

Malpositioned endocardial left ventricular pacing lead extraction with transcatheter cerebral embolic protection in the setting of multiple prior embolic strokes



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Introduction

Pacing lead malposition in the left ventricle increases risk of embolic stroke. We present a case of malpositioned left ventricular (LV) lead extraction using transcatheter cerebral embolic protection (TCEP) for procedure-related stroke protection.

Case report

An 84-year-old man with paroxysmal atrial fibrillation, sick sinus syndrome, and single-chamber pacemaker implantation at an outside hospital 4 years prior to admission was admitted with transient stroke-like symptoms. He had experienced multiple prior embolic strokes following pacemaker implant despite therapeutic levels of warfarin, all of which had been treated and managed at an outside institution.

The patient presented to our hospital for the first time with right-sided facial droop, right-sided weakness, and transient aphasia, all of which had resolved upon arrival to the emergency room. Physical examination was notable for mild cognitive dysfunction, unchanged from baseline, according to the patient's family. A 12-lead electrocardiogram showed sinus rhythm with infrequent ventricular paced beats with right bundle branch block morphology. International normalized ratio was 3.0. Head computed tomography showed chronic infarctions of the left frontal lobe, left lateral pons, and bilateral cerebellum.

Chest radiograph showed a large hiatal hernia with rightward displacement of the cardiac silhouette (Figure 1). A left

KEY TEACHING POINTS

- Lead malposition may occur owing to inadvertent subclavian or axillary arterial access, presence of a patent foramen ovale or atrial septal defect, or interventricular septal perforation.
- Pacing leads in the left heart increase the risk of embolic stroke.
- Transcatheter embolic protection devices may have a role in left ventricular endocardial lead extraction if thrombus is clearly visualized by echocardiography, or in cases in which the presence of thrombus cannot be fully excluded despite a history of neurologic events.

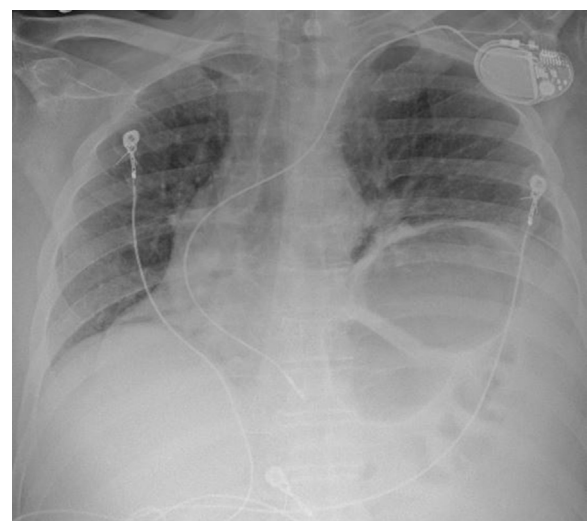


Figure 1 Chest radiograph of single-chamber pacemaker; a large hiatal hernia is present with rightward displacement of the cardiac silhouette.

KEYWORDS Embolic stroke; Left ventricular lead extraction; Malpositioned left ventricular pacing lead; Pacing lead thrombus; Transcatheter cerebral embolic protection
(Heart Rhythm Case Reports 2019;5:552–554)

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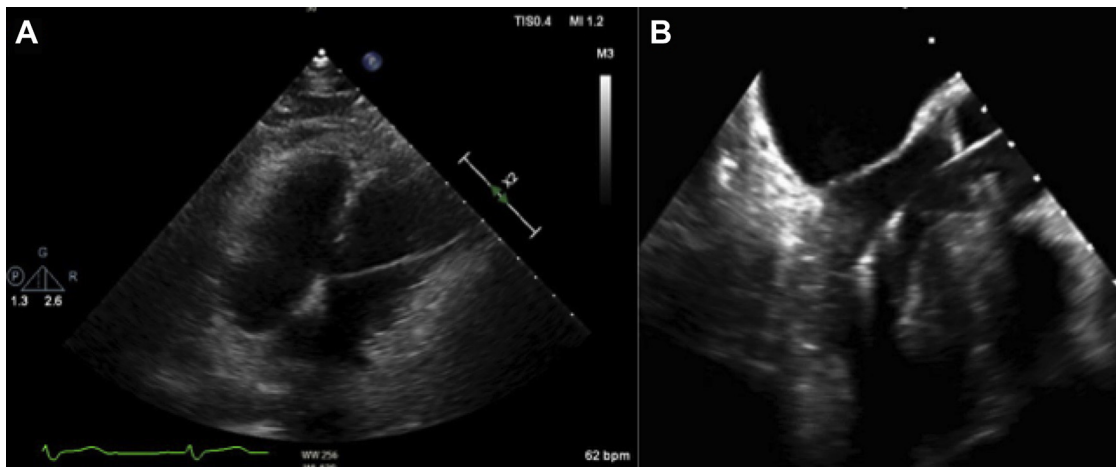


Figure 2 A: Transthoracic echocardiogram showing the pacing lead in the left ventricle. B: Transesophageal echocardiogram showing the pacing lead crossing retrograde across the aortic valve into the left ventricle.

pectoral single-chamber pacemaker was present. Transthoracic echocardiogram was performed and notable for the presence of a pacing lead crossing the aortic valve into the left ventricle (Figure 2A).

Transesophageal echocardiogram was performed but there was difficulty clearly visualizing the entirety of the lead; the possibility of thrombi or mobile fibrinous material on the lead itself was unable to be fully excluded (Figure 2B).

After full discussion with the patient and family regarding our concern for a malpositioned chronic LV endocardial pacing lead as the source for recurrent embolic strokes, informed consent was obtained to proceed with LV lead extraction. An aortogram was performed, and a TCEP device (Sentinel, Boston Scientific, Marlborough, MA) was deployed from a right radial arterial approach. Filters were positioned in the brachiocephalic and left common carotid arteries (Figure 3). The LV lead was extracted after screw retraction with manual traction. No thrombus or fibrinous material was visible upon inspection of the pacing lead. The TCEP device was then removed without evidence of debris in either filter. A new dual-chamber pacing system was implanted in the right atrium and right ventricle. The patient tolerated the

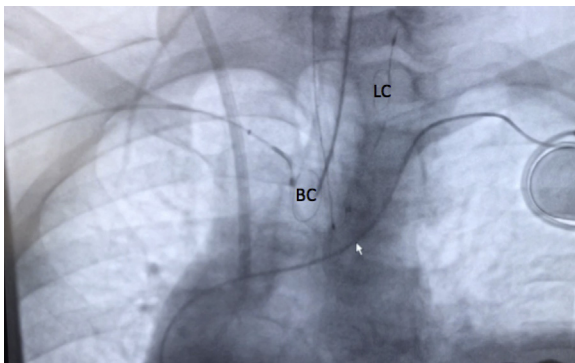


Figure 3 Transcatheter cerebral embolic protection device. Filters are positioned within the brachiocephalic (BC) and left common (LC) carotid arteries prior to left ventricular lead extraction.

procedure without complication and was discharged home the following day.

Discussion

Pacing lead malposition in the left ventricle was first reported in 1969.¹ The rate of inadvertent lead malposition is not fully known, likely related to underreporting.² Lead malposition may occur owing to inadvertent subclavian or axillary arterial access, presence of a patent foramen ovale or atrial septal defect, or interventricular septal perforation.^{1,3} The rate of embolic stroke related to a pacing lead in the left heart is also not fully known. Most patients presenting with neurologic symptoms do not have echocardiographically visualized thrombus on the pacing lead; however, evidence of thrombus by echocardiography is highly associated with neurologic symptoms.⁴ Once a malposition is diagnosed, treatment options for prevention of further neurologic events include endovascular lead extraction or open cardiac surgical extraction. Each treatment option involves risks that must be carefully considered, specific to each individual case.

To the best of our knowledge, this is the first case of TCEP deployed at the time of malpositioned LV lead extraction. Shown to be effective for capture of embolic debris at the time of high-risk transcatheter aortic valve replacement,^{5,6} TCEP may have a role in LV endocardial lead extraction if thrombus is clearly visualized by echocardiography, or in cases such as ours in which the presence of thrombus cannot be fully excluded despite a history of neurologic events. Of note, the TCEP device does not protect the left subclavian or left vertebral arteries from embolization. Further study is required to better understand the risks and benefits of TCEP at the time of malpositioned LV endocardial lead extraction.

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