Case Report

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Pacemaker leads and cardiac perforation

Andrew Peter Vanezis, Rachana Prasad and Richard Andrews
The Lincolnshire Heart Centre, Lincoln County Hospital, Lincoln LN2 5QY, UK
Corresponding author: Andrew Peter Vanezis. Email: andrew.vanezis@nhs.net

Lesson

This case series highlights the rare but potentially life threatening complication of ventricular perforation caused by pacemaker leads and discusses appropriate investigations and management strategies.

Keywords

arrhythmias, cardiovascular medicine, clinical, pacing and electrophysiology

The implantation rates of implantable cardiac devices such as permanent pacemakers, implantable cardiac defibrillators and cardiac resynchronisation therapy have steadily increased over the last two decades.[1] In 2013–2014, over 70,000 pacemakers and 10,000 implantable cardiac defibrillators were implanted in England.[2] In certain emergency situations, temporary pacing wire insertion continues to play a vital role in preserving cardiac output until there is either resolution of a reversible cause or implant of a permanent system.[3]

Complication rates of implanting such devices vary between 3% and 7.5%.[3–5] These variations are largely due to the specific device being inserted, the implanting centre, the venous access site, operator skill, patient cohort and the recording accuracy of complications. The most frequently occurring and potentially significant acute complications include pneumothorax (1.9%–3.7%), lead displacement (0.5%–4.8%) and myocardial perforation (0.37%–1%).[3–6] Myocardial perforation remains a rare but important complication of pacemaker insertion. Perforation can occur acutely (within 24 h after implantation), sub-acutely (between 24 h and one month after implantation) and chronically (occurring more than one month after implantation).[7] In this article, we report three cases of myocardial perforation as a consequence of pacing lead insertion highlighting the heterogeneity of the clinical presentation of this complication as well as the requirement for constant vigilance for the possibility of its occurrence.

Case 1

An 89-year-old woman was admitted with lethargy and syncope. She was in first-degree heart block with a rate of 32 beats per minute. Monitoring revealed periods of third-degree heart block which persisted despite cessation of bisoprolol 5 mg taken daily. A ventricular pacing, ventricular sensing, inhibition response, rate-adaptive pacemaker (VVIR) pacemaker was inserted via the left cephalic vein using an active fix lead. On deployment of the lead in the right ventricular apex, the patient immediately complained of severe back, neck and chest pain which was sharp in nature and accompanied by a sudden drop in blood pressure. Acute right ventricular perforation was suspected, and IV fluids and ephedrine were given. Bedside echocardiography revealed a small effusion with no signs of tamponade. The pacing lead was removed from the right ventricular apex and positioned at the right ventricular septum with good pacing parameters. Pericardiocentesis was not required, and the patient was managed conservatively and later discharged.

Case 2

An 89-year-old woman with a background of atrial fibrillation on warfarin was admitted with dyspnoea and nausea. She was found to be in complete heart block with a ventricular rate of 30 beats per minute. Due to haemodynamic compromise, a temporary pacing wire was inserted via the right femoral vein under fluoroscopic guidance into the right ventricular apex with good threshold. International normalised ratio was 3.6 at the time of the procedure. At the time of conversion to a permanent system, the international normalised ratio was 1.9; therefore, 10 mg of vitamin K was given 2 h prior to implant. A VVIR pacemaker with a passive lead in right ventricular apex was implanted. Very soon after temporary wire removal, the patient's blood pressure plummeted, and she became unresponsive. Suspected sub-acute perforation by the temporary pacemaker

wire causing pericardial effusion and cardiac tamponade was confirmed by bedside echocardiography. Emergency pericardiocentesis was performed, and a pericardial drain was sited. Blood, clotting products and inotropes were administered, and the patient was stabilised. Blood (1.3 l) was drained over two days after which the drain was removed with no re-accumulation, and the patient was later discharged.

Case 3

A 49-year-old man with dilated cardiomyopathy attended clinic with worsening dyspnoea on minimal exertion and signs of congestive cardiac failure. He underwent elective cardiac resynchronisation therapy-device implant. A Medtronic Attain Starfix® left ventricular lead was sited in a lateral branch of the coronary sinus. His symptoms improved markedly after the procedure; however, two years later, his exercise tolerance declined over the course of a few weeks. It was noted that the left ventricular lead was not capturing and increase of the lead output lead to phrenic nerve capture without biventricular pacing. There were no signs of lead fracture, crush or displacement with fluoroscopy. The patient was subsequently admitted to hospital with gradually worsening dyspnoea and a short history of pleuritic chest pain. A pericardial rub was evident, and echocardiography demonstrated a 1.5-cm circumferential pericardial effusion. Chronic perforation of the left ventricular lead through the roof of the coronary sinus was diagnosed. This lead was explanted percutaneously, and due to the patient's anatomy, an epicardial left ventricular lead was placed surgically. A few weeks after discharge, his level of function gradually returned to that experienced in the two years prior to left ventricular lead migration.

Discussion

Cardiac perforation can affect most parts of the heart that come in contact with a lead but the majority of perforations occur through the right ventricular apex, primarily because this remains a common site of deployment of the ventricular lead, and the myocardial wall is thinner here than at other common pacing sites such as the septum and right ventricular outflow tract.[8] Active fixation leads are associated with higher perforation rates than passive leads.[9] When using active leads, care must be taken to avoid overextension of the helix by following the manufacturer's guidance and by visualisation under fluoroscopy. Operators must also be vigilant of risk factors in their patients that may increase the likelihood of perforation including a low body mass index, age greater than 80 and the concomitant use of steroids.[10]

Pacemaker lead perforation may present in a number of circumstances and at different intervals after implantation. Lead perforation may present with chest pain, dyspnoea and/or symptoms associated with pericardial tamponade. The diagnosis of cardiac perforation should be considered in all patients in receipt of a pacemaker/complex device regardless of the time since insertion. Chest X-ray and echocardiography in some cases may allow for visualisation of the lead and a diagnosis of perforation, although this can often be challenging with plain X-rays. Echocardiography affords quantification of any pericardial effusion and tamponade. Where there is diagnostic doubt, computed tomogram may be used to aid diagnosis. In some cases, the patient may be asymptomatic and indeed incidental lead perforation detected by computed tomogram has been described in some studies without any adverse outcomes.[9] Tamponade necessitates emergency pericardiocentesis or surgical intervention, but in many cases, ventricular perforation can be managed conservatively by lead extraction and repositioning of a new lead in a different location.

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References

1. Bradshaw PJ, Stobie P, Knuiman MW, Briffa TG and Hobbs MST. Trends in the incidence and prevalence of cardiac pacemaker insertions in an ageing population. *Open Heart* 2014; 1: e000177.
2. NICOR. National audit of cardiac rhythm management devices, www.ucl.ac.uk/nicor/audits/cardiacrhythm/documents/annual-reports/CRM_National_Annual_Report_2013-14 (accessed 12 January 2017).
3. Link M, Mark Estes N III, Griffin J, et al. Complications of dual chamber pacemaker implantation in the elderly. *J Intervent Card Electrophysiol* 1998; 2: 175–179.
4. Aggarwal RK, Connelly DT, Ray SG, Ball J and Charles RG. Early complications of permanent pacemaker implantation: no difference between dual and single chamber systems. *Br Heart J* 1995; 73: 571–575.

5. Tobin K, Stewart J, Westveer D and Frumin H. Acute complications of permanent pacemaker implantation: their financial implication and relation to volume and operator experience. *Am J Cardiol* 2000; 85: 774–776.
6. Ellenbogen K, Hellkamp A, Wilkoff B, Camunãs J, Love J, Hadjis T, et al. Complications arising after implantation of DDD pacemakers: the MOST experience. *Am J Cardiol* 2003; 92: 740–741.
7. Laborderie J, Barandon L, Ploux S, Deplagne A, Mokrani B, Reuter S, et al. Management of subacute and delayed right ventricular perforation with a pacing or an implantable cardioverter-defibrillator lead. *Am J Cardiol* 2008; 102: 1352–1355.
8. Banaszewski M and Stępińska J. Right heart perforation by pacemaker leads. *Arch Med Sci* 2012; 8: 11–13.
9. Hirschl DA, Jain VR, Spindola-Franco H, Gross JN and Haramati LB. Prevalence and characterization of asymptomatic pacemaker and ICD lead perforation on CT. *Pacing Clin Electrophysiol* 2007; 30: 28–32.
10. Mahapatra S, Bybee KA, Bunch TJ, Espinosa RE, Sinak LJ, McGoon MD, et al. Incidence and predictors of cardiac perforation after permanent pacemaker placement. *Heart Rhythm* 2005; 2: 907–911.