Acute Pericardial Effusion during Endovascular Intervention for Superior Vena Cava Syndrome: Case Series and Review

Susan K. O'Horo, M.D., M.P.H.,¹ Gregory M. Soares, M.D.,²
and Gregory J. Dubel, M.D.²

ABSTRACT

We describe three cases of acute pericardial effusion during endovascular treatment of superior vena cava (SVC) syndrome, one of which resulted in fatal pericardial tamponade. SVC syndrome results from impaired venous return from the head and upper extremities to the right atrium. Malignancy and catheter-induced SVC stenosis accounts for the majority of cases of SVC syndrome. Endovascular therapy is the treatment of choice for SVC syndrome due to its rapid relief of symptoms and low morbidity. Acute pericardial effusion and pericardial tamponade may result from inadvertent extraluminal traversal of the SVC in the so-called danger zone above the right atrium, as well as secondary to dissection of wires in a location where fascial planes have been altered by neoplasia or inflammation.

KEYWORDS: Superior vena cava syndrome, pericardial effusion, pericardial tamponade, angioplasty complication

Objectives: Upon completion of this article, the reader should understand the anatomical relationship between the pericardium, heart, and superior vena cava, and how alterations to the pericardium and superior vena cava from malignancy or inflammation may complicate the treatment of superior vena cava syndrome.

Accreditation: Tufts University School of Medicine (TUSM) is accredited by the Accreditation Council for Continuing Medical Education to provide continuing medical education for physicians.

Credit: TUSM designates this educational activity for a maximum of 1 *AMA PRA Category 1 Credit*TM. Physicians should only claim credit commensurate with the extent of their participation in the activity.

Superior vena cava (SVC) syndrome occurs when there is impaired venous return from the upper extremities, head, and neck through the superior vena cava to the right atrium.¹ Symptoms of venous congestion may include facial edema, cough, dyspnea, orthopnea, hoarseness, nausea, headache, and plethora, or distention of superficial veins. Life-threatening complications may

include cerebral or laryngeal edema.² In the vast majority of cases, mortality is due to the underlying cause of SVC occlusion. The syndrome may result from pulmonary or mediastinal malignancy (80%), mediastinal fibrosis, or from indwelling catheters or cardiac pacer leads.^{3,4}

Treatment options for SVC syndrome include surgery, radiation therapy, chemotherapy, and percutaneous

¹Department of Radiology, Division of Interventional Radiology, Brigham and Women's Hospital, Boston, Massachusetts; ²Department of Radiology, Division of Interventional Radiology, Rhode Island Hospital, Providence, Rhode Island.

Address for correspondence and reprint requests: Susan K. O'Horo, M.D., M.P.H., Instructor, Department of Radiology, Division of Interventional Radiology, Brigham and Women's Hospital, 75 Francis Street, Boston, MA 02115.

Case-Based Interventional Radiology; Guest Editors, Ron C. Gaba, M.D., Derek L. West, M.D., James T. Bui, M.D., Charles A. Owens, M.D.

Semin Intervent Radiol 2007;24:82-86. Copyright © 2007 by Thieme Medical Publishers, Inc., 333 Seventh Avenue, New York, NY 10001, USA. Tel: +1(212) 584-4662.

DOI 10.1055/s-2007-971198. ISSN 0739-9529.

transluminal angioplasty (PTA) and stenting. Endovascular therapy has become the mainstay of treatment for SVC syndrome.⁵⁻⁷ Although there are well-described complications associated with endovascular management of this entity, documented acute pericardial effusion and tamponade have been reported only rarely.^{2,4,7-10} We describe three cases of documented acute pericardial effusion during attempted recanalization for SVC syndrome, one of which resulted in a fatal pericardial tamponade. A literature review of complications associated with endovascular intervention for SVC syndrome follows.

CASE SERIES

Case 1

The first patient was a 56-year-old woman with a history of breast cancer and right internal jugular vein chest port. The patient was receiving chemotherapy for metastatic disease and had multiple recurrent episodes of port malfunction previously treated successfully with lytic infusion and mechanical fibrin sheath stripping. One week after her most recent fibrin sheath stripping, the patient developed severe facial edema and orthopnea. Chest computed tomography (CT) demonstrated an abrupt narrowing of the SVC, without evidence of significant mediastinal tumor. Via the right common femoral vein, a 5F Davis catheter (Cook, Bloomington, IN) was advanced superiorly and a Terumo Glidewire (Terumo, Ashland, MA) was used to cross the lesion. The catheter was advanced superiorly, and a test

injection demonstrated contrast outlining the pericardial sac. The catheter was retracted inferiorly, and repeat imaging demonstrated continued contrast filling of the pericardial space (Fig. 1). The patient remained hemodynamically stable. A transthoracic sonogram demonstrated a small pericardial effusion. The procedure was abandoned, and the patient was transferred to the intensive care unit for monitoring. Because she remained asymptomatic from the effusion, she returned to the procedure room the next day, and access was gained to the right internal jugular vein. Venogram demonstrated occlusion of the SVC, with multiple collateral veins around the right neck and chest (Fig. 2). The lesion was successfully traversed from above via the right internal jugular vein access site using a Kumpe catheter (Cook, Bloomington, IN) and Glidewire. A 60 × 12 mm Wallstent (Boston Scientific, Natick, MA) was placed across the occlusion and dilated to 8 mm without event. Postintervention venogram demonstrated a widely patent SVC with minimal filling of collateral veins (Fig. 3). The patient experienced immediate symptomatic relief

Figure 1 Venogram demonstrates contrast filling pericardial cavity after 5F Davis catheter (arrow) was retracted inferiorly after pericardial entry was identified.

Figure 2 Venogram performed from right internal jugular vein demonstrates occlusion of superior vena cava, with numerous collateral vessels over neck and right shoulder. Partially visualized chemotherapy port remains in position over right chest.

Figure 3 Completion venogram demonstrating excellent flow across stented superior vena cava into right atrium and ventricle.

of her orthopnea, and after 24 hours her facial swelling had resolved. The patient remained asymptomatic at the time of her death from other causes, approximately 1 month later.

Case 2

The second patient was a 70-year-old woman with known stage 4 non-small cell lung carcinoma undergoing radiation therapy who presented with worsening shortness of breath and fatigue. Physical examination revealed moderate facial swelling and bilateral jugular venous distension. Chest CT demonstrated a right upper lobe mass extending into the mediastinum causing preocclusive stenosis of the SVC and narrowing the right main pulmonary artery. Subsequently, through a right common femoral vein access, a 3-mm J-tipped wire (Cook, Bloomington, IN) was advanced without difficulty across the SVC stenosis using a Davis catheter (Cook, Bloomington, IN). The Davis catheter was exchanged for a pigtail catheter (Cook, Bloomington, IN), and a subsequent venogram demonstrated a long

segment stenosis extending from the confluence of the brachiocephalic veins to the right atrium. A 16 mm × 40 mm Wallstent was deployed without complication. Poststenting angioplasty was performed using a 14 mm × 20 mm balloon. Immediately following deflation of the balloon, the patient became apneic with pulseless electric activity. Full resuscitative measures were begun. During the resuscitation a transthoracic sonogram revealed a large pericardial effusion that was not present on the previous day's chest CT. Despite pericardiocentesis with aspiration of nonclotting bloody fluid and exhaustive resuscitative efforts, the patient expired.

Case 3

The third patient was a 73-year-old woman with a chronic indwelling venous total parenteral nutrition access port for short gut syndrome. She presented with recurrent head and arm swelling but no respiratory symptoms. The patient had a previously placed SVC stent for catheter-induced SVC stenosis. Initially, access was obtained via the right internal jugular vein. However, the stent could not be traversed. The right common femoral vein was then accessed, and a 5F Davis catheter was advanced over a 3-mm J-tipped wire to the junction of the SVC and right atrium. Using a hydrophilic Glidewire, attempts were made to traverse the occluded stent. The wire appeared to traverse the stent, although somewhat more medial than expected. After advancing the catheter carefully over the wire, a hand injection was performed and revealed contrast outlining the pericardial space. The patient was asymptomatic. Intermittent fluoroscopy and ultrasound performed over a period of approximately 10 minutes revealed no enlargement of the cardiac silhouette. The patient was monitored in the recovery room for 6 hours, and she remained asymptomatic and hemodynamically stable. She was returned to her nursing home with plans to complete her intervention at a later date. The patient died of causes unrelated to her SVC occlusion 1 month later.

DISCUSSION

Treatment options for SVC syndrome include chemotherapy, radiation, endovascular repair, and open surgical reconstruction. Due to rapid relief of symptoms and minimal procedural morbidity and mortality versus other modalities, stenting has become the procedure of choice.¹⁻⁶ Previously reported complications of endovascular repair have included stent migration, restenosis, and venous thrombosis.² Fatal complications have been attributed to arrhythmias, acute right heart failure from fluid overload, pulmonary embolism, and intracerebral hemorrhage secondary to procedural thrombolysis.⁴ Care must also be taken not to cover the brachiocephalic vein, particularly in dialysis patients in whom

maintaining access is vital. Martin reported a fatal case of pericardial tamponade in a 59-year-old man with SVC syndrome secondary to small cell carcinoma.⁷ One half hour after so-called kissing Wallstents were placed, the patient decompensated. Autopsy revealed a small perforation of the medial wall of the SVC 1.5 cm above the right atrium. Boardman reported a nonfatal pericardial tamponade in a patient also with small cell carcinoma after PTA of a chronically occluded SVC.⁸ During PTA, the patient arrested, was successfully resuscitated, and a pericardial drainage catheter was placed into an effusion seen by ultrasound. In 2003, Forauer reported two cases of nonfatal pericardial tamponade during central venous manipulation for catheter placement.⁹ In one case, a malignant pericardial effusion enlarged during SVC stenting before placement of a port for chemotherapy, and during another case hemopericardium resulted from attempted traversal of a hemodialysis catheter-related stenosis. Brown has reported a case in which balloon dilation of a malignant SVC obstruction, before placement of a self-expanding stent, resulted in SVC rupture into the pericardium, tamponade, and death.¹⁰ We present three documented cases of acute pericardial effusion during endovascular treatment of SVC syndrome.

A review of the mediastinal anatomy is useful in understanding the mechanism of such complications. The anatomy of the pericardium and its relationship to the great vessels is complex. The pericardium is composed of an inner serous and an outer fibrous layer. The visceral portion of the serous pericardium, the epicardium, invests the heart and extends superiorly along the external surface of the great vessels for ~3 cm before folding inferiorly at the pericardial reflection line. Here it becomes the parietal pericardium and is continuous with the inner aspect of the fibrous pericardium. The SVC is thus incompletely covered by serous pericardium over a length of ~3.5 cm above the right atrium.^{11,12} It is at this level where inadvertent traversal from the SVC lumen to the pericardial space occurred in our first and second cases, as well as in each of the previously reported cases where documentation of the point of violation was available. This is also the level where the greatest narrowing was present in our third case. The presence of malignancy in our first two cases, as well as four of the five previously described cases, suggests that these complex anatomical boundaries may become altered or obliterated by neoplasia. Because our third case represented a benign cause of SVC syndrome, as did one of Forauer's two cases,⁹ it seems that both benign and malignant inflammatory alteration of normal tissue planes may predispose for this complication.

Other technical factors should be given consideration as well. In several reports, perforation was clearly related to the extraluminal passage of guidewires and catheter. In addition, in several of the previously reported cases, as well as in two of our cases, hydrophilic

wires were utilized for lesion traversal. This type of wire has a propensity for subintimal dissection, especially when crossing occlusions, which is sometimes a desirable quality. However, extra care to confirm intravascular wire location above and below occlusions must be exercised to avoid pericardial violation. In our third case, it is not clear whether hemopericardium occurred with occult extraluminal traversal during recanalization or at the time of dilation, as reported by Brown and Martin. In both situations, however, it appears damage occurred very near the SVC and right atrium junction. This suggests that although careful technique may help avoid extraluminal passage of the wire, some cases of rupture into the pericardium can occur as a result of disruption of the tissue planes, which have been previously compromised by inflammation or tumor.

CONCLUSION

Although endovascular management has become widely accepted as a first-line therapy for SVC syndrome of many etiologies, it is not without complications. In addition to more commonly reported adverse sequelae, the interventional radiologist must recognize the potential for acute pericardial effusion or tamponade. In hemodynamically compromised patients, the mainstays of prompt management should include volume resuscitation and pericardiocentesis.⁹ In hemodynamically stable patients, close physiological and imaging monitoring is imperative to ensure early recognition of enlargement of the effusion or tamponade. Finally, we suggest that the short (3.5 cm) region of SVC above the right atrium be regarded as a "danger zone," warranting particularly gentle catheter and wire manipulations with SVC syndrome of any cause.

REFERENCES

1. Hennequin LM, Fade O, Bernadac PA, et al. Superior vena cava stent placement: results with the Wallstent endoprosthesis. *Radiology* 1995;196:353-361
2. Hochrein J, Bashore TM, O'Laughlin MP, Harrison JK. Percutaneous stenting of superior vena cava syndrome: a case report and review of the literature. *Am J Med* 1998;104:78-84
3. Kee ST, Kinoshita L, Razavi MK, Nyman UR, Semba CP, Dake MD. Superior vena cava syndrome: treatment with catheter directed thrombolysis and endovascular stent placement. *Radiology* 1998;206:187-193
4. Sharafuddin MJ, Sun S, Hoballah JJ. Endovascular management of venous thromboembolic diseases of the upper torso and extremities. *J Vasc Interv Radiol* 2002;13:975-990
5. Kalra M, Gloviczki P, Andrews JC, et al. Open surgical and endovascular treatment of superior vena cava syndrome caused by nonmalignant disease. *J Vasc Surg* 2003;38:215-223

6. Lanciego C, Chacon JL, Julian A, et al. Stenting as first option for endovascular treatment of malignant superior vena cava syndrome. *AJR Am J Roentgenol* 2001;177:585–593
7. Martin M, Baumgartner I, Kolb M, Triller J, Dinkel HP. Fatal pericardial tamponade after Wallstent implantation for malignant superior vena cava syndrome. *J Endovasc Ther* 2002;9:680–684
8. Boardman P, Ettles DF. Cardiac tamponade: a rare complication of attempted stenting in malignant superior vena caval obstruction. *Clin Radiol* 2000;55:645–647
9. Forauer AR, Dasika NL, Gemmete JJ, Theoharis C. Pericardial tamponade complicating central venous intervention. *J Vasc Interv Radiol* 2003;14:255–259
10. Brown KT, Getrajdman GI. Balloon dilation of the superior vena cava (SVC) resulting in SVC rupture and pericardial tamponade: a case report and brief review. *Cardiovasc Intervent Radiol* 2005;28:372–376
11. Hollingshead WH. *Textbook of Anatomy*. 4th ed. Hagerstown, MD: Lippincott Williams and Wilkins; 1985
12. Langman J. *Atlas of Medical Anatomy*. St. Louis, MO: WB Saunders Co; 1978