Unique clinical presentation and management of lead-stent abrasion

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Introduction

Pacemaker-induced superior vena cava (SVC) syndrome is a rare complication of permanent pacemaker placement with a reported incidence of <0.1%.[1] While it has a low mortality risk, this complication is associated with significant morbidity. Treatment options include anticoagulation, thrombolysis, surgical SVC bypass, and percutaneous venoplasty. There is no current standard of care for the treatment of this complication.[2] In the past decade, there has been an increasing number of reports detailing the treatment of pacemaker-induced SVC syndrome with percutaneous stent placement and concomitant lead extraction and replacement.[3] We describe a case of pacemaker-induced SVC syndrome treated by lead extraction and vascular stent placement, resulting in an unusual clinical presentation of a novel mechanism of lead failure.

Case report

A 69-year-old man presented with traumatic syncope complicated by a displaced C4 fracture. An electrophysiology study revealed sinus node dysfunction and 2:1 infra-Hisian block. A dual-chamber permanent pacemaker was placed. The patient had no further syncope. Two years later, he presented with acute facial plethora and swelling. A chest computed tomography scan showed a mid-SVC stenosis with overlying thrombus. The patient was started on anticoagulation, which resulted in significant improvement in his symptoms. However, 2 years later, he developed recurrent facial swelling. A repeat chest computed tomography scan showed mid-SVC stenosis with complete obliteration of the SVC associated with extensive azygos and right internal mammary collateralization (Figure 1). The decision was made to perform a lead extraction followed by SVC and left brachiocephalic stent placement. Leadless pacemaker implantation via enrollment in the LEADLESS II study[4] was considered. However, because of the presence of elevated right ventricular systolic pressures as detected by echocardiographic measurement, he was not considered a candidate on the basis of the trial's initial exclusion criteria. Therefore, dual-chamber pacemaker implantation after lead extraction was planned.

Successful extraction of the pacing leads with laser energy was performed without complication. Access was retained, and balloon dilation of the SVC and left brachiocephalic and left subclavian veins was performed followed by implantation of multiple stents. After additional post–stent balloon dilation, 2 pacing leads were inserted via retained guidewire access. The patient tolerated the procedure well and was discharged on aspirin, clopidogrel and

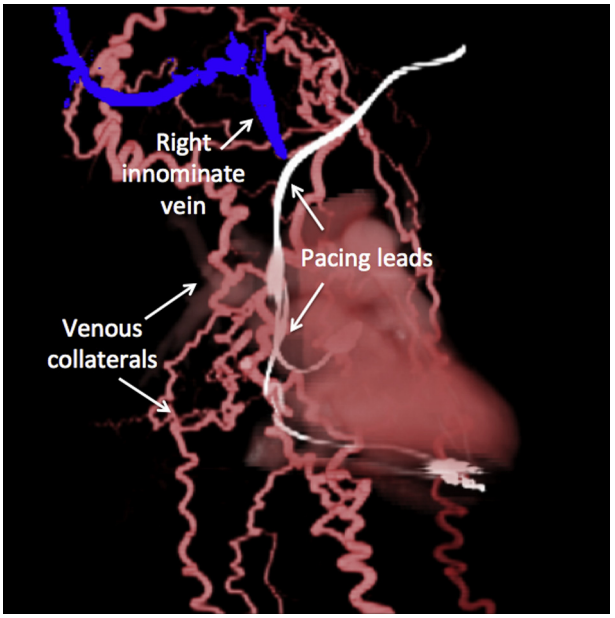

Figure 1 Three-dimensional rendering of chest computed tomography scan of the patient. Course of the transvenous atrial and ventricular pacing leads (*white*) are shown. At the junction of the right brachiocephalic vein (*blue*) and lead entry into the superior vena cava, there is complete absence of contrast consistent with complete superior vena cava occlusion. Extensive collateral venous flow is seen (*orange*).

KEYWORDS Lead insulation failure; Lead noise; Lead-stent abrasion; Pacemaker lead malfunction; Superior vena cava syndrome
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KEY TEACHING POINTS

- This case reports describes an unusual presentation of lead malfunction after venous stenting and transvenous pacemaker reimplantation for the treatment of pacemaker-induced superior vena cava syndrome.
- Stent-pacing lead abrasion led to pectoral stimulation in addition to electrical lead malfunction characterized by detection of lead noise and elevated pacing thresholds.
- Replacement of the transvenous pacing system with a leadless ventricular pacemaker resulted in improved outcomes with resolution of symptoms from superior vena cava syndrome as well as freedom from pacing malfunction.
- The case highlights the need for vigilance for complications from transvenous pacing lead placement through stented veins and the potential role of leadless pacing in patients with superior vena cava syndrome.

warfarin. After 2 weeks, the patient had significant improvement in his symptoms. However, device interrogation showed a decreased right ventricular lead impedance of 399 Ω from an implantation value of 761 Ω. Moreover, the right ventricular lead capture threshold increased to 2.5 V @ 0.4 ms from 0.5 V @ 0.5 ms at implantation. The patient was scheduled for close follow-up.

Nine days later, the patient presented with symptoms of chest thumping and left arm twitching (Supplemental Video 1). Device interrogation revealed further decrease in right ventricular lead impedance to 200 Ω as well as failure of lead capture at maximum device output. Furthermore, recorded lead electrograms showed noise in both the atrial and ventricular leads (Figure 2). These findings as well as the presence of pectoral stimulation suggested that the patient's presenting symptoms were likely related to acute insulation breach of both leads because of abrasion with the venous stent.

The patient underwent removal of both pacing leads. Fluoroscopy revealed close apposition of the pacing leads to the subclavian stent edge (Figure 3A). Returned product analysis confirmed cuts through the insulation in both the atrial lead (12.7 cm from the atrial lead pin) (Figure 3B) and the ventricular lead (22.3 cm from the ventricular lead pin) (Figure 3C). In order to avoid further complications from a transvenous pacing system, a repeat echocardiogram was recorded, which now revealed normal right ventricular systolic pressures. The patient was therefore eligible for the LEADLESS II study[4] and was enrolled in the study protocol. He underwent successful implantation of a leadless pacemaker (Nanostim, St. Jude Medical, St. Paul, MN) without complications. At 18 months of follow-up, his symptoms remain controlled.

Discussion

This case illustrates an unusual presentation of a rare complication of stenting for pacemaker-induced SVC syndrome.

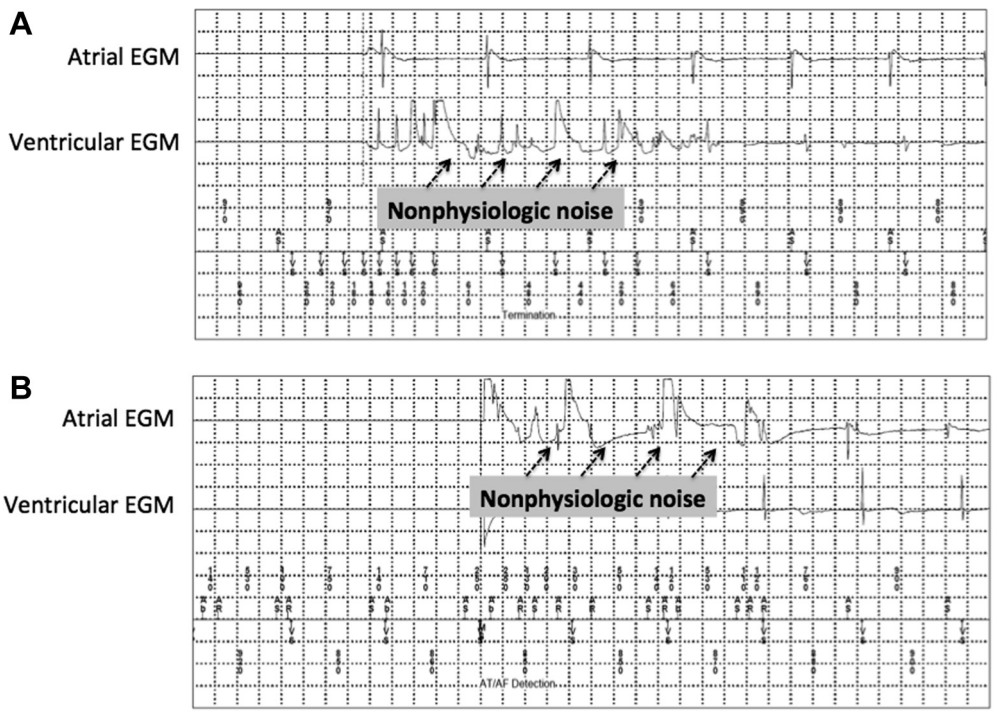

Figure 2 Intracardiac electrograms from pacemaker interrogation 3 weeks after device implantation. High-frequency, nonphysiologic signals (*dashed arrows*) are seen on the ventricular electrogram channel (**A**) and the atrial electrogram channel (**B**). EGM = electrogram.

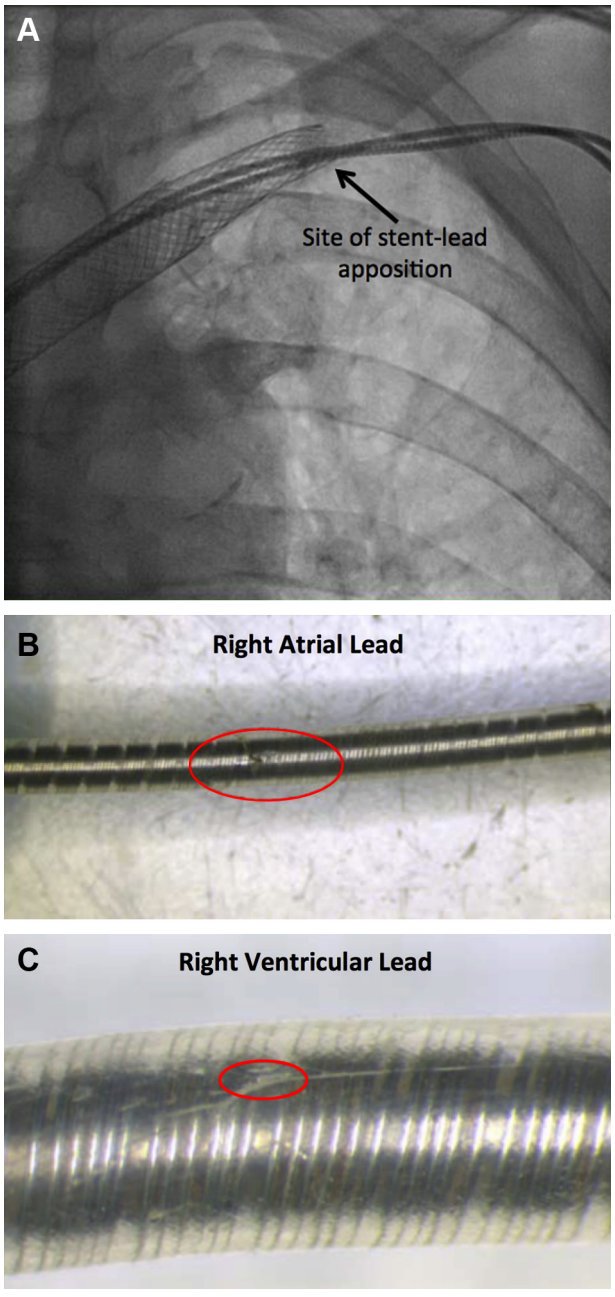

Figure 3 Procedural findings during lead explanation after the identification of electrical lead malfunction. **A:** Fluoroscopy reveals apposition of the pacing leads at the lateral edge of the left subclavian stent. **B:** Visual evidence of insulation damage of the atrial lead (*red oval circle*). **C:** Visual evidence of insulation damage of the ventricular lead (*red oval circle*).

Because of pacing lead insulation failure from abrasion by a subclavian venous stent edge, current leakage caused pectoral stimulation during pacing. In addition, noise was detected on both the atrial and ventricular pacing leads. Leadless pacing ultimately allowed more definitive treatment of transvenous pacemaker–induced SVC syndrome.

To our knowledge, there been a single case report on stent-associated damage to pacing leads, resulting in electrical malfunction.[5] In that case, it was hypothesized that a stent placed in the right innominate vein for treatment of SVC syndrome directly compressed an atrial pacing lead, causing complete electrical failure. However, lead explantation was not performed and there was therefore no direct evidence of stent-induced damage to the lead. In contrast, insulation damage was evident on analysis of both explanted leads in our case at precisely the site of stent edge contact with the leads in the subclavian region. Moreover, this damage led to local current leak, which was manifest by direct muscle stimulation at the site during pacing.

Our case report underscores several important points. Despite the availability of vascular stenting as an effective treatment modality for pacemaker-induced SVC syndrome, the need for continued pacing raises important considerations for the management of the patient. First, the risks and benefits of transvenous pacemaker implantation poststenting, epicardial pacing, and leadless pacing need to be weighed. If a transvenous pacemaker is placed, the risk of recurrent lead–induced venous occlusion is nontrivial. In addition, technical considerations during stent placement to minimize stent-lead abrasion should be made. Avoidance of close proximity of the stent to the entry site of the pacing leads may have reduced the likelihood of the kind of lead damage observed in our case. Nonetheless, the risk of stent-transvenous lead interaction cannot be completely eliminated in all cases, especially when the stenosis itself includes the entry point. Second, epicardial pacing can be considered but is more invasive and is associated with greater perioperative morbidity. Finally, leadless pacemakers offer the obvious advantage of avoiding transvenous leads altogether, which eliminates the risk of stent-lead interaction and may reduce the risk of recurrent venous occlusion. However, at this time, atrial leadless pacing is not available. Therefore, for patients in sinus rhythm with high ventricular pacing requirements, ventricular-only leadless pacing may lead to debilitating symptoms from pacemaker syndrome. As advances in leadless pacing technology are made, options for preventing and treating pacemaker-induced SVC syndrome should become more readily available.

Conclusion

Stent implantation for the treatment of transvenous pacemaker-induced SVC syndrome followed by lead re-implantation can lead to stent-lead abrasion. Leadless pacing can be an effective alternative to conventional pacing in patients with SVC syndrome.

Appendix

Supplementary data

Supplementary data associated with this article can be found in the online version at https://doi.org/10.1016/j.hrcr.2017.10.006.

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