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Treatment of pacemaker-induced superior vena cava syndrome by venoplasty with a coronary balloon

Le Tra Pham MBBS

Ha Noi Heart Hospital, Ha Noi, Vietnam

Correspondence
Le Tra Pham, Ha Noi Heart Hospital, 98 Tran Hung Dao Street, Ha Noi, Vietnam.
Email: leanhkado@gmail.com

Venous thrombosis or stenosis frequently occurs after implanting transvenous pacemaker leads, and it is usually asymptomatic. The reported incidence is 30%–64%. The mandatory treatments are balloon angioplasty, stenting, thrombolytic, mechanical thrombectomy, and venous grafting. We present a case with the special cooperation of an electrophysiologist and a coronary interventionist in Ha Noi Heart Hospital, Vietnam, to treat an implanted pacemaker patient with fracture ventricular lead and superior vena cava syndrome.

KEYWORDS

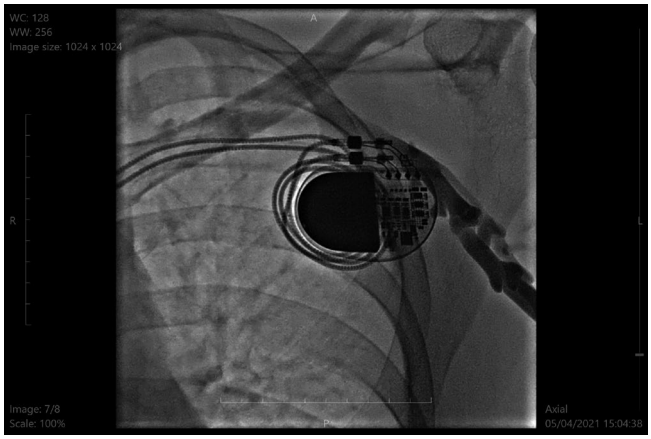
lead fracture, superior vena cava syndrome

1 | INTRODUCTION

Venous thrombosis or stenosis frequently occurs after implanting transvenous pacemaker leads, and it is usually asymptomatic. The reported incidence is 30%–64%.[1,2] But the incidence of pacemaker-induced superior vena cava syndrome is extremely low ranging from 1/40 000 to 1/250 patients.[3,4] The thrombus formation, stenosis, and inflammation process on the vessel wall can be related to the mechanical stress caused by pacemaker leads and ultimately leads to obstruction and occlusion. It usually occurs in the early stage but sometimes, we also detect a few cases in the later stage (2–5 years) after implanting a pacemaker. Venous obstruction can be asymptomatic because of the progressive formation of collateral veins including azygous veins. But in some scenarios, without collateral circulation, the patient can develop intermediate to severe symptoms like headache, inability to bend over without flushing, exercise-induced flushing. The mandatory treatments are balloon angioplasty, stenting, thrombolytic, mechanical thrombectomy, and venous grafting. We present a case with the special cooperation of an electrophysiologist and a coronary interventionist in Ha Noi Heart Hospital, Vietnam to treat an implanted pacemaker patient with fracture ventricular lead and superior vena cava syndrome.

2 | CASE PRESENTATION

A 43-year-old male patient was admitted to our Emergency Department for two episodes of presyncope, fatigue, and many specific symptoms like chest pain, shortness of breath, feeling of fullness in head and ear, swelling of face and neck, and coughing. All of these symptoms make us concern about SVC syndrome. He was implanted a DDDR pacemaker to treat third-degree AV block after prosthetic valve replacement 5 years ago in our Cardiac Electrophysiology Department. His vital signs are as follows: heart rate: 40 beats/min, blood pressure: 120/80 mmHg, temperature: 37°C, respiratory rate: 20 beats/min, and ECG: third-degree AV block with ventricular response rate is 40–50 beats/min. After checking the permanent DDDR pacemaker, we realized that the ventricular lead had been an inability to pace. Under fluoroscopy, we detected the fracture ventricular lead confidently (Figure 1). By explaining to the patient and his relatives about his illness condition and risk of sudden death without new ventricular lead, they ultimately accepted to implant one more ventricular lead, so we prepared the patient to insert a new ventricular lead by replacing the fracture 2 days after his admission. After puncturing the left subclavian vein and inserting sheath, we were in trouble with inserting 0.035 Terumo hydrophilic guidewire



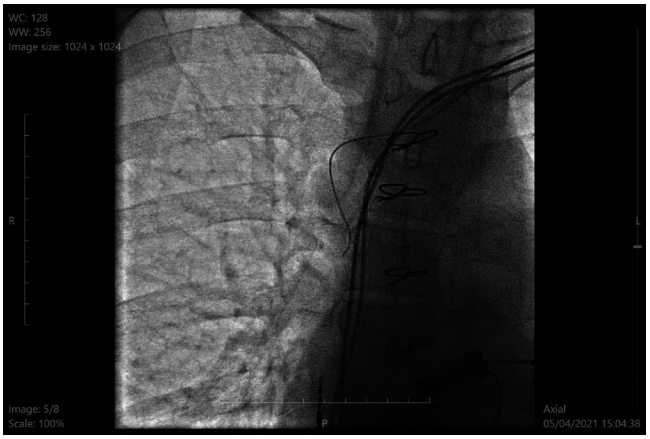


FIGURE 1 Fracture RV lead under fluoroscopy

FIGURE 2 Trouble with inserting 0.035 Terumo hydrophilic guidewire to pass SVC obstruction

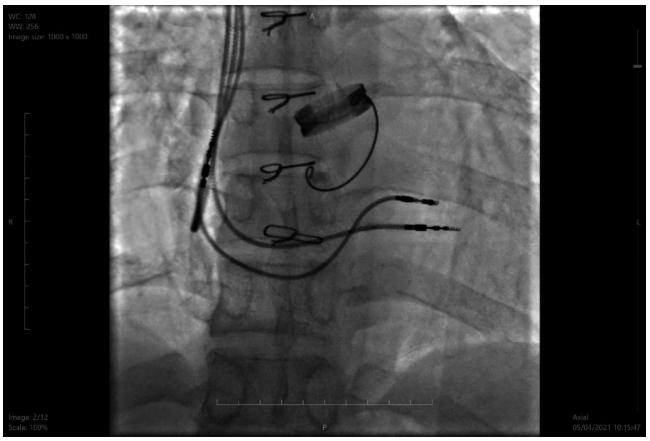


FIGURE 4 Final result—successfully implanting new RV lead on the low anterior septum RV

through the superior vena cava (SVC), the guidewire could not pass through the superior vena cava in spite of our encouraging to manipulate (Figure 2). A venogram was done and the obstruction SVC was confirmed (Figure 3). The site of obstructed SVC is just inferior to right brachiocephalic 3 cm and the length of obstructed SVC is about 4 cm. With little experience in these scenarios and without equipment like venous balloons, venous stents, and excimer laser sheaths during lead extraction in our institute, we consulted the coronary interventionist before consulting the cardiac surgery and tried to use Juking Right guiding catheter 6F and a 0.014 inch coronary wire (Asahi Sion Blue 0.014 × 180 cm) without curve bending to pass through the obstruction, and fortunately, it was too easy in the first time. Our coronary interventionist used a small over-the-wire coronary balloon (3.0 × 20 mm) to dilate slowly, carefully, and gradually SVC with pressure ranging from 18 to 24 atm. We dilated the SVC four times, did a venogram again, and confirmed that the obstruction was resolved partly without any evidence of SVC perforation (contrast flowed slowly via SVC without contrast leaking—the contrast flowing via SVC is better). Rewiring through SVC by 0.035 Terumo hydrophilic guidewire was successful. We continuously inserted the long sheath (Medtronic 060037A Introducer Sheath 6Fr × 23 cm)

FIGURE 3 Venogram confirmed obstruction in SVC and collateral circulation in azygos vein

over the wire, through the obstruction, withdrew the wire, cut the hemostasis valve afterward, and ultimately inserted the new ventricular lead in the luminal of the long sheath successfully. After implantation the new ventricular lead to the high right ventricular (RV) septal, we cut carefully along the trunk of the long sheath, withdrew its while withholding the ventricular lead to stabilize the ventricular lead tip on high ventricular septal to prevent its malformation. And the final result of this special procedure is shown in Figure 4. One month after finishing the procedure, the patient is now stable, has no coughing, no swelling of face and neck, no chest pain, shortness of breath, feeling fullness in head and ear anymore.

3 | DISCUSSION

We presented a rare case diagnosed pacemaker-induced SVC syndrome and fracture RV lead treated successfully with venoplasty by a coronary balloon without stenting SVC. SVC syndrome is rare

but severe and difficult to treat in clinical practice especially with nonexperience electrophysiologists. The only way to confirm the diagnosis is a venogram, and computed tomography cannot detect completely the obstruction in SVC. Venography not only can help us to detect the obstruction but also can provide characterization anatomy of SVC, the site, the extent of the obstruction to make the best decision to treat the patient. Most fractures lead site occurs in the area lateral to the subclavian vein, and the most selective treatment of many authors is implanting new lead into the right heart.

Many authors report successfully treat these patients with venoplasty with or without stenting SVC. But the complications like SVC leaking, perforation, trauma can occur during the procedure. So that we consulted the coronary interventionist to make the decision and choose the treatment strategy (using coronary wire and balloon to resolve the obstruction, using a long sheath and modifying it to insert and implant RV lead) for this patient and fortunately it is successful.

Although there is a lack of evidence or research comparing the outcome or prognosis between endovascular surgery (thoracotomy) and venoplasty with or without SVC stenting, clinician physicians should try venoplasty the first and if it fails, the surgery is the last resort. By publishing this case study, we want to share our real experience, point of view in SVC syndrome treatment in our center in Vietnam which is a developing country, and shortage of equipment like excimer laser sheath and venous stent. Despite the reconstructed SVC can occur in the future because there are three leads in SVC, but with our poor condition and patient's financial condition, we think it is acceptable to try this way to treat him.

CONFLICT OF INTEREST

The author declares no conflict of interest for this article.

ORCID

Le Tra Pham https://orcid.org/0000-0003-3958-3648

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