Delayed Presentation of Totally Avulsed Right Superior Vena Cava After Extraction of Permanent Pacemaker Lead

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LEACCHE, M., ET AL.: **Delayed Presentation of Totally Avulsed Right Superior Vena Cava After Extraction of Permanent Pacemaker Lead.** Pacemaker lead extraction has been shown to be an effective and safe treatment for infected permanent pacemaker leads, however, they may lead to potentially serious complications, usually occurring during the extraction procedure. This report describes a case of a 48-year-old woman with a patent persistent left SVC and an infected permanent pacemaker lead of a DDD pacing system who underwent transvenous laser-assisted lead extraction using a combined SVC and femoral approach. Two days after the procedure the patient developed symptoms of SVC obstruction requiring surgical intervention. The right SVC was found to be almost completely destroyed with only a thin strip of the lateral wall intact and active bleeding. The probable causative mechanisms and surgical management are discussed. (PACE 2004; 27:262–263)

right superior vena cava avulsion

Introduction

Pacemaker lead extraction is the treatment of choice for infected permanent pacemaker leads. The probability of major complications during this procedure is 0.6–3.3%, and since the majority are present during the extraction procedure they can be treated immediately. This report describes a patient with a persistent left superior vena cava (SVC) in whom total avulsion of the right SVC occurred during lead extraction with a delayed presentation.

Case Report

A 48-year-old woman was admitted with an infected permanent pacemaker lead of a DDD pacing system that was placed for sinus node dysfunction when she was 28 years old. At the age of 7, the patient underwent an atrial septal defect (ASD) repair with pericardial patch via sternotomy. In addition, the patient had a patent persistent left SVC. Transvenous, laser-assisted lead extraction was undertaken using a combined SVC and femoral approach. The procedure was complicated by a mild transient hypotension related to extraction of the lead from the right SVC. A small nonexpanding hematoma abutting the right SVC was documented on intraoperative transesophageal echocardiography (TEE), but the patient remained stable and the procedure was completed. The ventricular pacing lead system was removed only partially, while the

atrial leads were completely removed. A temporary transvenous pacemaker was installed.

On postoperative day 1 the patient developed mild tachycardia. Symptoms of SVC obstruction began as facial and right arm swelling. Chest computed tomography (CT) revealed a hematoma measuring 4.5 cm in diameter in the region of the right SVC and a moderate right pleural effusion (Fig. 1). Intravenous contrast failed to document extravasations but also revealed a completely occluded right SVC. Two days after permanent pacemaker lead extraction, the patient demonstrated a 6-point decrease in hematocrit and further signs of SVC syndrome. Cardiac surgery was consulted and the patient was brought to the operating room for control of bleeding, reconstruction of the right SVC, and concomitant placement of epicardial pacing leads. The previous median sternotomy was reopened on femoral cardiopulmonary bypass. After partial removal of a well-formed thrombus, the right SVC was found to be almost completely destroyed with only a thin strip of the lateral wall intact. The thrombus and small remaining part of the permanent pacemaker lead were then removed. There was no major backbleeding from the avulsed subclavian and jugular veins, which extended underneath the clavicle. At this point, reconstruction of the right SVC was considered.² However, since the patient had significant chronic stenosis of the right SVC and a widely patent left SVC, ligation of the right SVC was considered to be the best course of action. Therefore, the right subclavian and jugular veins were ligated. The right SVC and right atrium were oversewn using 4-0 prolene. The patient was weaned from cardiopulmonary bypass. Permanent epicardial ventricular and atrial pacing electrodes were

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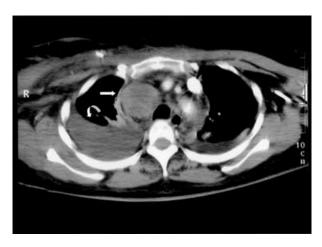


Figure 1. Contrast-enhanced computed tomography scan showing the hematoma in the region of the right superior vena cava (straight arrow) and the right pleural effusion (curved arrow).

applied and connected to the new DDD pacemaker. The patient was extubated on postoperative day 2, after her oropharyngeal swelling had decreased. She was discharged a week later in good condition with complete resolution of her SVC syndrome.

Discussion

After implantation of permanent pacemakers 0.5–7% of these become infected.³ In the case of a localized infection, local treatment of the pocket and contralateral insertion of a new pacemaker unit is the preferred treatment, while an infected pacemaker lead requires its extraction. Internal traction techniques using locking stylets and outer sheets via the implant vein (the superior vein approach) to grab the pacemaker lead, or sheaths, snares, and retrieval baskets via the femoral vein have been reported to be able to extract a high proportion of permanent pacemaker leads.³

Recently, laser sheaths, using photoablation, have been introduced to interrupt the scar tissue binding the lead to the myocardium or veins and, therefore, minimizing the risk of injury during permanent pacemaker lead extraction.⁴ The SVC is a thin low blood pressure vessel that can be injured

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during stenting or venous catheter procedures. Injury is mostly limited to perforation or laceration. Previously, a laceration of the SVC due to polymer sheath advancement leading to the hemothorax has been described, but its presentation was immediate.³ Smith et al.⁵ described a delayed SVC perforation after wallstent insertion for SVC obstruction related to mediastinal malignancy. The delayed presentation, 6 months after the procedure, was probably due the gradual erosion of the SVC made fragile from chemotherapy and tumor shrinkage of the SVC wall. The present report is the only case of a SVC avulsion during a transvenous pacemaker lead extraction with a delayed presentation. In the present study the delayed presentation in the patient was due the presence of a left SVC that is commonly associated with congenital cardiac defects and with an ASD. Sinus node dysfunction may also be present with an ASD,⁶ which in this case led to pacemaker implantation for the patient.

Normally, a persistent left SVC would be insignificant with regard to hemodynamics. However, in this patient, because of the chronic stenosis in her right SVC, likely related to the indwelling permanent pacemaker leads, the left SVC was widely patent, delaying the symptoms of SVC syndrome. Later a large hematoma developed leading to the development of obstruction not only in the right SVC, but also in the left SVC. Due to the presence of a widely patent left SVC, the presentation of this complication was not immediate and did not manifest until at least 1 day after the operation, when the hematoma was large enough to obstruct the left SVC.

After removal of the large thrombus, it was discovered that the right SVC was mostly destroyed. In a patient with normal anatomy, SVC reconstruction would have been appropriate, and this was considered at first. However, the presence of a left SVC provided an alternative because of its wide patency. Thus, after ligation of the remaining right SVC, ligation of the jugular and subclavian veins was performed. This allowed blood to be shunted through the left SVC, which was widely patent after evacuating the hematoma, thus relieving the SVC syndrome.