# Endovascular port-a-cath rescue in acute thrombotic superior vena cava syndrome

Hanno Amberger, MD,<sup>a</sup> Iris Baumgartner, MD,<sup>a</sup> Nils Kucher, MD,<sup>b</sup> and Marc Schindewolf, MD,<sup>a</sup> Bern, Zurich, Switzerland

### **ABSTRACT**

Acute superior vena cava (SVC) syndrome is managed by endovascular recanalization, venoplasty with stenting, and anticoagulation. It is often associated with central venous catheters. We present a case of a 55-year-old woman with acute SVC syndrome due to port-a-cath-associated thrombosis of the SVC and the brachiocephalic and subclavian veins who was treated with catheter-based thrombectomy and local spray thrombolysis, venoplasty, and stent placement. Port-a-cath restoration was achieved in the same session by endovascular snaring and repositioning. This case demonstrates that reoperation with surgical catheter removal and reinsertion of central venous catheters with possible complications (eg. rethrombosis, bleeding) can be avoided by single-session endovascular management. (J Vasc Surg Cases and Innovative Techniques 2019;5:169-73.)

**Keywords**: Catheter-directed thrombolysis; Port-a-cath snaring; Port-a-cath-associated thrombosis; Superior vena cava syndrome; Upper extremity venous thrombosis

Superior vena cava (SVC) syndrome is often associated with central venous catheters, hemodialysis catheters, or pacemaker electrodes. Endovascular recanalization procedures are a mainstay therapy to enable quick relief from symptoms in these vascular emergencies. Venoplasty with stenting is often necessary to ensure a good long-term result. In situ catheters reaching into the SVC might be jailed by stent placement between the stent and the vessel wall in the process and hence could be compromised in their function.

We report a case in which we chose to perform a temporary snaring procedure of a port-a-cath tube in the setting of SVC syndrome to avoid catheter removal and later reinsertion with the associated risks and to minimize health care-related cost. The patient consented to publication of this report.

#### **CASE REPORT**

A 55-year-old woman (155 cm, 55 kg) who had undergone hemicolectomy because of an obstructing adenocarcinoma of the ascending colon 6 months ago presented in the emergency department because of a progressive swelling of her face and neck and arms, cyanosis and edema of both hands, and

From the Division of Angiology, Swiss Cardiovascular Center, Inselspital, Bern University Hospital, Bern, and Clinic for Angiology, University Hospital Zurich, Zurich.

Author conflict of interest: none.

Correspondence: Hanno Amberger, MD, Swiss Division of Cardiology, Cardiovascular Center, Freiburgstrasse 18, 3008 Bern, Switzerland (e-mail: hanno. amberger@insel.ch).

The editors and reviewers of this article have no relevant financial relationships to disclose per the Journal policy that requires reviewers to decline review of any manuscript for which they may have a conflict of interest.

2468-4287

© 2019 The Author(s). Published by Elsevier Inc. on behalf of Society for Vascular Surgery. This is an open access article under the CC BY-NC-ND license (http://creativecommons.org/licenses/by-nc-nd/4.0/).

https://doi.org/10.1016/j.jvscit.2019.03.005

shortness of breath at rest for the last 12 days. Until 20 days ago, she had been receiving subsequent adjuvant chemotherapy with FOLFOX, which was administered through a porta-cath. In addition to the presenting symptoms, the physical examination showed dilated jugular veins in the upright position. There were no local signs of infection in the area of the implanted port-a-cath and no edema of the lower extremities. Her medical history further showed a substituted hypothyroidism after thyroidectomy for struma nodosa. Initial diagnostic workup contained a computed tomography scan, which showed thrombosis of the SVC reaching from the caval confluence of the azygos vein to both brachiocephalic veins into the left subclavian vein (Fig 1). Vascular compression by a tumor or lymphadenopathy and pulmonary embolism were ruled out. Echocardiography showed normal systolic and diastolic function of the heart, no valvulopathies, and normal-sized left and right ventricles. No intracardiac clots were detected. Laboratory analysis showed normal red and white blood cell counts. The international normalized ratio was 0.91. All electrolytes, C-reactive protein, thyroid-stimulating hormone, and kidney function parameters were within normal range.

Therapeutic procedure and assessment. Because of the newly diagnosed thrombosis, the patient was treated with a therapeutic dose of unfractionated heparin (Liquemin; Drossapharm, Basel, Switzerland) and transferred to our hospital for endovascular revascularization.

We performed venography over the right basilic vein and confirmed the diagnosis of an occlusion of the SVC. We opted for a catheter-based thrombectomy (AngioJet 8F ZelanteDVT; Boston Scientific, Marlborough, Mass) with PowerPulse spray delivery of 10 mg of alteplase (Actilyse; Boehringer Ingelheim, Basel, Switzerland) into the thrombus. Removal of fresh thrombotic material revealed extended post-thrombotic changes in the proximal brachiocephalic vein and the SVC. To completely unmask the extent of post-thrombotic changes, we placed a valved-tip, multiple-side hole lysis catheter



**Fig 1.** Computed tomography scan **(A)** and phlebography **(B)** of thrombosis of the superior vena cava (SVC) and both brachiocephalic veins (*arrow*).

(Cragg-McNamara; ev3 Neurovascular, Irvine, Calif) for local thrombolysis during 15 hours (2 mg/h for 5 hours, 1 mg/h for 10 hours). Second-look phlebography the next day further showed a high-grade (75%), chronic, post-thrombotic stenosis of the SVC, which was associated with the tip position of the port-a-cath that had been placed through the left subclavian vein (Fig 2). To ensure unrestrained venous inflow and to prevent secondary SVC syndrome, we decided to perform percutaneous transluminal angioplasty and stenting of the lesion. However, to prevent jailing of the port-a-cath tube by stent placement to keep it functional in case of future chemotherapy and to avoid surgical removal and the need for reimplantation with subsequent risk of thrombosis progression during perioperative pausing of anticoagulation, endovascular repositioning of the port-a-cath tube before percutaneous transluminal angioplasty and stenting was considered in a first approach. Over an 8F sheath access in the right basilic vein, the stenosis was passed using a 0.035-inch guidewire (Radiofocus Guidewire M, stiff type; Terumo, Tokyo, Japan) and a Navicross support catheter (Terumo). Afterward, a snare (Atrieve Vascular Snare Kit 18-30 mm; Argon Medical Devices, Plano, Tex) was inserted over a delivery catheter, and the tip of the port-a-cath was captured in the SVC and pulled back into the right brachiocephalic vein (Fig 3). Then, balloon angioplasty (Atlas Gold 12/40 mm; Bard Peripheral Vascular, Tempe, Ariz), stent implantation (sinus-XL, 18/60 mm; OptiMed, Ettlingen, Germany), and postdilation (Atlas Gold 14/40 mm) were performed over a 10F sheath (Avanti sheath introducer; Cordis, Miami Lakes, Fla) access in the right common femoral vein. After successful stent implantation, the catheter was snared from the right common femoral vein and pulled back into the stent (Fig 4). Venography at the end of the examination showed an unrestricted venous inflow (Fig 5).

An oral anticoagulation regimen with rivaroxaban 15 mg twice daily was initiated for 3 weeks, followed by 20 mg every day.

The follow-up visit after 3 months showed no clinical signs of a stent occlusion. Duplex ultrasound confirmed normal venous



**Fig 2.** High-grade chronic, post-thrombotic stenosis of the superior vena cava (SVC) at the tip position of the porta-cath after AngioJet thrombectomy (*arrow*).

inflow. The port-a-cath was still in situ with regular function. A decision was made to continue the anticoagulation regimen with rivaroxaban until port-a-cath explantation.

# **DISCUSSION**

An SVC syndrome caused by a lumen-obstructing thrombosis is often associated with central venous

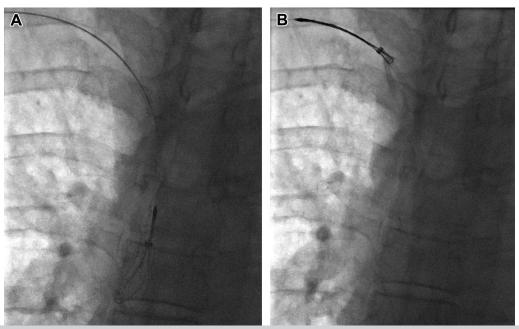
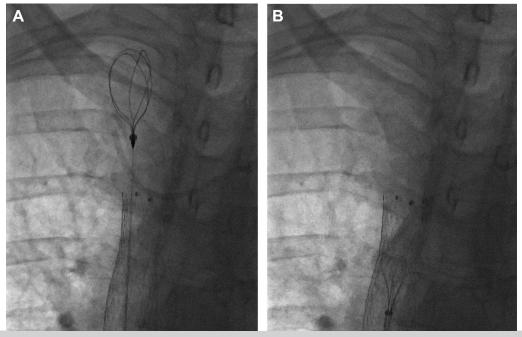


Fig 3. A and B, Snaring of the port-a-cath tube from the superior vena cava (SVC) into the right brachiocephalic vein using an Atrieve Vascular Snare Kit 18-30 mm (Argon Medical Devices, Plano, Tex).

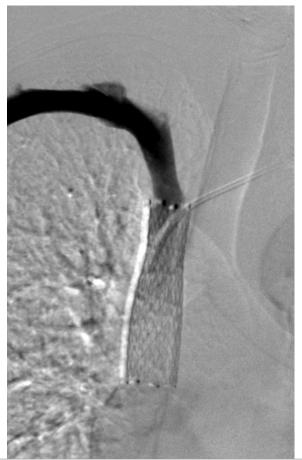


**Fig 4. A** and **B**, Snaring of the catheter from the right common femoral vein back into the stent using an Atrieve Vascular Snare Kit 18-30 mm.

catheters or pacemaker electrodes. The growing use of these central venous devices is reflected in increasing cases of thrombosis of the vena cava,<sup>3-5</sup> which occurs in 0.2% to 3.3% of these patients. Prothrombotic conditions (eg, cancer, chemotherapy, thrombophilia, and external compression) increase the risk of thrombosis.<sup>6</sup> In patients

with an underlying malignant disease, the incidence of thrombosis associated with the use of central venous catheters is estimated to be up to 30%. A feared complication is progression of thrombosis into the SVC. The symptoms caused by a vena cava thrombosis depend on the time of thrombus progression to flow-

Amberger et al



**Fig 5.** Venous inflow after restoration of the superior vena cava (SVC) with port-a-cath in place.

limiting obstruction and the capacity of collateral vein drainage through the azygos vein, internal mammary veins, lateral thoracic vein, and paraspinal and esophageal veins.<sup>2,12</sup>

If there is an acute occlusion of the SVC, the most common symptoms are swelling of the face and neck and arms, cyanosis, venous stasis on the shoulders and neck, orthopnea, and dyspnea. Headaches, dizziness, syncope, confusion, and somnolence (caused by increasing intracranial pressure) as well as pulmonary embolism and hemodynamic instability can quickly lead to life-threatening situations that are associated with unfavorable outcomes.<sup>2</sup>

In the past, SVC syndrome not associated with cancer has been treated conservatively with anticoagulants, diuretics, and corticosteroids, keeping the patient in an upright position with head elevation. A surgical treatment option is a median sternotomy with venous bypass graft. In past years, endovascular recanalization procedures have evolved as a mainstay of invasive therapy to enable quick relief from symptoms in these vascular emergencies. Catheter-based therapy and thrombolysis must be considered in acute onset of SVC syndrome

(symptoms <14 days, progressive symptoms, lifethreatening situations).<sup>2,8</sup> Computed tomography angiography is recommended to exclude the most common causes (tumors, lymphoma, struma) of SVC syndrome, to exclude pulmonary embolism, and to evaluate proximal thrombosis extension. For anatomic reasons, venoplasty with stenting is necessary when there is a relevant chronic stenosis (usually due to fibrosis or tumor mass) left after the thrombus burden is dissolved because of better patency rates after stenting in those cases than with angioplasty alone. The technical success rate of stenting of the SVC or its branches is 95% to 100%, with a 2-year primary patency rate up to 76% and secondary patency rates up to 95%.<sup>14</sup> Acute complications of stent placement for SVC syndrome have been reported in 3% to 7% of patients, including infection, pulmonary embolism, stent migration, hematoma at the insertion site, supraventricular arrhythmia, bleeding, and perforation (rarely).

Because pulmonary embolism as a complication of SVC stenting is rare and placement of an SVC filter bears the risk of severe complications, SVC filter placement to prevent pulmonary embolism should be performed only in special situations.<sup>1</sup> In situ catheters (eg. portacath, hemodialysis catheters) pose the problem that they would be jailed by stent placement between the stent and the vessel wall and hence would be compromised in their function.<sup>15</sup>

## **CONCLUSIONS**

To avoid catheter removal and reinsertion with the associated risks (bleeding, pneumothorax, rethrombosis, thrombosis progression, pulmonary embolism, infection) and to minimize health care-related cost, single-session temporary snaring of the catheter with endovascular replacement is a feasible treatment option after thrombectomy and therapy for the SVC syndrome.<sup>7</sup>

#### **REFERENCES**

- Engelberger RP, Kucher N. Management of deep vein thrombosis of the upper extremity. Circulation 2012;126: 768-73.
- 2. Kucher N. Clinical practice. Deep vein thrombosis of the upper extremities. N Engl J Med 2011;36:861-9.
- 3. Joffe HV, Kucher N, Tapson VF, Goldhaber SZ. Upper-extremity deep vein thrombosis: a prospective registry of 592 patients. Circulation 2004;110:1605-11.
- Rice TW, Rodriguez RM, Light RW. The superior vena cava syndrome: clinical characteristics and evolving etiology. Medicine (Baltimore) 2006;85:37-42.
- Wilson LD, Detterbeck FC, Yahalom J. Clinical practice. Superior vena cava syndrome with malignant causes. N Engl J Med 2007;356:1862-9.
- Stuck AK, Engelberger RP, Saengprakai W, Kucher N. Pharmacomechanical or ultrasound-assisted thrombolysis, balloon angioplasty and provisional surgical decompression for upper extremity deep vein thrombosis due to thoracic outlet syndrome. Thromb Res 2016;145:109-11.

- Hasskarl J, Koberich S, Frydrychowicz A, Illerhaus G, Waller CF. Complete caval thrombosis secondary to an implanted venous port—a case study. Dtsch Arztebl Int 2008;105:18-21.
- 8. Lordick F, Hentrich M, Decker T, Hennig M, Pohlmann H, Hartenstein R, et al. Ultrasound screening for internal jugular vein thrombosis aids the detection of central venous catheter-related infections in patients with haemato-oncological diseases: a prospective observational study. Br J Haematol 2003;120:1073-8.
- Madan AK, Allmon JC, Harding M, Cheng SS, Slakey DP. Dialysis access-induced superior vena cava syndrome. Am Surg 2002;68:904-6.
- Otten TR, Stein PD, Patel KC, Mustafa S, Silbergleit A. Thromboembolic disease involving the superior vena cava and brachiocephalic veins. Chest 2003;123:809-12.
- Stockx L, Raat H, Donck J, Wilms G, Marchal G. Repositioning and leaving in situ the central venous catheter during percutaneous treatment of associated superior vena cava

- syndrome: a report of eight cases. Cardiovasc Intervent Radiol 1999;22:224-6.
- 12. Wudel LJ Jr, Nesbitt JC. Superior vena cava syndrome. Curr Treat Options Oncol 2001;2:77-91.
- Gray BH, Olin JW, Graor RA, Young JR, Bartholomew JR, Ruschhaupt WF. Safety and efficacy of thrombolytic therapy for superior vena cava syndrome. Chest 1991;99:54-9.
- Urruticoechea A, Mesia R, Dominguez J, Falo C, Escalante E, Montes A, et al. Treatment of malignant superior vena cava syndrome by endovascular stent. Lung Cancer 2004;43: 209-14.
- 15. Bovenschulte H, Bangard C, Lackner KJ. Superior vena cava stent implantation: preserving an indwelling port catheter by temporary shift of the brachiocephalic vein. Rofo 2010;182:274-5.

Submitted Nov 13, 2018; accepted Mar 11, 2019.