] (2018)	85	AC/TT	RPVO was associated with elevated PAP
Wan et al[20] (2018)	239	AC	RPVO, in a graded fashion, was an independent predictor of recurrent VTE

Abbreviations: AC, anticoagulation; CTEPH, chronic thromboembolic pulmonary hypertension; PAP, pulmonary artery pressure; PE, pulmonary embolism; RPVO, residual pulmonary vascular obstruction; TT, thrombolytic treatment; VTE, venous thromboembolism.

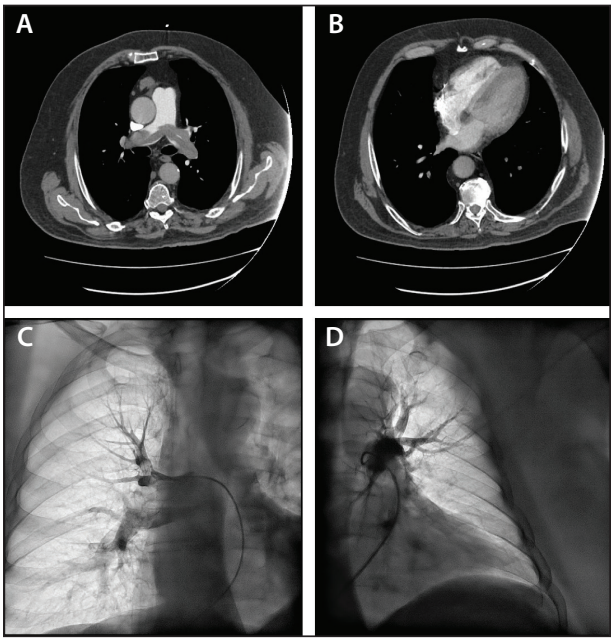

Figure 2. Diagnostic imaging. CTA demonstrating an extensive saddle PE (A) and a modestly enlarged right ventricle (B). Pulmonary angiogram confirming large clot burden in both the right (C) and left (D) PAs.

lidocaine was infused into the right groin for anesthesia. A bolus of heparin was used for anticoagulation with a target activated clotting time of 225 to 250 seconds.

We achieved access in the right femoral vein using a micropuncture technique to insert a Supra Core guidewire (Abbott Vascular) and a Preface sheath (Biosense Webster). Right heart catheterization was performed using a 7-F pulmonary capillary wedge catheter, which was then exchanged for a Grollman catheter (Cook Medical) placed in the left main PA. Selective left pulmonary angiography was performed using 20 mL of contrast and demonstrated near total occlusion of the proximal left main PA, with minimal filling of the left lower lobe. To prepare for thrombectomy of the left PA, the access site was serially dilated to accommodate a 22-F DrySeal sheath (Gore & Associates), which was used to deliver the Triever20 catheter (T20; Inari Medical). The T20 catheter was then easily navigated to the left main PA (Figure 3A). Two aspirations were performed and yielded a moderate amount of organized clot. The FlowTriever catheter was then used to engage the remaining clot. A medium-sized (11–14 mm) FlowTriever device was deployed in the left inferior PA, and two discs were unsheathed (Figure 3B). The next aspiration yielded additional clot. The guidewire was then directed out more laterally, and two more aspirations yielded further thrombus. Pulmonary angiography demonstrated improved flow to the left lung with minimal residual clot.

Right pulmonary angiography confirmed the presence of a significant amount of thrombus, as previously seen on CTA. The right main PA, truncus anterior, and main PA were all affected. We were able to almost completely remove the extensive clot burden via two aspirations (Figure 3C) and achieved significantly improved flow throughout the right lung. The baseline PA pressure was normal at 35/13/22 mm Hg and was unchanged at the end of the procedure. The catheters and sheath were removed, and closure was achieved via a figure-of-eight stitch. Altogether, we were able to remove a large amount of clot from a bilateral/saddle PE to minimize residual clot burden with a total of 80 mL of contrast and 21.9 minutes of fluoroscopy time in a single session, without the need for thrombolytic drugs (Figure 4). The total procedure time was < 60 minutes.

The patient was transferred to a telemetry floor postprocedure and was ambulating the next morning. He was transitioned from intravenous heparin to oral apixaban and was discharged on postprocedure day 2. He was seen in follow-up at 1 week and 1-, 3-, and 6-month time points. His functional status returned to normal by the 1-week appointment and remained good throughout the follow-up period. His repeat echocardiogram demonstrated normal RV size and systolic function at 3 months postprocedure.

SUMMARY

We successfully treated a hemodynamically stable patient (intermediate-low risk) who had an extensive saddle PE via the FlowTriever System in a single session

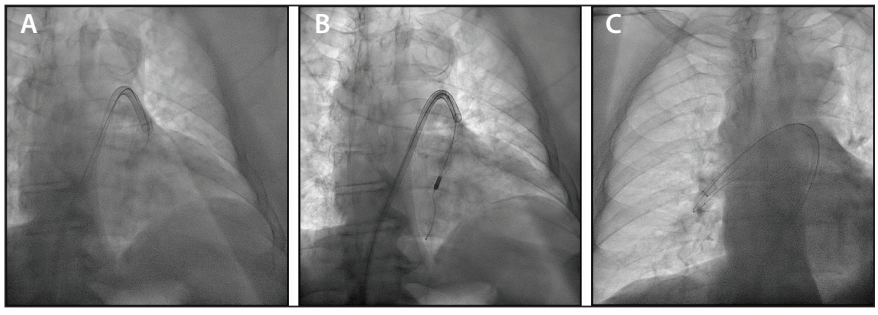

Figure 3. Periprocedure imaging. Pulmonary angiogram of the T20 catheter during aspiration on the left side (A). Two disks of the FlowTriever catheter deployed from the T20 catheter (B). The T20 catheter during aspiration on the right side (C).

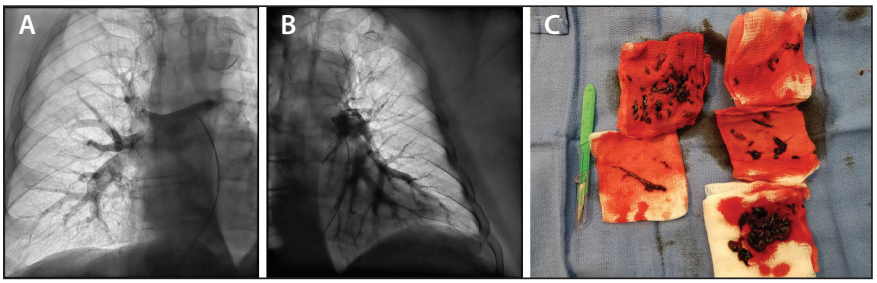

Figure 4. Post-FlowTriever pulmonary angiogram and extracted clot. Pulmonary angiogram showing restored perfusion on the right side (A) and restored flow to the left lung (B). Organized extracted clot (C).

with rapid resolution of clot burden and symptoms. As described previously, there are ample data suggesting that residual clot in PEs can increase the risk of adverse events, including recurrence, heart failure, and death. Using the FlowTriever System in this case allowed effective and rapid resolution of thrombus burden, hopefully improving the patient's chances for a favorable near- and intermediate-term outcome. This effective mechanical debulking tool is a valuable new option to rapidly, safely, and effectively remove large amounts of clot without the risks associated with thrombolytics. It might be an attractive treatment option in a wide range of patients with PE. Future studies will be required to demonstrate the importance of clot debulking as it pertains to clinical outcomes.

1. Konstantinides SV, Torbicki A. Management of pulmonary embolism: recent evidence and the new European guidelines. Eur Respir J. 2014;44:1385-1390.
2. Kuo WT, Sista AK, Faintuch S, et al. Society of Interventional Radiology position statement on catheter-directed therapy for acute pulmonary embolism. J Vasc Interv Radiol. 2018;29:293-297.
3. Tapson VF, Platt DM, Xia F, et al. Monitoring for pulmonary hypertension following pulmonary embolism: the INFORM study. Am J Med. 2016;129:978-985.e2.
4. Schultz J, Giordano N, Zheng H, et al. EXPRESS: a multidisciplinary pulmonary embolism response team (PERT) - experience from a national multicenter consortium [published online January 11, 2019]. Pulm Circ.
5. Secemsky E, Chang Y, Jain CC, et al. Contemporary management and outcomes of patients with massive and submassive pulmonary embolism. Am J Med. 2018;131:1506-1514.e0.
6. Kucher N, Rossi E, De Rosa M, Goldhaber SZ. Massive pulmonary embolism. Circulation. 2006;113:577-582.
7. Elias A, Mallett S, Daoud-Elias M, et al. Prognostic models in acute pulmonary embolism: a systematic review and meta-analysis. BMJ Open. 2016;6:e010324.
8. Barco S, Mahmoudpour SH, Planquette B, et al. Prognostic value of right ventricular dysfunction or elevated cardiac biomarkers in patients with low-risk pulmonary embolism: a systematic review and meta-analysis. Eur Heart J. 2019;40:902-910.
9. Meneveau N, Ider O, Seronde MF, et al. Long-term prognostic value of residual pulmonary vascular obstruction at discharge in patients with intermediate- to high-risk pulmonary embolism. Eur Heart J. 2013;34:693-701.
10. Meneveau N, Ming LP, Séronde MF, et al. In-hospital and long-term outcome after sub-massive and massive pulmonary embolism submitted to thrombolytic therapy. Eur Heart J. 2003;24:1447-1454.
11. Fernandes T, Planquette B, Sanchez O, Morris T. From acute to chronic thromboembolic disease. Ann Am Thorac Soc. 2016;13(suppl 3):S207-S214.
12. Pesavento R, Filippi L, Palla A, et al. Impact of residual pulmonary obstruction on the long-term outcome of patients with pulmonary embolism. Eur Respir J. 2017;49:1601980.
13. Planquette B, Ferré A, Peron J, et al. Residual pulmonary vascular obstruction and recurrence after acute pulmonary embolism. A single center cohort study. Thromb Res. 2016;148:70-75.
14. Planquette B, Sanchez O, Marsh JJ, et al. Fibrinogen and the prediction of residual obstruction manifested after pulmonary embolism treatment. Eur Respir J. 2018;52:1801467.
15. Prandoni P, Lensing AW, Prins MH, et al. Residual venous thrombosis as a predictive factor of recurrent venous thromboembolism. Ann Intern Med. 2002;137:955-960.
16. Sanchez O, Helley D, Couchon S, et al. Perfusion defects after pulmonary embolism: risk factors and clinical significance. J Thromb Haemost. 2010;8:1248-1255.
17. Chopard R, Genet B, Badoz M, et al. Long-term clinical implications of residual pulmonary vascular obstruction after pulmonary embolism: a ventilation-perfusion lung scan follow-up study at two timepoints. J Am Coll Cardiol. 2016;67(suppl 1):2046.
18. Miniati M, Monti S, Bottai M, et al. Survival and restoration of pulmonary perfusion in a long-term follow-up of patients after acute pulmonary embolism. Medicine (Baltimore). 2006;85:253-262.
19. Mrozek J, Petrova J, Vaclavkova J, et al. Reperfusion after pulmonary embolism - long-term follow-up, risk factors, clinical impact. Biomed Pap Med Fac Univ Palacky Olomouc Czech Repub. 2018;162:121-126.
20. Wan T, Rodger M, Zeng W, et al. Residual pulmonary embolism as a predictor for recurrence after a first unprovoked episode: Results from the REVERSE cohort study. Thromb Res. 2018;162:104-109.
21. Wartski M, Collignon MA. Incomplete recovery of lung perfusion after 3 months in patients with acute pulmonary embolism treated with antithrombotic agents. THESEE study group. Tinzaparin ou heparin standard: evaluation dans l'embolie pulmonaire study. J Nucl Med. 2000;41:1043-1048.
22. Hvid-Jacobsen K, Fogh J, Nielsen SL, et al. Scintigraphic control of pulmonary embolism. Eur J Nucl Med. 1988;14:71-72.

FlowTriever and ClotTriever Combination Treatment of Extensive Bilateral Lower Extremity DVT and IVC Filter Thrombosis

Thomas S. Maldonado, MD, FACS
Schwartz-Buckley Professor of Surgery
Director of Venous Thromboembolic Center
New York University Langone Medical Center
New York, New York
thomas.maldonado@nyulangone.org
Disclosures: None.

Caval thrombosis has been reported to have a < 10% incidence rate in patients with contemporary implanted inferior vena cava (IVC) filters.[1] However, IVC filter thrombosis rates in the broader literature vary, often ranging from 2% to 30% and even up to 50% in some studies.[1,2] If an IVC filter becomes thrombosed, symptoms can include pain and edema of both lower extremities, as well as renal failure if the thrombus extends into the suprarenal IVC.[1,3] The risk of a subsequent PE is another serious consequence, as thrombus may eventually extend above the filter, permitting embolization.[2,4] Additionally, the presence of a filter does not appear to confer an advantage in preventing PE if IVC thrombosis occurs.[5] Compared with lower extremity DVT, the mortality risk in those with IVC involvement is doubled. If left untreated, significant morbidities of IVC thrombosis include postthrombotic syndrome in up to 90%, severe claudication in 45%, PE in 30%, and venous ulceration in 15%.[6] In this case report, we present a case of extensive bilateral DVT extending into the IVC.

PATIENT PRESENTATION

A 67-year-old woman with a body mass index of 32 kg/m^2 was recently hospitalized for lower back pain and bilateral leg swelling. Her medical history included cancer and a clotting disorder (factor V Leiden), and she had an OptEase IVC filter (Cordis, a Cardinal Health company) in place. Although she was initially discharged on apixaban, anticoagulation was discontinued due to severe epistaxis. She presented to our institution with worsening bilateral leg swelling and a tense sensation in both legs that she described as "bursting." Her severe leg pain rendered her unable to walk, making her wheelchair dependent. The initial diagnosis revealed a slightly increased creatinine level (from 0.9 to 1.7 mg/dL), and a review of a recent CTA demonstrated acute thrombosis of the IVC, which extended to the level of the IVC filter. In addition, her left common and external iliac veins were also affected, and edema was visualized in the left thigh.

PROCEDURAL OVERVIEW

Venous DUS confirmed bilateral DVT affecting the right common iliac and external iliac veins and the left common iliac, external iliac, common femoral, great saphenous, and femoral veins. DUS also confirmed caval thrombosis extending to the level of the IVC filter. Due to the recent, acute, severe thrombosis of the IVC filter and the extensive bilateral lower extremity DVT resulting in debilitating swelling, we decided to plan for mechanical and aspiration thrombectomy using both the ClotTriever (Inari Medical) and FlowTriever Systems. First, we achieved internal jugular access to place a temporary suprarenal filter (Figure 1). We then achieved access at the right femoral vein to prepare for the mechanical thrombectomy procedure with the ClotTriever System.

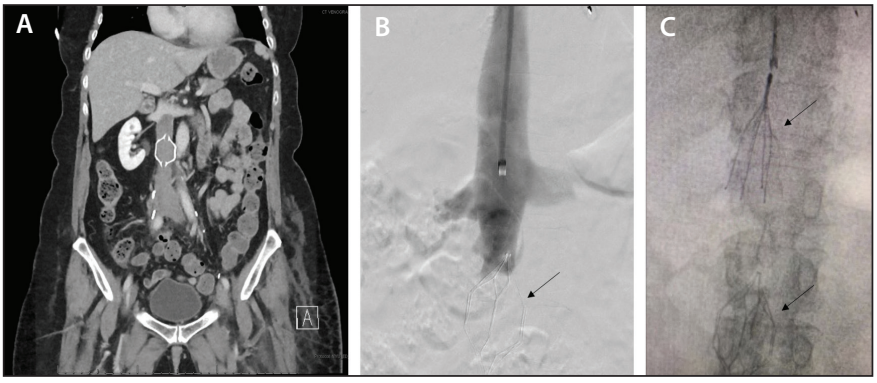

Figure 1. Initial CTA (A). Venogram obtained from the initial internal jugular access for deployment of the second filter (suprarenal); the initially present OptEase filter is marked via an arrow (B). Postdeployment of the second filter with black arrows marking both the OptEase initial filter and the newly deployed suprarenal filter (C).

The ClotTriever System comes with a 13-F specialty sheath that allows insertion of the ClotTriever catheter over a 0.035-inch guidewire. After it is advanced and positioned beyond the targeted thrombus, the ClotTriever catheter can be unsheathed. This deploys the coring element and allows for wall apposition. Attached to the laser-cut coring element is a woven nitinol collection bag. To separate the clot from the vessel wall and collect it in the integrated collection bag, the catheter is slowly pulled back toward the sheath. Once the initial pullback is completed, the ClotTriever catheter can be collapsed and removed from its sheath. Outside the body, the collected clot is cleaned off to prepare the device for reinsertion. The procedure can be repeated at the discretion of the operator until maximum clot capture is achieved.

Due to the presence of the filter, positioning the ClotTriever catheter past the clot in the IVC was not an option. Therefore, we placed a stiff Meier guidewire (Boston Scientific Corporation) up and over the confluence (Figure 2). Positioning the tip of this wire distally in the left popliteal vein assured that the ClotTriever catheter could track without relapsing into the IVC. We successfully positioned the ClotTriever catheter coring element past the clot in the left femoral vein before deploying and engaging the system. We performed four slow pullbacks with a magnified view on the confluence, closely monitoring the coring element and collection bag passing over this section. A subsequent venogram confirmed optimal, bilateral restoration of flow (Figure 3). To subsequently target the caval thrombosis, we upsized to a 20-F DrySeal sheath to prepare for FlowTriever thrombectomy via the Triever20 (T20) catheter. The T20, which tracks over a 0.035-inch Amplatz Super Stiff guidewire (Boston Scientific Corporation), has an aspiration-based mechanism of action. Once the T20 is positioned adjacent to the clot, a vacuum is manually created in the system using a 60-mL custom large-bore syringe that is attached to a side port tubing connector. Next, this vacuum is released, which will extract the clot via powerful suction through the catheter into the syringe. Blood loss per pass is limited to a 60-mL maximum in the rare event no clot is retrieved.

In the presented case, we navigated the Meier guidewire through the OptEase and suprarenal filters and then advanced the T20 into the IVC below the OptEase filter. We performed

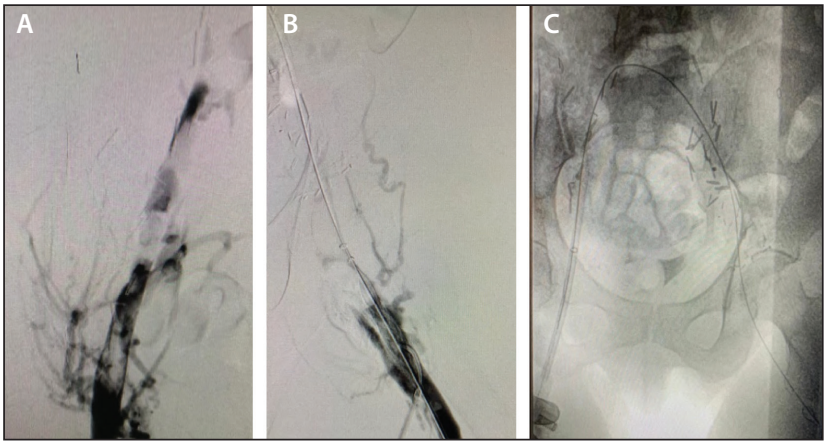

Figure 2. Initial right-side venogram (A). Initial left-side venogram (B). Postdelivery of the guidewire via the up-and-over technique over the confluence and deployment of the ClotTriever sheath via right femoral vein access (C).

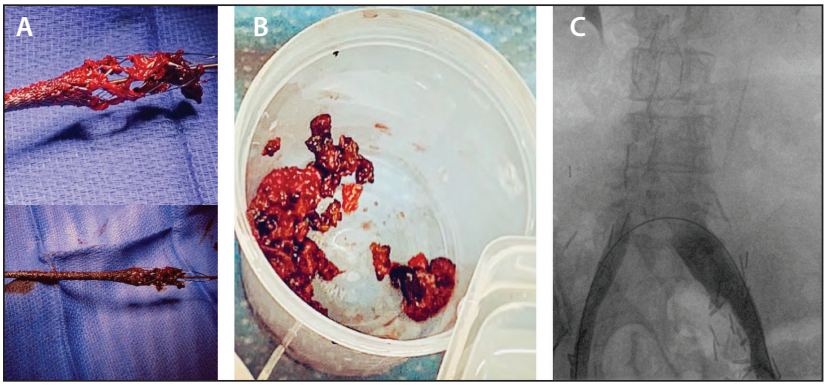

Figure 3. The ClotTriever coring element and collection bag containing a large amount of clot removed from the patient (A). Organized thrombus removed via three passes of the ClotTriever catheter (B). Postthrombectomy venogram showing bilaterally restored flow (C).

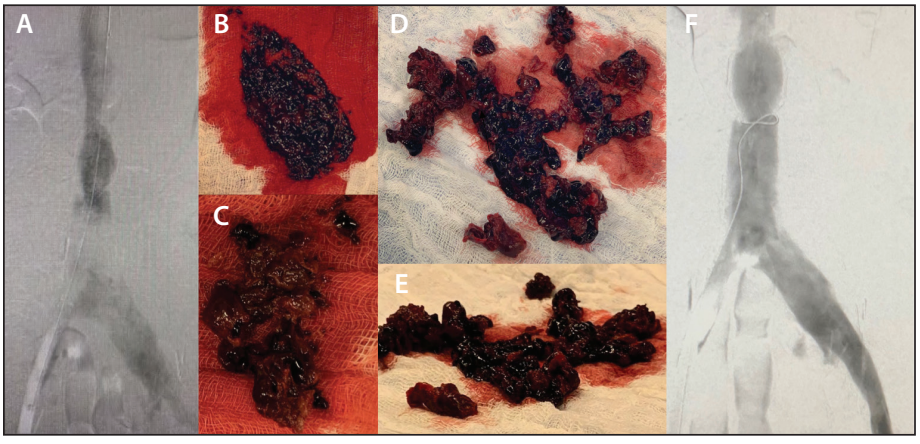

Figure 4. Venogram before aspiration (A). Thrombus removed after a first aspiration with the T20 catheter (B, C). Aspirated thrombus after two more aspirations (D). Aspirated thrombus after a fourth and final aspiration (E). Final result showing restored flow in the IVC (F).

four successful aspirations, removing significant amounts of fresh and organized clot (Figure 4). The suprarenal IVC filter was then retrieved, leaving the original OptEase filter in place. Follow-up imaging suggested the caval thrombosis was efficiently cleared and flow was restored. The entire procedure of clearing the thrombosed IVC and the bilateral DVT was performed in one session without the use of thrombolytics, thus reducing the bleeding risk[7] and consequent intensive care unit stay associated with thrombolytics. The patient was discharged on postoperative day 1 on appropriate anticoagulation.

SUMMARY

We safely and successfully treated a patient with extensive bilateral DVT and IVC filter thrombosis using mechanical and aspiration thrombectomy via the ClotTriever and FlowTriever Systems. When untreated, caval thrombosis—especially in the setting of an indwelling IVC filter—can result in severe morbidity for patients. Treatment options that enable safe and effective clot clearance are limited, and they often require exposing patients to the bleeding risks associated with thrombolytic drugs. DVT with a thrombosed filter can be associated with severe symptoms, as was the case with our patient who suffered from debilitating pain, showed signs of edema, and was incapacitated regarding her ability to walk. Having two highly effective devices specifically designed to target venous clot at our disposal allowed the fast, efficient treatment of the bilateral DVT and caval thrombosis below the filter. Importantly, this was accomplished in one session without the need

for thrombolysis and its associated risks. Furthermore, no capital equipment was required, nor was a perfusion team necessary because blood loss was minimal. As demonstrated in our case, the combined use of ClotTriever and FlowTriever offers a safe and efficient solution for removing large quantities of clot in complex settings of iliofemoral and caval thrombosis, including a thrombosed IVC filter.

1. Grewal S, Chamarthy MR, Kalva SP. Complications of inferior vena cava filters. Cardiovasc Diagn Ther. 2016;6:632-641.
2. Van Ha TG. Complications of inferior vena caval filters. Semin Intervent Radiol. 2006;23:150-155.
3. Sildiroglu O, Ozer H, Turba UC. Management of the thrombosed filter-bearing inferior vena cava. Semin Intervent Radiol. 2012;29(1):57-63.
4. Girard P, Meyer G, Parent F, Mismetti P. Medical literature, vena cava filters and evidence of efficacy. A descriptive review. Thromb Haemost. 2014;111:761-769.
5. Teter K, Schrem E, Ranganath N, et al. Presentation and management of inferior vena cava thrombosis. Ann Vasc Surg. 2019;56:17-23.
6. Alkhouli M, Morad M, Narins CR, et al. Inferior vena cava thrombosis. 2016;9:629-643.
7. Fleck D, Albadawi H, Shamoun F, et al. Catheter-directed thrombolysis of deep vein thrombosis: literature review and practice considerations. Cardiovasc Diagn Ther. 2017;7(suppl 3):S228-S237.

A Single-Session Combination Treatment of PE and DVT in a Patient With Postoperative VTE

Suman Annambhotla, MD
Vascular Surgeon
Longstreet Clinic
Northeast Georgia Medical Center
Gainesville, Georgia
sutheman@gmail.com
Disclosures: Consulting for Medtronic and Inari Medical.

Approximately 90% of symptomatic PEs originate from venous thrombus of the lower extremities.[1] Furthermore, mortality rates associated with recurrent PE are significant,[2,3] likely due to the patient's compromised cardiac reserves from the original PE. Consequently, if DVT is present, care should be taken to address both the PAs and the remaining DVT, particularly if thrombus affects areas that are more prone to embolization, such as the iliocaval region. A meta-analysis published in 2016 found that concomitant DVT conferred an almost doubled risk for early death postindex PE.[4] The prevalence of PE-associated DVT is high, but numbers in the literature have historically varied. An early meta-analysis found DVT rates ranging from 10% to 93% in different studies of proven PE, with a pooled estimate of 36%.[5] A later meta-analysis showed a higher prevalence with an overall occurrence of 56%,[4] and an even more recent study reported a rate of 70.6% in 428 consecutive patients with PE who were specifically screened for concomitant DVT.[6] An increased awareness among clinicians in general could be one possible explanation for these higher rates in recent years. However, improved diagnostic algorithms and optimized triage efforts in certain institutions could also contribute to the higher numbers, and consequently, a previous lack of screening might have contributed to earlier underestimation.

This presented case represents the frequent finding of PE-associated DVT—more specifically, a patient with PE and additional lower extremity symptoms from a thrombosed IVC filter. Although a symptomatic patient with a thrombosed IVC filter can be enough to warrant intervention, possible complications can be of additional concern, such as renal failure, thrombus affecting the suprarenal IVC, or subsequent PE if thrombus would eventually extend above the filter.[7-10] Given the symptoms of the patient in this case, as well as the risk of a potential future breakthrough PE and a higher chance of death associated with concomitant DVT and IVC thrombosis, we planned to actively intervene in both venous thromboembolism presentations. To do so, we opted for the FlowTriever, a designated thrombectomy catheter that has been uniquely designed to extract large amounts of venous thrombus.

PATIENT PRESENTATION

A 63-year-old man who was recovering in the intensive care unit from a coronary artery bypass for a myocardial infarction developed PE during his postoperative course. His symptoms included significant hypoxemia that required high-flow nasal oxygen. He also described dyspnea on exertion and chest pain. We also found him to have right heart strain, and we confirmed a large pulmonary thrombus burden on CTA (Figure 1A). The patient had undergone recent IVC filter placement and now showed moderate leg edema, suggesting a potential additional lower extremity DVT, possibly to the level of the IVC filter.

PROCEDURAL OVERVIEW

To further investigate, the patient was brought to the catheterization laboratory for pulmonary angiography and possible subsequent thrombectomy. He was placed in the supine position, sedated, and prepared in the usual sterile fashion. Right femoral vein access was achieved using standard techniques, and a combination Glidewire Advantage (Terumo Interventional Systems) and Glidecath catheter (Terumo Interventional Systems) was used to gain positioning in the main PA, followed by

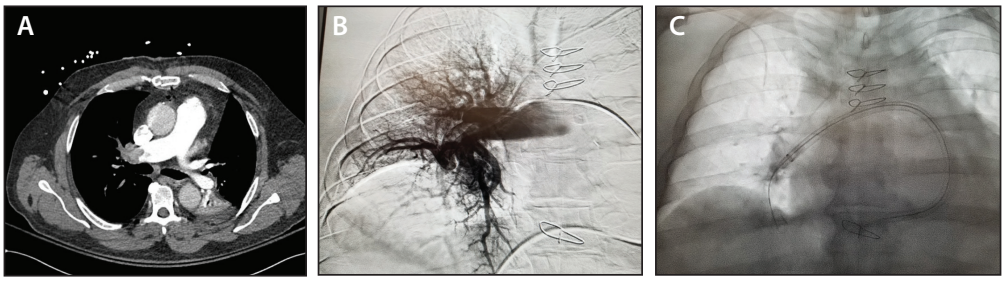

Figure 1. Diagnostic imaging. Initial CT confirming PE (A). Pulmonary angiogram confirming the significant thrombus burden in the right main PA (B). The positioned FlowTriever T20 catheter preaspiration (C).

placement of an angled pigtail catheter for imaging. The pulmonary angiogram confirmed a significant thrombus burden in the right main PA toward the segmental and lobar branches (Figure 1B). Given that we suspected concomitant DVT due to leg edema, we also performed venography, revealing an extensive thrombosis of the IVC filter. Upon those two findings, we decided to move forward with mechanical thrombectomy and start systemic heparinization.

To prepare for FlowTriever thrombectomy, a 22-F DrySeal sheath was placed, and a 0.035-inch Amplatz Super Stiff guidewire was inserted through the main PA trunk that we previously traversed. Next, the Triever20 (T20) catheter of the FlowTriever System was advanced to the PA thrombus burden (Figure 1C). The T20 catheter can essentially function as both an advanced aspiration catheter and a guide for the FlowTriever catheter, which is the system's second component and is reserved for more tenacious thrombus. The T20 catheter's aspiration mechanism relies on the generation of a vacuum within the system. Once the mouth of the T20 catheter is positioned adjacent to thrombus, a vacuum is manually created by pulling back a 60-mL custom large-bore syringe that is attached to a side port tubing connector at the sheath. Once established, the vacuum can be released by opening the respective port. This will extract thrombus via powerful suction through the T20 catheter and into the syringe. In the case of more tenacious thrombus, the FlowTriever catheter can be deployed. When it is inserted and advanced through the T20 catheter into the thrombus, its self-expanding braided nitinol disks actively engage and liberate thrombus for aspiration. The FlowTriever thrombectomy procedure can be repeated several times, at the discretion of the operator and depending on the various locations of thrombus and improvement in distal flow on repeat angiography.

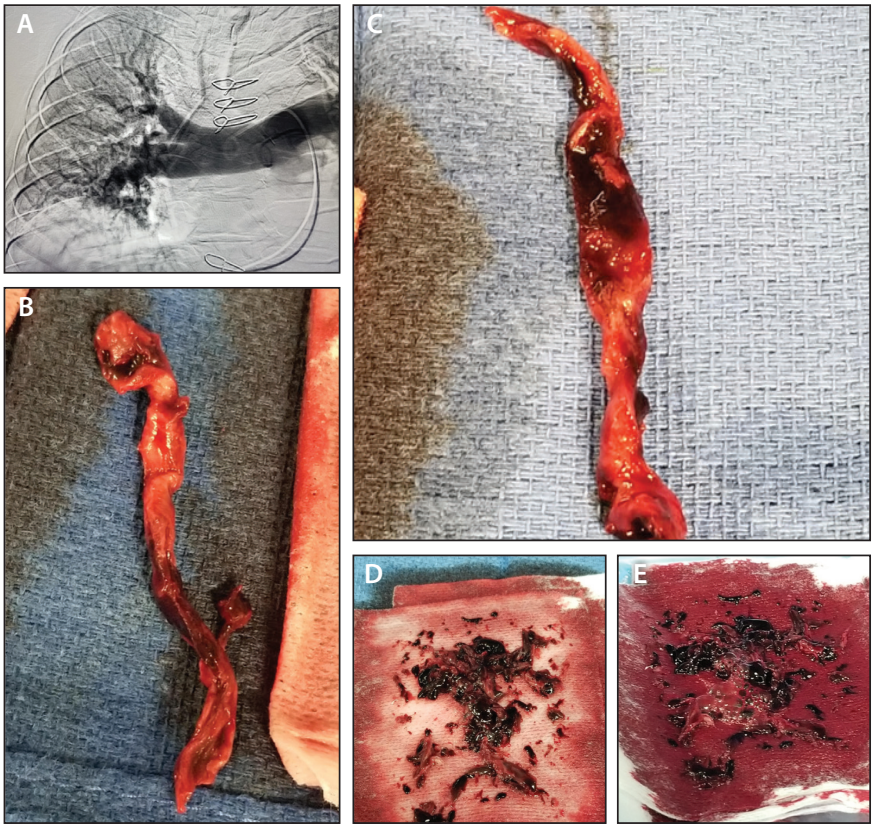

Figure 2. Procedural outcome of FlowTriever PE thrombectomy. Post-FlowTriever thrombectomy pulmonary angiogram confirming improved perfusion (A). Extracted thrombus (B–E), with B and C showing highly organized thrombus.

Once we positioned the T20 catheter in the right main PA, we performed four aspiration passes and removed a large thrombus burden. The patient's

vital signs and oxygenation improved immediately. We performed one last pass to ensure optimal clearance of the thrombus, and a final right PA angiogram confirmed significant improvement and restoration of perfusion (Figure 2).

To address the patient's concomitant IVC thrombosis, we pulled the T20 carefully past the filter back toward the sheath. When it was positioned proximal to the IVC thrombus, we once more engaged the system and performed four aspirations. This allowed for complete clearance of the thrombosed filter. The postthrombectomy venogram confirmed the excellent result and showed optimal blood flow toward the heart (Figure 3). In the last steps, we removed the T20 and the 22-F DrySeal sheath, placed a 3-0 nylon stitch at the groin site, and held manual pressure until hemostasis was achieved.

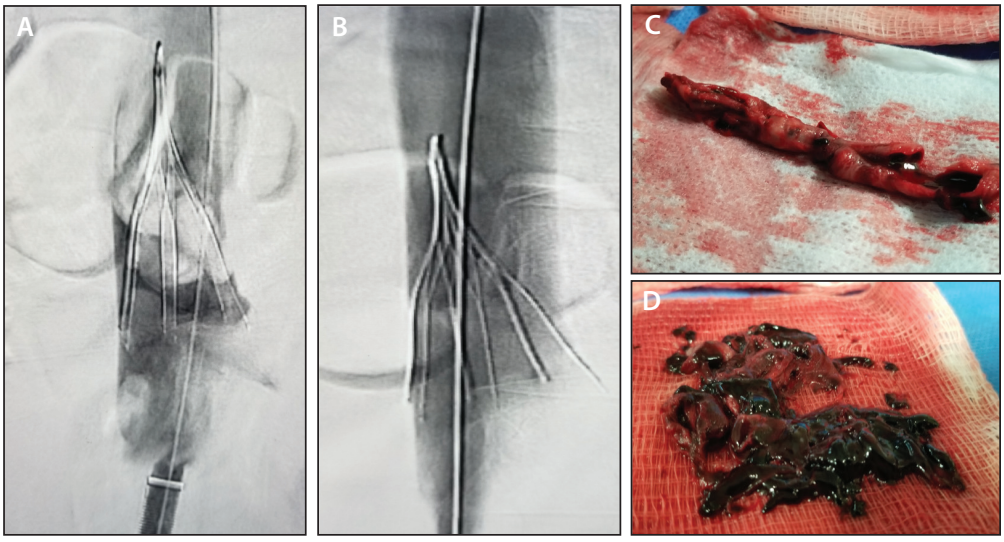

Figure 3. Procedural outcome of the IVC thrombectomy. Pre-FlowTriever thrombectomy IVC venogram (A). Postprocedural venogram confirming thrombus removal and improved flow to the heart (B). Extracted thrombus (C, D), with C showing highly organized thrombus.

Of note, the estimated total blood loss in this single-session PE thrombectomy with subsequent DVT intervention was limited to 100 mL. The patient tolerated the procedure well and was transferred to recover in his room in stable condition. He described an immediate improvement in his breathing and was de-escalated from oxygen.

SUMMARY

We successfully treated an extensive PE and concomitant IVC thrombosis with the FlowTriever System in a single session with no need for thrombolytic therapy, minimal blood loss, and no complications. The patient tolerated the procedure well and reported immediate symptom improvement. As mentioned, concomitant DVT presents a common and clinically relevant finding in PE, conferring a higher mortality risk. In our case, it also specifically affected the IVC in a patient with a filter in place. The ability to address this complex DVT presentation within the same procedure as the index PE in an effective and safe manner provides a valuable option to effectively treat patients, quickly improve their symptoms, and hopefully optimize their chances for a long-term positive outcome. ■

1. Lee JS, Moon T, Kim TH, et al. Deep vein thrombosis in patients with pulmonary embolism: prevalance, clinical significance and outcome. Vasc Specialist Int. 2016;32:166-174.
2. D'Agostino C, Zonzin P, Enea I, et al. ANMCO position paper: long-term follow-up of patients with pulmonary thromboembolism. Eur Heart J Suppl. 2017;19(suppl D):D309-D332.
3. Goldhaber SZ, Visani L, De Rosa M. Acute pulmonary embolism: clinical outcomes in the International Cooperative Pulmonary Embolism registry (ICOPER). Lancet. 1999;353:1386-1389.
4. Becattini C, Cohen AT, Agnelli G, et al. Risk stratification of patients with acute symptomatic pulmonary embolism based on presence or absence of lower extremity DVT: systematic review and meta-analysis. Chest. 2016;149:192-200.
5. van Rossum AB, van Houwelingen HC, Kieft GJ, Pattynama PM. Prevalence of deep vein thrombosis in suspected and proven pulmonary embolism: a meta-analysis. Br J Radiol. 1998;71(852):1260-1265.
6. Hirmerova J, Seidlerova J, Chudacek Z. The prevalence of concomitant deep vein thrombosis, symptomatic or asymptomatic, proximal or distal, in patients with symptomatic pulmonary embolism. Clin Appl Thromb Hemost. 2018;24:1352-1357.
7. Sildiroglu O, Ozer H, Turba UC. Management of the thrombosed filter-bearing inferior vena cava. Semin Intervent Radiol. 2012;29:57-63.
8. Grewal S, Chamarthy MR, Kalva SP. Complications of inferior vena cava filters. Cardiovasc Diagn Ther. 2016;6:632-641.
9. Girard P, Meyer G, Parent F, Mismetti P. Medical literature, vena cava filters and evidence of efficacy. A descriptive review. Thromb Haemost. 2014;111:761-769.
10. Van Ha TG. Complications of inferior vena caval filters. Semin Intervent Radiol. 2006;23:150-155.