Endovascular Treatment with a Venovo Stent for Superior Vena Cava Syndrome in a Patient Undergoing Hemodialysis

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Abstract

Superior vena cava (SVC) syndrome is caused by obstruction of venous return at the SVC. The clinical symptoms include neck and head swelling, dyspnea and even airway compromise. We report a 72-year-old male who presented with SVC syndrome and was successfully treated with SVC stenting.

Keywords: superior vena cava syndrome, endovascular treatment, venovo stent

Background

Superior vena cava (SVC) syndrome is caused by either partial or complete obstruction of blood flow at the SVC. Clinically, this obstruction most commonly results from thrombus or tumor compression of the vessel walls, and patients may develop a series of symptoms such as neck and facial swelling, dyspnea, and cough. We report a case with SVC syndrome, including the clinical manifestations, diagnostic approach, and therapeutic strategy.

Case Presentation

A 72-year-old man has a medical history of diabetes, hypertension, old stroke, coronary artery disease, and end stage renal disease under regular hemodialysis. He presented to our emergency department with vomiting, shortness of breath, and progressive facial swelling over three days. Physical examination showed that his consciousness was clear, body temperature was 37.1°C, heart rate was 80 beats per minute, respiratory rate was 18 breaths per minute, and blood pressure was 130/70 mmHg. Additionally, neck jugular vein engorgement, right facial edema (Figure 1), and bilateral clear breath sounds were found. Laboratory data disclosed elevated white blood count ($12x10^3$/uL) with segment predominance (85.3%), and an increased hs-Troponin I level of 13072 pg/mL (reference range, 0~26 pg/mL). Electrocardiogram (ECG) showed no specific ST-T change (Figure 2). Chest X-ray film revealed neither cardiomegaly, pleural effusion, nor widened mediastinum (Figure 3). He was diagnosed with non-ST elevation myocardial infarction (NSTEMI) based on the

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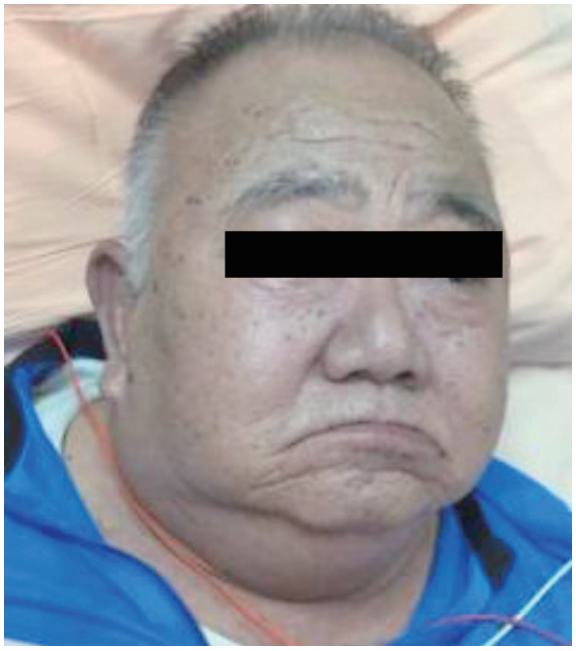

Figure 1. A 72-year-old man suffered from vomiting, dyspnea, and progressive facial swelling. Physical examination showed neck jugular engorgement and right facial edema.

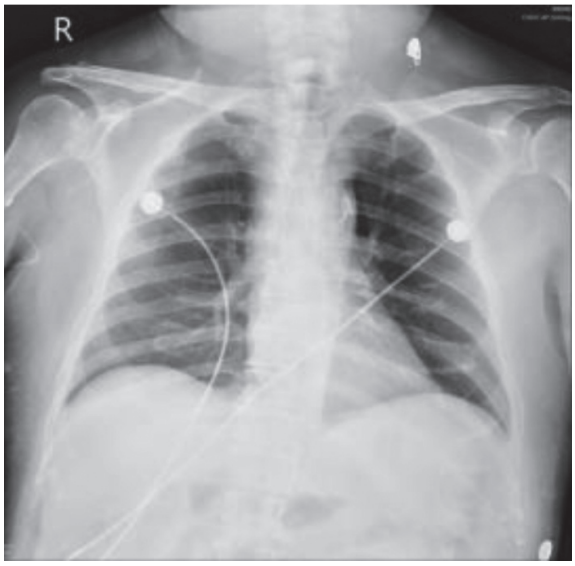

Figure 3. Chest X-ray film reveals no cardiomegaly, pleural effusion, or widened mediastinum.

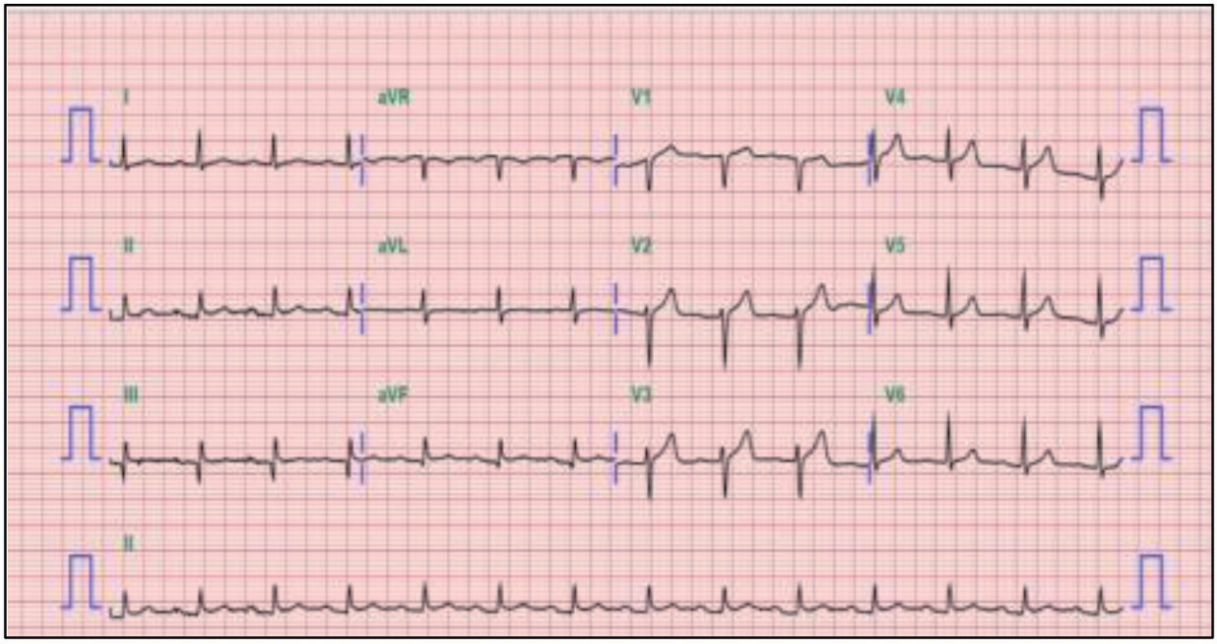

Figure 2. 12-lead ECG shows sinus rhythm and non-specific ST-T changes.

above mentioned findings, and treated with dual antiplatelet therapy (DAPT) and heparinization.

However, orthopnea, facial flushing, cyanotic lips, and right upper arm swelling could not be explained by NSTEMI. Tracing the patient's history, it was found he had just shifted from Hickman catheter to arteriovenous shunt for hemodialysis six months prior. Therefore, a chest computed tomography (CT) was arranged on suspicion of SVC syndrome, which revealed focal stenosis of the SVC accompanied by upstream total thrombotic occlusion (Figure 4). Hence, SVC thrombus complicated by SVC syndrome was diagnosed. Subsequently, we surveyed his coagulation, e.g., protein C, protein S, and anti-thrombin III, and the tests were all within normal range. Endovascular treatment consisted of direct stenting using a Venovo (Bard) self-expandable stent in size 16mm x 80mm over the total occluded SVC. We subsequently post-dilated with a 14 mm x 40 mm balloon (Figure 5). His dyspnea and facial swelling gradually subsided, and we shifted his medication to anti-coagulant (rivaroxaban) plus single anti-platelet agent (clopidogrel). He was then discharged with outpatient department follow-up, and there has been no recurrence so far.

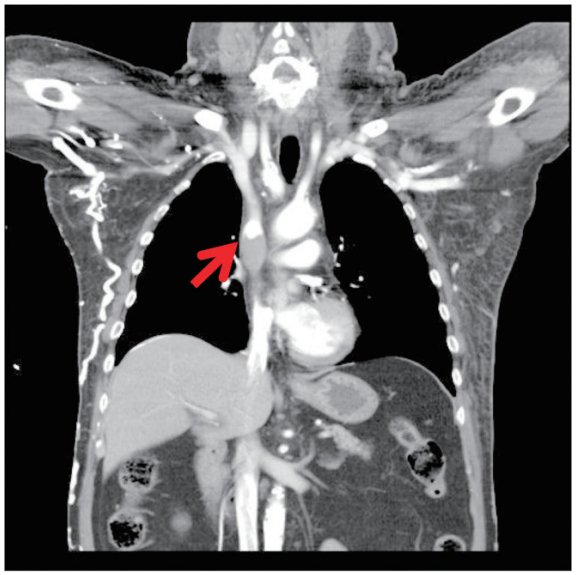

Figure 4. Chest CT shows focal stenosis of SVC associated with upstream total thrombotic occlusion (red arrow).

Discussion

Superior vena cava (SVC) syndrome refers to superior vena cava obstruction with severe reduction in venous return from the head, neck, and upper extremities. Impairment of blood flow through the SVC leads to venous engorgement proximal to the site of the obstruction.

SVC syndrome could be caused by external compression, neoplastic invasion, or internal obstruction. Malignant tumors, such as lung cancer, non-Hodgkin lymphoma and metastatic tumors, are responsible for the majority of cases. Other benign etiologies include mediastinal fibrosis, vascular disease, post-radiation therapy, and infections.[1] However, with the expanding use of intravascular devices such as central venous catheters, defibrillators, and pacemakers, the prevalence of benign causes of SVC syndrome is increasing, accounting for up to 40% of cases.[2]

Patients with SVC syndrome may present to the emergency department with a variety of symptoms, for example, facial swelling or flushing, dyspnea, cough, upper arm swelling, headache, or lightheadedness. The characteristic physical findings are distended neck veins, cyanosis, facial edema, and sometimes the development of tortuous collateral veins on the anterior chest wall.[3]

The diagnosis of SVC syndrome is usually based on history (e.g., a known malignancy) and physical examination, but imaging studies can be helpful to reach the diagnosis more accurately. Chest X-ray film may reveal mass lesion, pleural effusion, or widening of the superior mediastinum. A chest CT with contrast not only affords the diagnosis, but also gives information about the possible etiology. Magnetic resonance imaging (MRI) is increasingly used to diagnose SVC obstruction, but patients may have difficulty remaining supine for the entire examination process because of dyspnea.

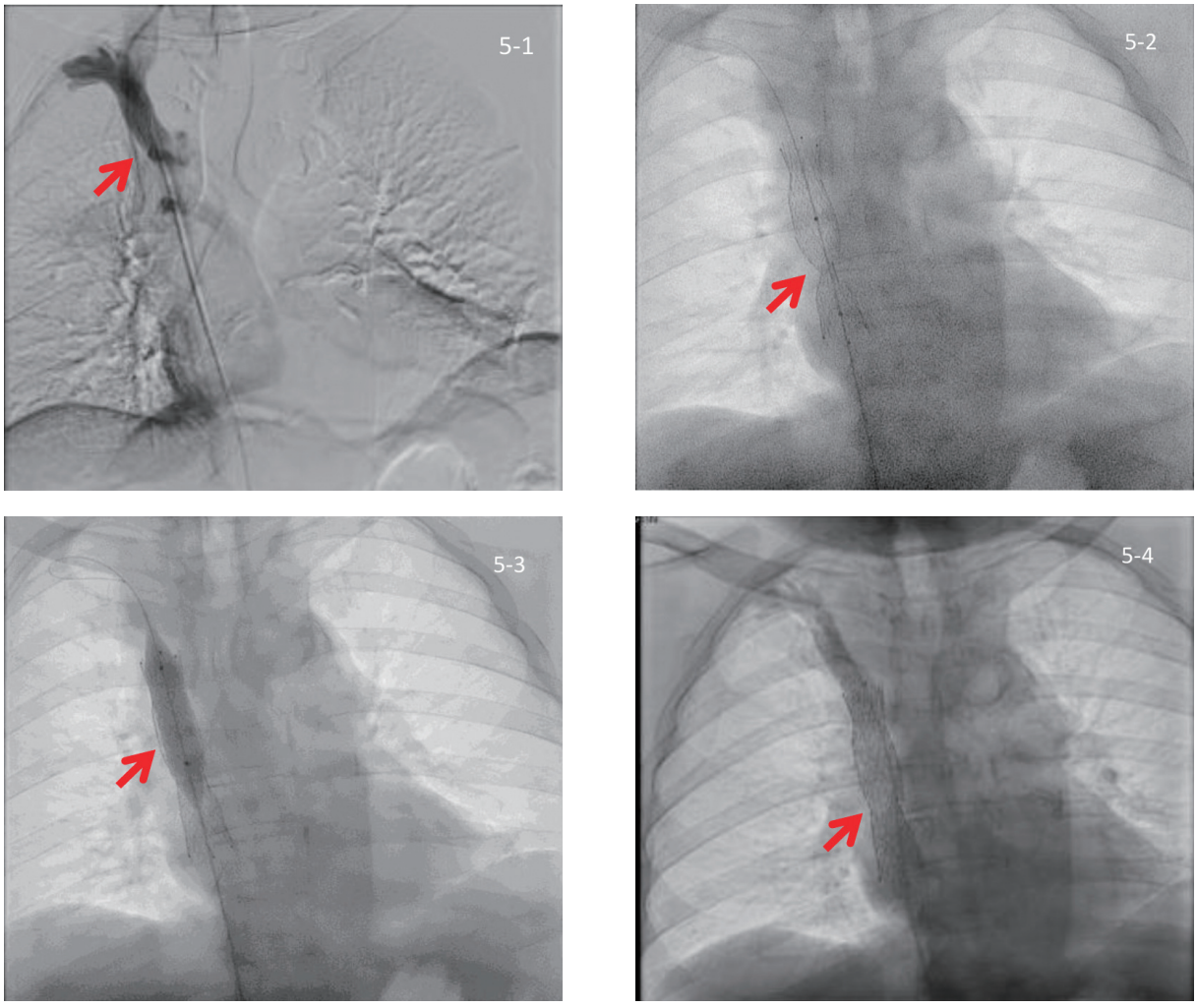

Figure 5. Cardiac angiography reveals SVC total occlusion (5-1, red arrow). Therefore, balloon angioplasty with subsequent SVC stenting (5-2 and 5-3, red arrow) was performed. The final result shows good expansion of the stent (5-4, red arrow).

In most patients, the initial therapeutic strategy includes timely diagnosis followed by timely treatment of the malignancy. Radiotherapy, chemotherapy, and surgical resection of tumors can provide curative treatment for underlying malignant etiologies. Since the first report of stenting of an SVC syndrome in 1986, early management with endovascular intervention including angioplasty and stent placement has been the usual first choice.[4] Patients suffering from SVC syndrome caused by indwelling catheters and pacemakers often receive endovascular treatment, which could be safe, effective, and less invasive.[5] On the other hand, surgical repair is the choice for patients for whom endovascular therapy is not suitable. Anti-coagulation agents, which are frequently applied in patients with atrial fibrillation, deep vein thrombosis, pulmonary embolism, or hypercoagulable states, are recommended for patients receiving SVC stent and used in those with an intravascular device-associated thrombus.[6]

We herewith report a male patient on regular hemodialysis therapy with SVC syndrome

who was the first reported case treated for SVC syndrome with a Venovo self-expandable stent. To maintain stent patency and prevent restenosis, anti-coagulation- (rivaroxaban) plus anti-platelet agents (clopidegrol) were given, whereby Clopidogrel exhibits higher potency in its anti-platelet aggregation effect.[7] In a study comparing 1896 rivaroxaban and 4848 warfarin users with stage 4 or 5 chronic kidney disease or undergoing hemodialysis, rivaroxaban appears to be associated with significantly less major bleeding compared to warfarin.[8]

Conclusion

Clinical features of SVC syndrome can be subtle or dramatic, requiring expeditious attention and intervention. Benign etiologies such as stenosis or occlusion of central veins in hemodialysis patients are common, especially with previous intravascular catheter- or device use. Palliative therapy with endovascular treatment plus anti-coagulatives may offer a useful treatment strategy.

References

1. Zimmerman S, Davis M. Rapid Fire: Superior vena cava syndrome. *Emerg Med Clin North Am* 2018;36(3):577-84.
2. Agarwal AK, Khabiri H, Haddad NJ. Complications of vascular access: Superior vena cava syndrome. *Am J Kidney Dis* 2017;69(2):309-13,
3. Wilson LD, Detterbeck FC M, Yahalom J. Superior vena cava syndrome with malignant causes. *N Engl J Med* 2007;356:1862-9.
4. Rachapalli V, Boucher LM. Superior vena cava syndrome: role of the interventionalist, *Can Assoc Radiol J* 2014;65(2):168-76.
5. Sfyroeras GS, Antonopoulos CN, Mantas G, et al. A review of open and endovascular treatment of superior vena cava syndrome of benign eetiology. *Eur J Vasc Endovasc Surg* 2017;53:238-254.
6. Ratzon R, Tamir S, Friehmann T, et al. Thrombosis, anticoagulation and outcomes in malignant superior vena cava syndrome. *J Thromb Thrombolysis* 2019; 47(1):121-8.
7. Alonso-Coello P, Bellmunt S, McGorrian C, et. al. Antithrombotic therapy in peripheral artery disease: antithrombotic therapy and prevention of thrombosis, 9th ed. American College of Chest Physicians evidence-based clinical practice guidelines. *Chest* 2012;141 (2 Suppl):e669S-90S
8. Coleman CI, Kreutz R, Sood NA, et al. Rivaroxaban versus warfarin in patients with monvalvular atrial fibrillation and severe kidney disease or undergoing hemodialysis. *Am J Med* 2019;132(9):1078-83.