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CASE REPORT
Electrophysiology

Inadvertent septal perforation during conduction system pacing device implant: a case report

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Background There has been recent growing interest in the use of conduction system pacing (CSP) for both bradycardia and heart failure indications. There remains a paucity of data, however, regarding complications related to the intraventricular septum associated with CSP implant and the management of these events.

Case summary We present a case of a patient with non-ischemic dilated cardiomyopathy presenting for cardiac resynchronization therapy in whom left bundle branch area pacing was complicated with interventricular septal perforation and managed intra-procedurally with repositioning of the lead to provide His bundle pacing (HBP) for QRS correction of underlying left bundle branch block. Post-procedure echocardiography did not show persistent ventricular septal defect. Left ventricular ejection fraction improved from 13% four months before implant to 30% at 32 months post-implant. Corrective HBP pacing thresholds showed a rise at 3-year follow-up.

Discussion Interventricular septal perforation during CSP is a possible complication during lead fixation. Pre-operative septal assessment with imaging can be helpful to provide important septal anatomical features. Septal perforation can be managed appropriately with lead repositioning intra-procedurally and close follow-up.

Keywords Conduction system pacing • Interventricular septal perforation • His bundle pacing (HBP) • Cardiac resynchronization therapy • Left bundle branch area pacing (LBBAP) • Case report

ESC curriculum 5.9 Pacemakers • 5.11 Cardiac resynchronization therapy devices • 6.2 Heart failure with reduced ejection fraction

Learning points

- Intraprocedural septal perforation is a potential complication of left bundle branch area pacing (LBBAP) and pre-procedural imaging can help predict potential risk.
- Interventricular septal perforation can be diagnosed real-time via fluoroscopic images, contrast injection, or monitoring the electrogram at the tip of the lead.

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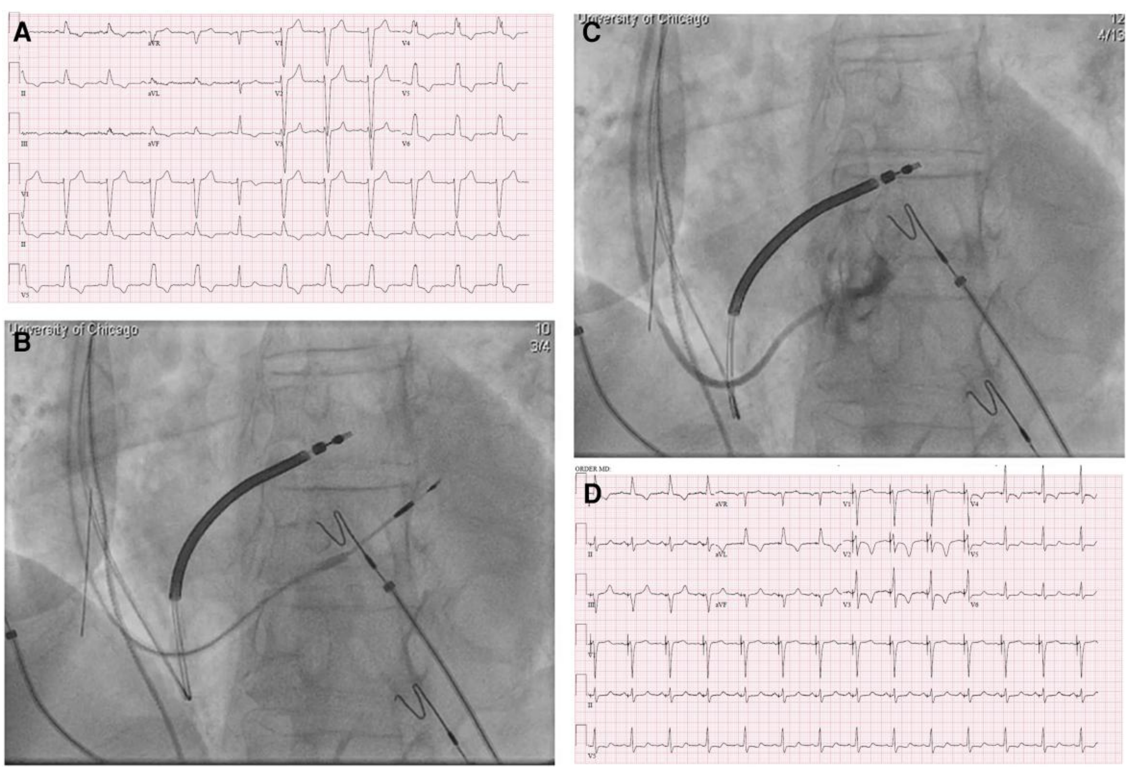
Introduction

In recent years, there has been a surge of interest in conduction system pacing (CSP) for routine clinical practice.[1] In particular, left bundle branch area pacing (LBBAP) offers potential advantages of shorter procedural times and more stable lead thresholds than His bundle pacing (HBP).[2] With that noted, complications associated with LBBAP are distinct from HBP or those encountered during traditional right ventricular lead implant. These include the risk of intraprocedural or post-operative septal perforation, cardiac troponin elevation from septal injury, and permanent right bundle branch injury, among others.[3] Unrecognized acute septal perforation may also be associated with a risk of loss of capture and increased risk of thromboembolism.[3,4,5] We present a case of a heart failure (HF) patient presenting for cardiac resynchronization therapy (CRT) in whom LBBAP was complicated with acute interventricular septal perforation and managed intra-procedurally with repositioning of the lead to provide HBP.

Summary figure



Panel A Baseline electrocardiogram showing sinus rhythm with wide QRS duration of 148 ms and left bundle branch block morphology. **Panel B** Fluoroscopic image of interventricular septal penetration during lead fixation showing lead migration to the left ventricle. **Panel C** Contrast injection during the procedure confirming interventricular communication with the left ventricle. **Panel D** Electrocardiogram following His bundle pacing lead implantation showing non-selective His capture with QRS correction (QRS width = 126 ms).

Case summary

A 77-year-old male with cardiac history of dilated cardiomyopathy with systolic dysfunction was referred for elective CRT device implantation by the HF Service. His medical history was notable for hypertension, hyperlipidaemia, stage 3 chronic kidney disease, and chronic obstructive pulmonary disease (COPD). The patient had undergone cardiac magnetic resonance imaging (MRI) in the year before referral which demonstrated severely depressed left ventricular ejection fraction (LVEF) of 14% with diffuse thinning of the myocardium and patchy, non-specific late gadolinium enhancement consistent with a non-ischemic aetiology. He demonstrated New York Heart Association (NYHA) class III symptoms and had been maintained on guideline-directed medical therapy (GDMT) for HF including the use of metoprolol succinate 50 mg daily, combination sacubitril/valsartan (49/51 mg) twice daily, spironolactone 25 mg daily, aspirin 81 mg daily, and furosemide 20 mg daily. His LVEF based on transthoracic echocardiography four months before implant was calculated to be 13%, and baseline electrocardiogram (ECG) was notable for left bundle branch block (LBBB) with QRS duration 148 ms (*Summary Figure, Panel A*). As was the practice at our centre, we discussed the options of traditional biventricular pacing and consideration for CSP in the event of unsuccessful LV lead placement, or primarily only if conduction block could be confirmed with electrophysiology study (EPS).[6] The patient opted for EPS to guide CSP in a shared-decision making approach.

At the time of device implant, cardiovascular examination showed regular heart rate and rhythm, with audible first and second heart sounds and no extra sounds. There were no signs of jugular vein distention or peripheral oedema, and the pulses were palpable with equal strength at the distal arteries. The patient did not appear to be in respiratory distress, and the lungs were clear upon auscultation. EPS was performed with multielectrode mapping of the left septum via retrograde aortic approach. The patient's LBBB was verified to be due to cardiac conduction block in the left conduction system where a left-sided His was recorded. Pacing at this location was associated with QRS correction and selective left bundle branch capture. We then pursued device implant. After uncomplicated right ventricular defibrillator lead placement in the right ventricular outflow tract, a 3830 SelectSecure lead (Medtronic, Minneapolis, MN) was used to target the LBBAP. During LBBAP placement, lead fixation was pursued with 4–6 turns. At this point, the lead was noted to penetrate into the left ventricle (*Summary Figure, Panel B*). Contrast injection confirmed interventricular communication (*Summary Figure, Panel C*). The lead was retracted and subsequently fixated at the His location with a final pacing threshold of 1.0 V at 0.4 ms and an impedance of 513 ohms and non-selective QRS correction with HBP (*Summary Figure, Panel D*). Postoperative postero-anterior and lateral chest radiography confirmed a stable location (Supplemental *Figure*).

The patient demonstrated intra and post-procedural hypotension which was attributed to deep sedation. His mean arterial pressure (MAP) was maintained above 70 mmHg on dopamine continuous infusion of 10 mcg/kg/min and he was admitted to the critical care unit (CCU) for haemodynamic monitoring and supportive treatment. His dopamine was weaned off overnight. Echocardiography on the day after implant was similar to the pre-procedural exam, with unchanged severe LV systolic dysfunction. There were no echocardiographic signs of persistent ventricular septal defect (VSD) on colour Doppler imaging or with the use of echocardiographic contrast. ECG after the procedure showed sinus rhythm with ventricular pacing with a non-selective HBP paced morphology with QRS correction (126 ms; *Summary Figure, Panel D*). He remained hemodynamically stable and was transferred to the medical floor after overnight monitoring for one additional day of observation. His GDMT was restarted with half of his home dose of metoprolol succinate (25 mg once daily) and sacubitril/valsartan (24/26 mg twice daily). He was discharged on his prior-to-arrival dosing 2 days post-implant.

Follow-up echocardiography in near-term follow-up (2 months) showed no VSD. Empagliflozin (10 mg once daily) was also started at the 3-month visit. The NYHA Class documented by the treating HF team improved from Class III to Class I at 6-month follow-up. The patient's N-terminal pro-brain natriuretic peptide was 2387 pg/mL at implant and 525 pg/mL at routine clinic visit 2 years post-implant. When compared to baseline obtained 4 months before implant, echocardiography at 32 months post-implant showed reduction in left ventricular volumes (end-systolic volume 182 to 124 mL; end-diastolic volume 210 to 177 mL) and LVEF had increased from 13% to 30% (see Supplementary material online, *Videos 1 and 2*). At the 3-year follow-up visit, corrective HBP threshold had increased to 1.75 V at 0.4 ms with impedance of 285 ohms, and the device output was increased to ensure QRS correction was present. Continued clinical follow-up and echocardiography are planned.

Discussion

We describe a case of a CSP procedure that was complicated with septal perforation. Acute intraprocedural septal perforation has been associated with LBBAP, with an incidence that varies between 0.65 and 14.1%.[1,3,4] Late septal perforations have also been described.[5,7] Unipolar impedance along with current of injury (COI) amplitude and unipolar ECG morphology have been used to identify septal perforation in addition to high pacing thresholds. An intraprocedural pacing impedance <450 Ω has been suggested as a cut-off with high sensitivity (100%) and specificity (96.4%) for septal perforation.[4] Rapid reduction of the COI amplitude immediately after perforation has also been reported. The COI at the lead tip may demonstrate 'negative' QS pattern during complete perforation or a 'biphasic' RS pattern with partial lead perforation.[4] In another study by Vijayraman et al., it was noted that acute perforation was associated with a decrease of impedance >200 Ω.[2] In this patient's case, migration of the lead through the interventricular channel was noted using the left anterior oblique projection on fluoroscopy and confirmed with contrast injection. Prompt recognition of changes in lead tip electrogram or fluoroscopic motion can help facilitate lead retrieval and reduce the risk of later complication.

Pre-operative evaluation can be beneficial in preventing lead-related complications. Diagnostic imaging such as echocardiography, previous cardiac computed tomography (CT), or MRI can provide useful information in assessing septum thickness and underlying fibrosis. In this patient's case, the prior cardiac MRI revealed that the interventricular thickness at the base of the heart was thin (∼5 mm) while it was within normal limits at the mid-chamber (14 mm). These findings are consistent with prior work on patients undergoing CRT which has shown that dyssynchrony is associated with nonhomogeneous left ventricle wall thickness, including a thinner septum and thicker lateral and posterior myocardial segments.[8] A more basal location is targeted during LBBAP, and caution should be taken in patients with thinning at this location. The lead tip-to-ring distance of the 3830 is 11 mm to the proximal surface of the ring electrode and may be used as fluoroscopic marker during lead delivery.

Management of septal perforation depends on the complexity of the communication. Acute septal perforation related to CSP devices is considered a relatively benign event.[1–4] Repositioning of the lead at a different site and close follow-up have been effective in the management of reported cases. A similar approach was taken in our patient, with lead repositioning to the His bundle and close monitoring. Repositioning to a more distal site for left ventricular septal pacing may also be considered. Septal perforation has been linked with local thrombosis and concern regarding the use of anticoagulation to prevent thromboembolic events.[5] Larger diameter stylet-driven leads may be associated with higher risk, although iatrogenic VSD from LBBAP requiring device-based or surgical closure have yet to be reported. Larger studies are needed to establish the benefit of current approaches and limit avoidable related complications.

Conclusion

Inadvertent septal perforation is a possible complication of CSP, which has been mainly associated with LBBAP pacing. In this case, the complication was benign with no long-term consequence, although future data are needed to characterize overall impact and management across a diversity of lead types.

Lead author biography

Jurgen Shtembari, MD, is completing his residency at Mount Sinai Hospital in Chicago. He earned his medical degree from Charles University in Prague. He will be pursuing speciality training Cardiology at the Carle Foundation Hospital. He is interested in cardiac arrhythmias and electrophysiology.

Supplementary material

Supplementary material is available at *European Heart Journal – Case Reports* online.

Consent: The authors certify that informed consent was obtained in accordance to the Committee on Publication Ethics (COPE) guidelines.

The patient has given consent for images and other clinical information to be reported in the journal.

Conflict of interest: J.S. and D.B.S. report no relevant disclosures. R.T. reports speaking or consulting for Abbott, Biotronik, Boston Scientific, and Medtronic. G.A.U. reports speaking or consulting for Abbott, Biotronik, Boston Scientific, GE Healthcare, Medtronic, Philips BioTel, Rhythm Science, and Zoll Medical.

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Data availability

The data underlying this article are available in the article and in the online Supplementary material.

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