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Transvenous extraction of an eight-year-old ventricular lead accidentally implanted into the left ventricle

Przezżylna ekstrakcja 8-letniej elektrody komorowej omyłkowo implantowanej do lewej komory

Andrzej Kutarski[1], Radosław Pietura[2], Andrzej Tomaszewski[1], Marek Czajkowski[3], Krzysztof Boczar[1]

[1]Department of Cardiology, Medical University of Lublin, Poland
[2]Department of Interventional Radiology and Neuroradiology, Medical University of Lublin, Poland
[3]Department of Cardiac Surgery, Medical University of Lublin, Poland

Abstract

Incorrect implantation of a ventricular pacemaker (PM) lead into the left ventricle (LV) is a known problem associated with permanent pacing. The optimal management of such cases identified late has not been clearly established. Generally acceptable management options are: open-chest cardiac surgery using cardio-pulmonary bypass, chronic anticoagulation and antiplatelet-drugs therapy. Rarely, the problem is solved by percutaneous LV lead extraction. We present a case of a patient with DDD pacing and ventricular lead implanted incorrectly into the LV apex region via an atrial septal defect eight years ago. Chronic PM pocket infection developed after replacement of the device. Both leads were extracted percutaneously, and the embolic protection system (Filter-Wire EZ, Boston Scientific) was used to reduce cerebral circulation embolism. The hardest connective tissue adhesions affecting the lead and the anodal ring were found in the LV. Less dense surrounding fibrous tissue around the lead was present at all levels of the venous course of the lead and in the right atrium. Very small fragments of apparently connective tissue remnants were found in cerebral circulation protection filters, and had been removed after the procedure. We conclude that old, permanently implanted LV leads may be extracted percutaneously, especially when there is an increased risk of cardiac surgery, or where the patient's consent for surgical treatment is lacking. In order to perform the procedure it is recommended to establish a cerebral protection system and intraoperative transoesophageal echocardiography which are mandatory for successful lead removal.

Key words: endocardial left ventricular lead extraction, incorrect left ventricular pacing, unintentional left ventricular lead location

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INTRODUCTION

Incorrect left ventricular (LV) pacemaker (PM) lead implantation is a rare but major complication of permanent pacing system implantation [1–5] with an unknown incidence. The analysis of case reports [6–23] shows that patients in whom this complication is identified late are usually treated with chronic anticoagulation or antiplatelet therapy [6–13]. The presence of an embolic complication indicates the need to perform open-chest cardiac surgery [14–18]. Present guidelines do not clearly recommend percutaneous extraction of the leads implanted this way (class 3 recommendation) [5]. Nevertheless, there is a series of reports in the literature of percutaneous LV lead extraction [2, 3, 14, 19–23]. In our cardiac centre specialising in lead extraction, we have reported extraction of chronically implanted LV leads in three patients who underwent percutaneous lead extraction in the past without any complications. We present here the case of a patient undergoing removal of a strongly ingrown eight-year-old, endocardial LV lead due to severe PM pocket infection.

Address for correspondence:
Krzysztof Boczar, MD, Department of Cardiology, Medical University of Lublin, ul. Jaczewskiego 8, 20–954 Lublin, Poland, fax: +48 81 742 87 47, e-mail: krzysiek.boczar@gmail.com

CASE REPORT

A 46-year-old Caucasian male patient with brady-tachycardia syndrome and a presentation of sinus bradycardia with occasional, atypical atrial flutter episodes had received the DDD pacing system with a passive, bipolar atrial lead (Biotronik SX 53JBP) positioned in the right atrial appendage (RAA) and a passive, bipolar ventricular lead (Biotronik TIR 60 BP) in the right ventricle (RV) apex region eight years earlier. A conventional postero-anterior X-ray image did not raise any suspicion (Fig. 1A), but the morphology of QRS complexes in the ECG derived from the limb leads (a PM follow-up) was typical for LV pacing.

A large number of ECG examinations were performed but ECGs with atrial arrhythmia as well as atrial pacing did not reveal morphology due to normal atrio-ventricular (AV) conduction and pacing settings preferential for native AV conduction. The patient was asymptomatic as a result of anticoagulation treatment during the first year after PM implantation and, consequently, owing to a lack of stroke risk factors. He also received an antiarrhythmic drug in case of arrhythmia appearance (the 'pill in a pocket' option) only. The patient presented no stroke or other embolic episodes. The last hospitalisation took place one year earlier for routine PM unit replacement. Symptoms of local pocket infection appeared several months after the procedure. The patient attended his regional PM centre for follow-up. He presented a large, opened fistula, with partially protruding unit (Fig. 1B). A diagnostic swab collected from the fistula revealed a strain of methicillin-sensitive *Staphyloccus aureus*. As a result of an easy pus outflow from the infected PM pocket, there were no symptoms of systemic infection: C-reactive protein (CRP) 2 mg/L, no fever, and results of a full set of laboratory tests were within a reference range. Echocardiographic examination (including a transthoracic [TTE] and a transoesophageal [TEE] one) was performed in the local PM centre. Searching for vegetation, an atypical course of the ventricular lead was revealed. The lead passed through the foramen ovale and the left atrium (LA) into the LV apex.

After receiving informed consent for lead extraction, the patient was referred to our centre. On admission, the patient's general condition was very good. ECG presented normal sinus rhythm and typical picture of LV pacing following device reprogramming with short AV-delay setting. The first laboratory test results were: white blood cells 6,190/mm^3, CRP 0.910 mg/L. There were no signs of systemic infection. Further test results were also within the normal range (Fig. 1)

TTE and TEE were performed on admission, and they showed normal RAA lead location and a lack of the ventricular lead in the RV. The presence of an atrial septal defect (ASD) was diagnosed (on the basis of a colour Doppler examination), the ventricular lead passed through an ASD into the LA, crossed the mitral valve, and reached the LV. Signs of possible vegetation or thrombus were not found, although the distal part of the ventricular lead seemed to be thickened. The presence of ECHO-positive blood in the LA was not observed (Fig. 2).

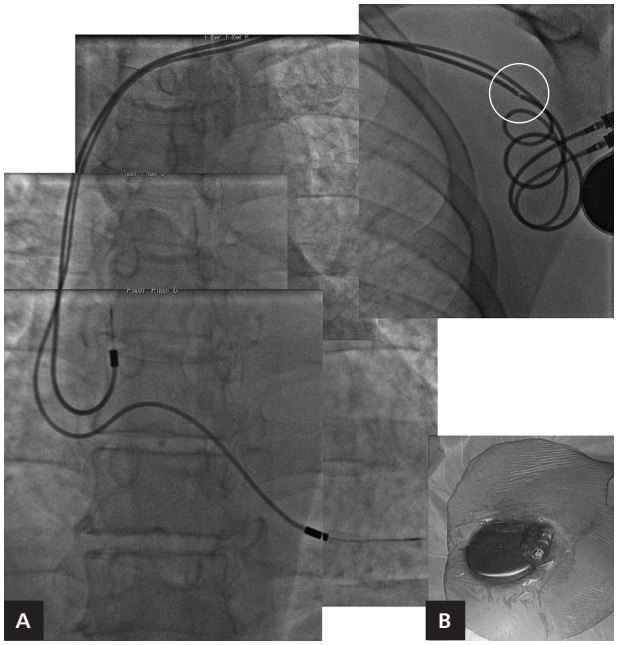

Figure 1. Postero-anterior X-ray view (**A**) and pacemaker pocket region conditions (**B**)

The patient was transferred to the operating room where a control fluoroscopy was repeated. The picture might have not caused any suspicions without ECHO findings; the position of the tricuspid valve on the basis of the course of the ventricular lead might have been considered slightly higher than usually observed (Fig. 1A).

It was decided to remove the system after a consultation process involving the invasive electrocardiologist, cardiac surgeon, interventional radiologist and echocardiographer. The patient accepted a proposal for percutaneous lead extraction. Temporary protection of the cerebral circulation was established, due to the high risk of stroke during this type of procedure.

Temporary cerebral circulation protection was performed via the femoral approach using 6 Fr guiding sheaths. After the baseline angiography was performed, the sheaths were advanced into both common carotid arteries. The Filter-Wirer EZ (Boston Scientific) was positioned in the distal straight segment of the extracranial parts of internal carotid arteries to capture the masses of potentially embolic material that might be released. Apart from the co-operation with an experienced interventional radiologist, complete cardiosurgical back-up was present on site. The patient received one quarter of the standard dose of intravenous heparin, and a regular dose of heparin solution was used to clean the operating tools. The filter catheters passing through the aorta and jugular arteries are seen on Figures 3–6.

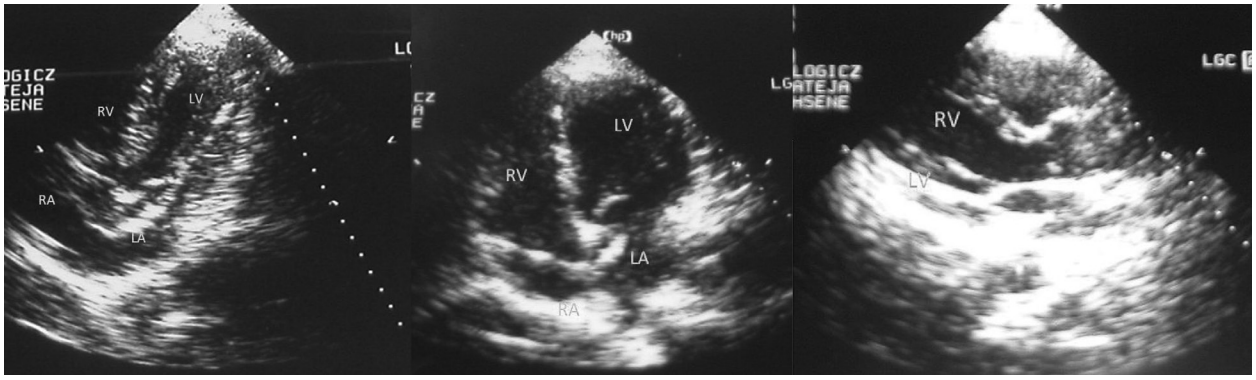

Figure 2. Transthoracic echocardiography before LV lead extraction; LA — left atrium; RA — right atrium; LV — left ventricle; RV — right ventricle

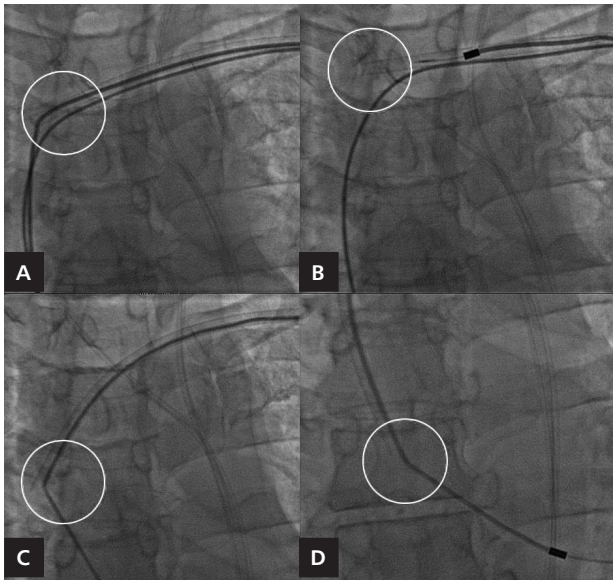

Figure 3. Right side lead extraction. Atrial lead extraction (**A**, **B**). A vascular and right heart part of the ventricular lead flotation (**C, D**)

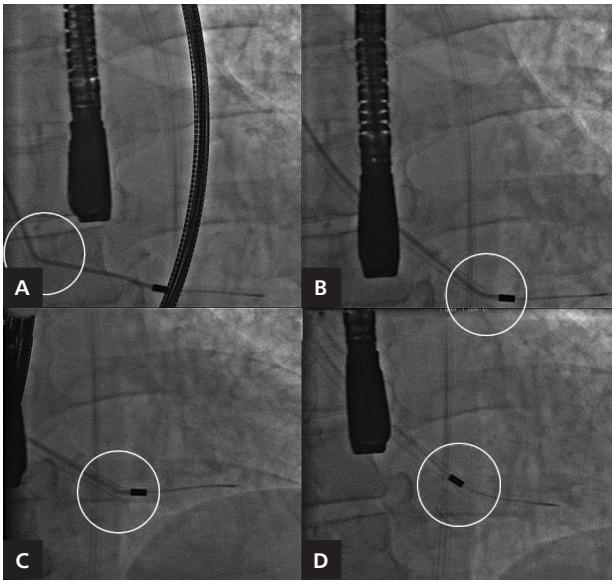

Figure 4. **A–D.** Left side lead extraction. Step by step, the distal part of the lead was excised

The next step was the PM removal and evacuation of the abscessed pocket remnants. Lead connectors were cut off and standard stylets were introduced into both leads. When the leads became mobile at their entrance into the venous system, an ECHO oesophageal tube was inserted and the expansion of jugular filters was checked (Fig. 3).

Both leads were extracted with the use of mechanical systems (polypropylene 11.5 F Byrd dilators (white) and pin-vise Cook). The right atrial lead was successfully extracted initially (Figs. 1A, B). Subsequently, we started to extract the ventricular lead. As it was necessary to rotate along two angulations, we selected and used only the internal sheath from the pair of telescopic Byrd dilators. We observed mid-strong connective tissue adhesions on the venous and on the right heart lead course (Fig. 4).

We observed a very strong connective tissue adhesion surrounding the part of the lead localised in the left heart. The strongest and most challenging connective tissue bridges had developed mainly near the anodal ring of the lead. This was the reason for the prolonged extraction with the use of a Byrd dilator in the left heart. An additional problem was associated with high intra LV pressure, which resulted in a consistent tendency for bleeding via the Byrd dilator sheath. The whole procedure was guided and monitored by TEE (Fig. 5).

Finally, the tip of the lead was released and the lead was removed via the Byrd sheath. The wound was closed and active drainage was applied for heparin-related haematoma prevention. Control TEE presented no fluid in the pericardium and a small connective-tissue 'sheet' flopping in the region previously occupied by the distal part of the LV lead. After

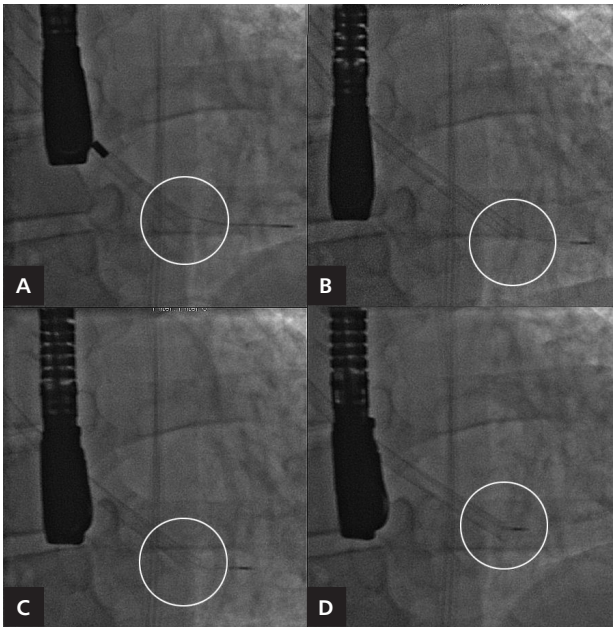

Figure 5. **A–D.** Left side lead extraction. The most dramatic moment. The threat of lead abruption emerged because it was strongly ingrown in the left ventricular endocardium

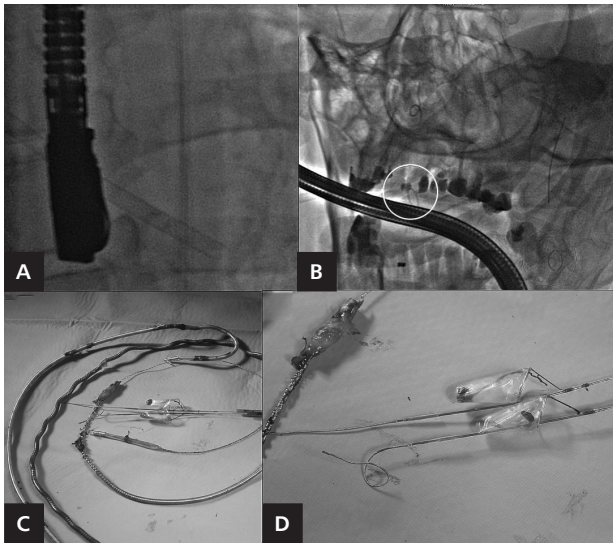

Figure 6. Landscape after the battle. The left ventricular lead removed (**A**). The Filter-Wirer EZ, Boston Scientific (**B**) was removed a few minutes later. The picture presented at the operating table (**C**, **D**). The left ventricular lead in a very poor condition, mostly destroyed during the final part of its extraction. Inside the filters there are very small fragments of white-coloured tissue and some blood clots

a subsequent 10-min observation, the protection device was closed using the retrieval sheath and removed. A small volume of embolic material was found (Fig. 6).

TEE performed the next day brought similar findings. ECG Holter monitoring presented the normal sinus rhythm with tendency for bradycardia. The patient received anticoagulation, and two days after the procedure was discharged in a good condition with recommendations to perform a control examination in an outpatient clinic and verify indications for permanent pacing. Finally, the patient was referred for an ablation procedure of the arrhythmia substrate.

DISCUSSION

Since the beginning of permanent cardiac pacing, there have been various procedural complications, such as incorrect implantation of the ventricular lead tip into the middle cardiac vein or into the LV chamber via an atrial septal defect [3, 8, 9, 11, 12, 18] or foramen ovale [3, 10, 12–14, 16, 20]. Extremely rare cases of implantation via the subclavian artery, aorta and aortic valve have also been observed [7, 17, 19]. Furthermore, cases of implantation of the ventricular lead through the interatrial or AV septum perforation also exist in the literature [2, 15]. Incorrect LV pacing from the middle cardiac vein can be undiagnosed, but entails a significant risk of increasing the ventricular pacing threshold and a sudden loss of pacing. On the other hand, unrecognised LV intracardiac permanent pacing, in spite of its known haemodynamic advantages, carries a high risk of systemic embolism, mainly recurrent cerebral strokes [1–4, 6, 17, 19]. One can presume that chronic anticoagulation reduces the risk of systemic embolisation events, because long-term asymptomatic outcomes in patients receiving anticoagulation or antiplatelet drugs have been reported [4, 8–11, 13, 15, 16, 18, 20, 21, 23]. There are no unequivocal recommendations for the management of such patients in cases of severe infective complications such as chronic pocket infection or lead dependent infective endocarditis. The main therapeutic option remains to remove the lead during open-chest cardiac surgery, using cardio-pulmonary bypass; this type of surgery is difficult to perform because a ventricular lead can be strongly ingrown into the endocardium of the LV wall (conventional passive bipolar leads are the most difficult to extract). Moreover, it is very difficult to dissect connective tissue enclosures along the course of a lead in the right atrium and in the venous system. According to the 2009 Heart Rhythm Society statement, left heart endocardial lead extraction is a class 3 recommendation. This means that "percutaneous lead extraction is not indicated in such patients and additional techniques, including surgical back-up, may be used if the clinical scenario is compelling." [5]. We decided that when all possible safety precautions had been complied with, the procedure may be less invasive to a patient than open-chest cardiac surgery.

We found a very small volume of potentially embolic material in removed jugular filters after the procedure. The material macroscopically resembled connective tissue strands

with small blood clots. The routine usage of cerebral circulation protection systems (as described above) during percutaneously implanted LV lead extraction is recommended. A prolonged dissection process with the Byrd dilator sheaths in the LV was extremely confusing and stressful, due to very strong connective tissue bindings along the course of the lead in the LV. Connective tissue adhesions enclosing the lead at the level of the right heart and along the course of the lead in the venous system were less resistant for dissection, and so we managed to complete this stage of the procedure quickly and without complications. Unfortunately, during the subsequent stage of the procedure it was not possible to apply our knowledge and experience gained during coronary sinus LV lead extraction to the intracardiac LV lead extraction [24, 25].

On the basis of our experience in lead extraction, all measures should be taken to complete the procedure without complications. Selection of the management option for patients with a PM lead implanted accidentally in the LV should account for the phenomenon of progressive lead ingrowth, doubling the risk of lead extraction every three years of lead presence in the heart and a small but significant chance of system infection during unit replacement.

CONCLUSIONS

It is possible, in selected cases, that incorrectly long-term LV lead implants can be extracted percutaneously, especially in patients with an increased risk of an open-chest cardiac surgery or in cases where there is no patient consent for such a procedure. The use of a cerebral circulation protection system and co-operation with a cardiac surgeon and an experienced interventional radiologist during the procedure are essential measures for the safety of the method.

Conflict of interest: *none declared*

References

1. Sharifi M, Sorkin R, Sharifi V, Lakier JB. Inadvertent malposition of a transvenous-inserted pacing lead in the left ventricular chamber. Am J Cardiol, 1995; 76: 92–95.
2. Agnelli D, Ferrari A, Saltafossi D, Falcone C. A cardiac embolic stroke due to malposition of the pacemaker lead in the left ventricle. A case report. Ital Heart J Suppl, 2000; 1: 122–125.
3. Van Gelder BM, Bracke FA, Oto A et al. Diagnosis and management of inadvertently placed pacing and ICD leads in the left ventricle: a multicenter experience and review of the literature. Pacing Clin Electrophysiol, 2000; 23: 877–883.
4. Ghani M, Thakur RK, Boughner D et al. Malposition of transvenous pacing lead in the left ventricle. Pacing Clin Electrophysiol, 1993; 16: 1800–1807.
5. Wilkoff BL, Love CJ, Byrd CL et al. Heart Rhythm Society; American Heart Association. Transvenous lead extraction: Heart Rhythm Society expert consensus on facilities, training, indications, and patient management: this document was endorsed by the American Heart Association (AHA). Heart Rhythm, 2009; 6: 1085–1104.
6. Ergun K, Tufekcioglu O, Karabal O et al. An unusual cause of stroke in a patient with permanent transvenous pacemaker. Jpn Heart J, 2004; 45: 873–875.
7. Paravolidakis KE, Hamodraka ES, Kolettis TM et al. Management of inadvertent left ventricular permanent pacing. J Interv Card Electrophysiol, 2004; 10: 237–240.
8. Altun A, Akdemir O, Erdogan O et al. Left ventricular pacemaker lead insertion through the foramen ovale: a case report. Angiology, 2002; 53: 609–611.
9. Vanhercke D, Heytens W, Verloove H. Eight years of left ventricle pacing due to inadvertent malposition of a transvenous pacemaker lead in the left ventricle. Eur J Echocardiogr, 2008; 9: 825–827.
10. Kumar S, Ghosh P, Ghosh AK et al. Inadvertent transvenous left ventricular pacing through an unsuspected atrial septal defect. J Indian Med Assoc, 2006; 104: 522–524.
11. Engström A, Holmberg B, Månsson A, Carlsson J. Inadvertent malposition of a transvenous pacing lead in the left ventricle. Herzschrittmacherther Elektrophysiol, 2006; 17: 221–224.
12. Ciolli A, Trambaiolo P, Lo Sardo G et al. Asymptomatic malposition of a pacing lead in the left ventricle: the case of a woman untreated with anticoagulant therapy for eight years. Ital Heart J, 2003; 4: 562–564.
13. Kusniec J, Mazur A, Hirsch R, Strasberg B. Left ventricular malposition of a transvenous cardioverter defibrillator lead: a 3-year follow-up. Pacing Clin Electrophysiol, 1998; 21: 1313–1315.
14. Konings TC, Koolbergen DR, Bouma BJ et al. Iatrogenic perforation of the posterior mitral valve leaflet: a rare complication of pacemaker lead placement. J Am Soc Echocardiogr, 2008; 21: 512.
15. Ergun K, Cagli K, Sahin O et al. Atrioventricular membrane perforation: a very rare complication of transvenous pacemaker implantation. J Am Soc Echocardiogr, 2005; 18: 71–74.
16. Seki H, Fukui T, Shimokawa T et al. Malpositioning of a pacemaker lead to the left ventricle accompanied by posterior mitral leaflet injury. Interact Cardiovasc Thorac Surg.,2009; 8: 235–237.
17. Liebold A, Aebert H, Muscholl M, Birnbaum DE. Cerebral embolism due to left ventricular pacemaker lead: removal with cardiopulmonary bypass. Pacing Clin Electrophysiol, 1994; 17: 2353–2355.
18. Splittgerber FH, Ulbricht LJ, Reifschneider HJ et al. Left ventricular malposition of a transvenous cardioverter defibrillator lead: a case report. Pacing Clin Electrophysiol, 1993; 16: 1066–1069.
19. Reising S, Safford R, Castello R et al. A stroke of bad luck: left ventricular pacemaker malposition. J Am Soc Echocardiogr, 2007; 20: 1316.
20. de Cock CC, van Campen CM, Kamp O, Visser CA. Successful percutaneous extraction of an inadvertently placed left ventricular pacing lead. Europace, 2003; 5: 195–197.
21. Nägele H, Schneider JC, Knobelsdorff GV et al. Excimer laser-assisted extraction of an infected bipolar left ventricular pacing lead implanted 10 years ago. Pacing Clin Electrophysiol, 2001; 24: 388–390.
22. Orlov MV, Messenger JC, Tobias S et al. Transesophageal echocardiographic visualization of left ventricular malpositioned pacemaker electrodes: implications for lead extraction procedures. Pacing Clin Electrophysiol, 1999; 22: 1407–1409.
23. Trohman RG, Wilkoff BL, Byrne T, Cook S. Successful percutaneous extraction of a chronic left ventricular pacing lead. Pacing Clin Electrophysiol, 1991; 14: 1448–1451.
24. Wilkoff BL, Belott PH, Love CJ et al. Improved extraction of ePTFE and medical adhesive modified defibrillation leads from the coronary sinus and great cardiac vein. Pacing Clin Electrophysiol, 2005; 28: 205–211.
25. Bulava A, Lukl J. Postmortem anatomy of the coronary sinus pacing lead. Pacing Clin Electrophysiol, 2005; 28: 736–739.