

Delayed perforation of the right ventricle as a complication of permanent cardiac pacing – is following the guidelines always the right choice? Non-standard treatment – a case report and literature review

Późna perforacja prawej komory jako powikłanie stałej stymulacji serca – czy przestrzeganie wytycznych jest zawsze właściwym wyborem? Postępowanie niestandardowe – opis przypadku i przegląd piśmiennictwa

Anna Rydlewska¹, Barbara Małecka¹, Andrzej Ząbek¹, Piotr Klimeczek², Jacek Lelakowski¹, Mieczysław Pasowicz², Marek Czajkowski³, Andrzej Kutarski⁴

¹ Department of Electrophysiology, The John Paul II Hospital, Krakow

² Center for Diagnosis, Prevention and Telemedicine with One-Day Diagnostic Unit, The John Paul II Hospital, Krakow

³ Department of Cardiology, Medical University, Lublin

⁴ Department of Cardiosurgery, Medical University, Lublin

Abstract

A case of a delayed perforation of the right ventricle by the pacemaker lead in a 67-year-old woman is presented. Perforation, mimicking stenocardial symptoms, was incidentally diagnosed on a computed tomography chest scan. Percutaneous lead extraction was successfully performed, with simultaneous implantation of a new pacemaker lead.

Key words: permanent heart stimulation, complication, delayed perforation of the right ventricle

Kardiologia Polska 2010; 68: 357-361

Introduction

Heart perforation is a rare complication of pacemaker implantation. Acute, subacute and delayed lead perforations have been defined. Acute lead perforation occurs within 24 h after implantation and is considered to be associated with more severe clinical presentation: heart tamponade or even death.

Subacute and delayed perforations are defined in contrast to acute perforation; to diagnose them normal chest X-ray and electrical parameters within 24 h after implantation are required [1]. Subacute and delayed perforations are differentiated according to whether the time of their presentation is over one month after implantation. Incidence of delayed lead perforation has been estimated by Khan et al. at 0.1-0.8% for pacemaker implantation and 0.6-5.2% in ICDs [2].

Probably the lower incidence and more benign clinical presentation in delayed perforations is associated with 'self-sealing' properties of the ventricle wall: by fibrosis, muscle contraction or by the lead itself [3, 4].

Risk factors of lead perforation have not been defined yet. The type of lead used might influence the incidence of perforation. It occurs more often in the case of the following leads:

1. for temporary stimulation,
2. atrial,
3. with active fixation system,
4. defibrillator leads, with double spirals (more wires, stiffer),
5. when excessive length during implantation is left,
6. small diameter (increased force per unit area),
7. so-called high resistance (small tip surface).

Address for correspondence:

Andrzej Ząbek MD, Oddział Kliniczny Elektrokardiologii, Krakowski Szpital Specjalistyczny im. Jana Pawła II, ul. Prądnicka 80, 31-202 Kraków, tel.: +48 12 614 20 00, e-mail: andrzej_j_z@poczta.onet.pl

There have been many reports of a higher perforation rate for certain lead types (e.g. Riata ST Jude Medical leads), which might indicate that certain constructive properties of these leads are associated with increased perforation risk, but it has not been proved in any larger trial [1, 5-8].

Also the lead tip location is a factor. Perforations occur more frequently in the apical location, as compared with septal position or in the right ventricular outflow tract, which is probably due to the thinner muscle in the right ventricle apex [1, 2, 6, 9-11].

Apart from the lead type and its location, also the heart muscle itself may favour perforation. There has been a case report on perforation in a patient with congenital cardiomyopathy [12]. Anticoagulation therapy and steroid use within 7 days of implantation may also have a negative impact. The elderly, women, and patients with low body mass (BMI < 20 kg/m²) are also prone to perforation [6, 9].

In the case of defibrillator leads, the perforation rate rises with the number of shocks delivered [5]. Chest trauma, especially soon after implantation, may also indicate a necessity to exclude perforation [5, 10].

Subacute and delayed right ventricle perforation may have various clinical presentations, from incidentally diagnosed asymptomatic perforation to death [1-16, 18, 19]. The following clinical symptoms of lead perforation have been described:

1. chest pain,
2. dyspnoea,
3. syncope (due to improper stimulation or its complete failure),
4. inadequate ICD shocks,
5. muscle or diaphragm stimulation,
6. abdominal pain (due to diaphragm stimulation or lead migration to the peritoneal cavity),
7. hiccup (as a symptom of phrenic nerve stimulation),
8. mammary haematoma,
9. consequences of diaphragm, lung, chest wall perforation,
10. pleural or pericardial effusion, rarely demanding drainage.

The most common symptom described in the literature has been the failure of the leads to pace or sense appropriately [1-3, 5-16, 18, 19]. Decreased lead impedance and R-wave amplitude, as well as increased pacing threshold (or even failure to pace), are the most widely described observations. However, there have been some reports on delayed perforations with normal electrophysiological parameters [4]. Therefore, a conclusion may be made that whereas improper pacemaker function may indicate perforation, its normal function does not exclude it.

Visualisation is an important stage in perforation diagnosis. The key tests are: chest X-ray, transthoracic (TTE) or transoesophageal (TEE) echocardiography and computed tomography. The X-ray may show the lead's migration outside the heart: into the pleural cavity, peritoneal cavity

or to the chest wall. It also allows assessment of pleural effusion. Perforation is suspected when less than 3 mm separate the lead tip from the epicardial fat [13].

Echocardiography may show lead presence in the pericardial sac or pericardial effusion. However, it is often false negative, showing no pericardial effusion, or no evidence of the lead's migration, while the perforation is confirmed in other tests [1, 2, 12-15].

Computed tomography (CT) in the perforation diagnosis seems to play a crucial role, becoming a golden standard in its visualisation [1, 5, 6, 14, 15]. Cases of asymptomatic perforations identified on CT scans, performed for other medical indications, are described in the literature. Perforation rates were 15% for atrial leads and 6% for ventricular leads. Small pericardial effusion in patients with and without perforation have been present in 20 and 19% respectively [4].

Case report

A 67-year-old woman with coronary heart disease risk factors – overweight, smoking cigarettes, with arterial hypertension and hyperlipidaemia – had a CT scan of the coronary arteries performed due to uncharacteristic chest pain.

Three years before she had undergone atrioventricular pacemaker implantation [pacemaker DDDR-Vitatron C60DR, atrial lead Biotronic Selox JT 53 implanted in the auricle of the right atrium and ventricular lead with active fixation system Vitatron Cristalline Actifix ICF 09B implanted on the anterior wall of the right ventricular outflow tract (RVOT)] because of sick sinus syndrome with atrioventricular block. During implantation normal sensing and pacing parameters were achieved, and post-implantation X-ray showed normal lead position.

After the implantation, the patient complained of persistent, uncharacteristic chest pain. The pain was stabbing, localised in the apex and retrosternal region, not associated with physical exercise nor body position.

Regular telemetric control (every 6 months) showed normal pacemaker function. Considering the uncharacteristic symptoms and cardiovascular risk factors, coronary heart disease diagnostics was performed. The TTE performed in April 2009 showed no evident pathologies, and, significantly, no pericardial effusion.

Angio-CT of the coronary arteries performed in August 2009 showed the lead tip outside the right ventricle, in the ventricle wall and partially in the pericardial sack (Figure 1).

The patient was admitted to hospital and further diagnosis was performed. On admission, the patient was haemodynamically stable. The pacemaker control presented normal sensing and pacing parameters. Chest X-ray showed no dislocation of the leads. The TTE revealed pericardial effusion up to 24 mm behind the right ventricle, the inferior and posterior wall, with mobile localisation of the lead tip, with no evident penetration to the pericardium and with the

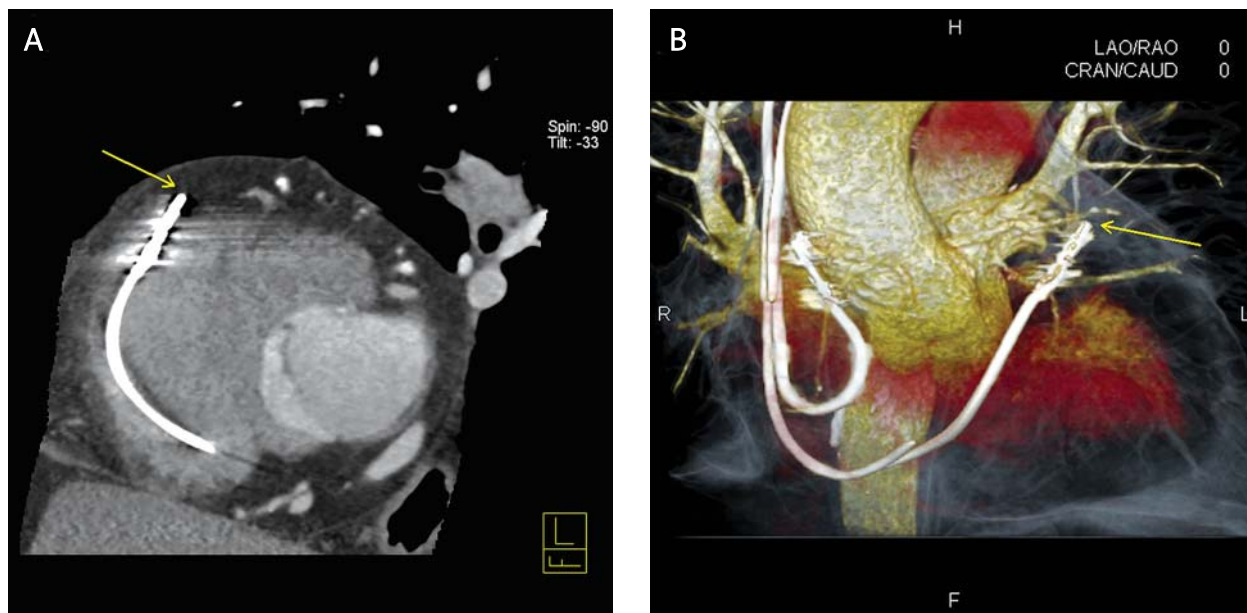


Figure 1. Picture of the heart in CT. **A** – visible perforation by ventricular lead to the pericardium (arrow); **B** – volume digital reconstruction: visible 2 leads of atrioventricular stimulation system. Ventricular lead is in the RVOT. Lead’s tip is visible outside the outline of the heart in the pericardium (arrow)

blood flow between the right ventricle and the pericardium visualised by the colour Doppler spectrum (Figure 2).

During the following days of hospitalisation, the patient was monitored echocardiographically: slight regression in the quantity of pericardial effusion was noted (to 16 mm behind the right ventricle and the inferior wall) and signs of effusion organisation were observed. Telemetric control of the pacemaker showed its normal function.

Considering the overall clinical picture and the available literature, it was decided to perform percutaneous lead extraction with cardiosurgical backup and with simultaneous new lead implantation.

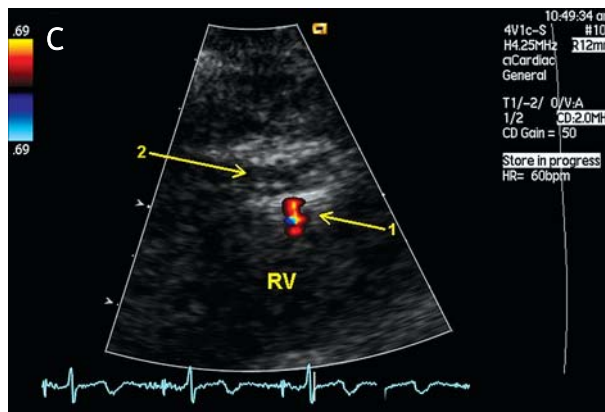
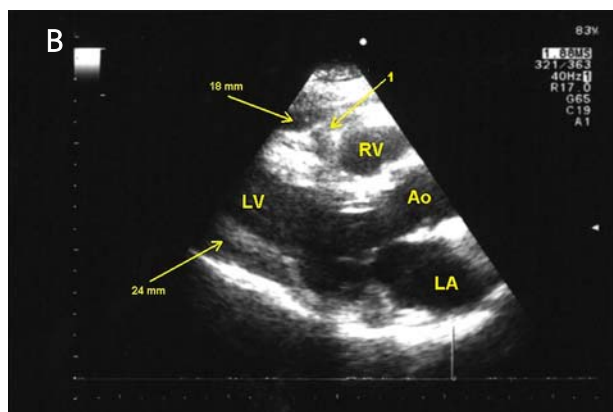
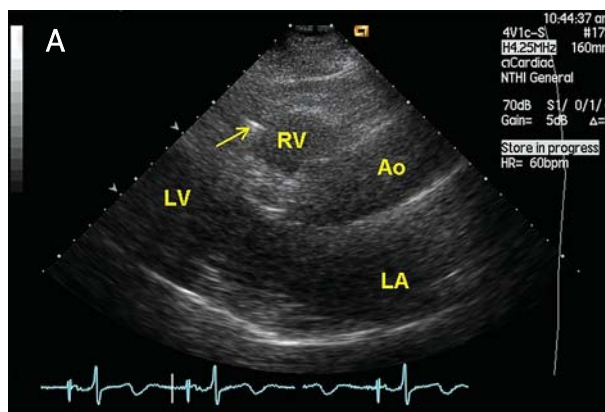


Figure 2. TTE before the removal procedure: **A** – visible lead in the right ventricle with probable perforation to the pericardium (arrow); **B** – liquid in the pericardium up to 24 mm behind the left ventricular posterior wall and up to 18 mm before the right ventricular anterior wall (arrows). Arrow 1 – pacemaker lead; **C** – blood flow between liquid space in the pericardium (arrow 2) and right ventricle lumen in Doppler examination (arrow 1)

Ao – aorta, LA – left atrium, LV – left ventricle, RV – right ventricle

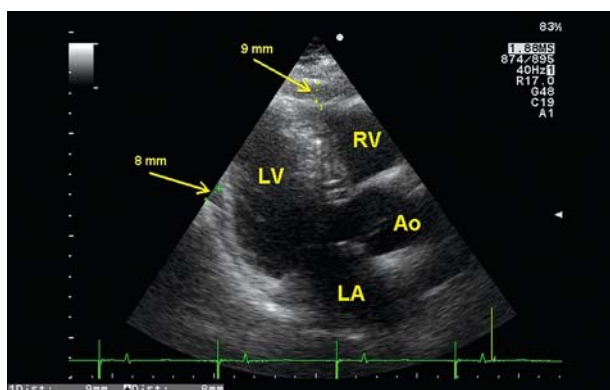


Figure 3. TTE after the removal procedure: visible liquid in the pericardium up to 8 mm behind the posterior wall and up to 9 mm before the anterior wall (arrows)

Ao – aorta, LA – left atrium, LV – left ventricle, RV – right ventricle

The patient was transported to the centre specializing in percutaneous lead extraction. In the operating room, with basic live function monitoring, with anaesthesiological, cardiosurgical and echocardiographic backup, right ventricle lead percutaneous extraction was performed.

The procedure was performed through the left subclavian vein, using green polypropylene Byrd dilator sheaths (10.0/12.1 F), to separate the lead from the adhesions to the vessels and heart. In order to stiffen the lead during its extraction, a stylet without anchoring function was used. The lead was removed without complications, and no symptoms of tamponade were observed during or after the procedure. Simultaneously, a new active fixation lead was implanted in the middle part of the ventricular septum. The patient was discharged in a good general condition. In the follow-up, the symptoms began to retreat. In the TTE performed 2 weeks after the procedure, small effusion was seen (up to 9 mm behind the right ventricle, the inferior and posterior wall) (Figure 3).

Discussion

Most authors agree that the best method of lead perforation treatment is percutaneous extraction in the operating room with TTE and TEE monitoring during and/or after the procedure, with the cardiosurgical and anaesthesiological team ready to intervene, even if such a necessity is rare [1, 2, 8, 14, 16, 19]. Such practice is inconsistent with the HRS expert consensus (2009), which classifies percutaneous lead extraction as class III indication, level of evidence C [17]. Percutaneous extraction of the perforating lead, as not requiring general anaesthesia, has been performed in case of a very high operation risk [12]. Described cases of open surgery lead removal include suspected digestive tract perforation [1], patients with high risk of tamponade during operation and

with atypical location of the perforating lead [3, 6, 9-11, 13, 15, 18]. In most of these cases, a new pacemaker was implanted, placing the lead tip in a different location than before (that is in the RVOT if the tip had been placed in the apex, and vice versa). In the case of open surgery, epicardial leads have been used [1, 6]. In one case, a new pacemaker was not implanted at the patient's demand [10]. There has also been one case in which the perforating atrial lead was not removed, and the DDD mode was switched to the VVI mode, due to the high operation risk [14]. Hirschl et al. indicate that removing delayed asymptomatic lead perforations with normal pacemaker function is disputable [4]. In the postoperative period, echocardiographic monitoring is necessary, as there have been cases of cardiac tamponade described [1].

The case presented above is atypical considering the literature, mainly due to its very late presentation (3 years after implantation). It is worth underlining that there was no evident cause of the perforation (e.g. trauma) in the anamnesis. It also seems as if we have captured the moment of the full lead perforation with effusion formation. During the observation, we noted slight regression in the effusion quantity and its organisation. Another rare symptom was normal pacemaker electrophysiological parameters.

Conclusions

Lead perforation of the heart remains a rare, but genuine threat. Sometimes it creates a real diagnostic puzzle and has confusing clinical presentation. Normal pacemaker electrophysiological function does not exclude lead perforation. Optimal treatment for such a condition seems to be percutaneous extraction of the perforating lead with cardiosurgical backup, though this opinion is inconsistent with the HRS expert consensus.

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