ESC European Society of Cardiology

European Heart Journal - Case Reports (2018) **2**, 1–5
doi:10.1093/ehjcr/yty127

CASE REPORT

Unusual presence of 'ghosts' following lead extraction for recurrent reactive pericarditis: a case report

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Received 6 May 2018; accepted 18 October 2018; online publish-ahead-of-print 26 November 2018

Background The presence of a persistent fibrous sheath in right-sided heart chambers after transvenous lead extraction has already been described in some studies as echocardiographic tubular mobile masses called 'ghosts'. Their presence has been associated with cardiac device-related infective endocarditis or local device infection, but to the best of our knowledge, this is the first case where 'ghosts' have been reported among non-infected patients.

Case summary We present a case of a 73-year-old woman hospitalized due to worsening dyspnoea and a significant pericardial effusion, relapsed after pericardiocentesis with removal of about 1500 mL of non-haemorrhagic fluid. The patient's history revealed a previous dual-chamber pacemaker implantation due to symptomatic sick sinus syndrome. Transoesophageal echocardiography (TOE), essential to exclude endocarditis vegetations suggested an etiopathogenesis of mechanical irritation caused by the distal end of the passive fixation atrial lead on the right atrial appendage wall. Considering the echocardiographic report and the condition of reactive pericarditis with the early relapse of the significant pericardial effusion after pericardiocentesis, we opted for a lead removal procedure to eliminate the stimulus causing the irritation, with transoesophageal echocardiographic monitoring, thus the early detection of a 'ghost' was possible.

Discussion This is the first clinical case describing the presence of fibrin 'ghosts' sometime after the implantation of a pacemaker, highlighting a non-exclusively infectious genesis, and emphasizing the importance of TOE for the early detection of this post-extraction complication and its monitoring.

Keywords Case report • Ghosts • Recurrent pericarditis • Transvenous lead extraction • Cardiac rhythm device

Learning points

- A persistent fibrous sheath in right-sided heart chambers after transvenous lead extraction has already been described as echocardiographic tubular mobile masses called 'ghosts'.
- This clinical case describes for the first time the presence of fibrin 'ghosts' even after a brief period from the implantation of a pacemaker, highlighting a non-exclusively infectious genesis.

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Handling Editor: Riccardo Liga
Peer-reviewers: Rami Riziq Yousef Abumuaileq and Robert Schönbauer
Compliance Editor: Anastasia Vamvakidou
Supplementary Material Editor: Peregrine Green

Introduction

After the transvenous lead extraction (TLE), the presence of a persistent fibrous sheath in the right-sided heart chambers has been described as echocardiographic masses called 'ghosts'. Their presence has been associated with cardiac device-related infective endocarditis (CDRIE) or local device infection (LDI) and consequently with a worse long-term outcome.[1]

Timeline

12 September 2017	Dual-chamber pacemaker implantation for symptomatic sick sinus syndrome
30 October 2017	Hospital admission due to worsening dyspnoea with evidence of significant pericardial effusion which required a first procedure of pericardiocentesis
15 November 2017	Second pericardiocentesis because of the early relapse of pericardial effusion and a transvenous lead extraction procedure to eliminate the irritative stimulus
17 November 2017	Re-implantation of a dual-chamber pacemaker
27 November 2017	Patient discharged
15 December 2017	Follow-up transoesophageal echocardiography showed reduced floating masses in the superior vena cavaand small pericardial effusion
23 January 2018	Follow-up chest computed tomography scan excluded the presence of 'intraluminal filling defects' related to episodes of thromboembolism

Case summary

A 73-year-old woman was admitted to our department due to worsening dyspnoea and weakness (in the absence of other symptoms) and evidence of significant pericardial effusion (*Figure 1*). The patient's history revealed dyslipidaemia, a previous thyroiditis Hashimoto (with normal thyroid function for 3 years) and a dual-chamber pacemaker implantation, less than 50 days previously for symptomatic sick sinus syndrome with bradycardia/tachycardia episodes (a passive fixation lead inserted into the right atrial appendage and an active fixation lead into the right ventricular apex). She was on therapy with furosemide 25 mg o.d. and Atorvastatin 20 mg o.d.

On admission she was haemodynamically stable (heart rate 85 b.p.m., blood pressure 105/60 mmHg, respiratory frequency 23 r.p.m., and temperature 36.7°C). Physical examination documented muffled heart sounds, reduced breath sounds in all lung fields and distended jugular veins. Electrocardiogram (ECG) showed sinus rhythm and diffuse low voltage. Transthoracic echocardiography revealed a circumferential pericardial effusion (>20 mm) with initial right ventricular diastolic collapse, inter-ventricular dependence, respiratory variations in mitral flow and inferior vena cava dilatation.

Laboratory investigations showed white blood cells 10.02 $\times$ 10^9/L (normal ranges 4–10), C-reactive protein 1.2 mg/dL (normal ranges 0–1), creatinine 1.51 mg/dL (normal ranges 0.5–1.1), and troponin I 16 ng/L (normal ranges 0–34). Anti-nuclear, anti-myeloperoxidase, anti-proteinase-3 antibodies, and Q-test for tuberculosis were negative.

Two hours after admission, pericardial effusion required pericardiocentesis for both haemodynamic relief and for diagnostic purposes, removing about 1500 mL of non-haemorrhagic fluid and almost complete resolution of the symptoms.

Whole body computerized tomography (CT), after pericardiocentesis, excluded neoplastic pathology and showed signs of pericardial inflammation, so we started therapy with ibuprofen 400 mg t.i.d. and colchicine 0.5 mg o.d. Transoesophageal echocardiography (TOE) did not demonstrate lead and valve vegetations, but it was not possible to exclude, with certainty, that the atrial lead minimally crossed the right appendage wall. Laboratory findings of pericardial fluid (exudative/reactive) confirmed the diagnosis of pericarditis and excluded perforation or malignant tumour, showing few macrophages and neutrophils (<250/μL) in the absence of erythrocytes and malignant cells. Bacterial cultures, polymerase chain reaction analysis and adenosine deaminase for mycobacteria were all negative. These findings supported a mechanical irritative mechanism caused by the tip of the atrial lead on the visceral pericardium.[2,3]

Considering the echocardiography, close temporal relationship and the progressive increase of pericardial effusion with recurrence of similar symptoms, 2 weeks after pericardiocentesis, we decided to perform TLE to eliminate the irritative stimulus.

In a hybrid operating room, a 9 Fr of pigtail catheter was positioned to drain about 600 mL of non-haemorrhagic pericardial fluid and to prevent any possible acute fluid collection after removal. The removal procedure was performed percutaneously, in deep sedation with TOE monitoring. After the disconnection of the leads from the generator, the fixation screw of the ventricular lead was first retracted. Checking the spontaneous cardiac rhythm, both leads were extracted by performing simple traction, with a substantial resistance of the right atrial lead (as if the tip was imprisoned in the myocardium).

On removal of both pacing leads, TOE showed an echogenic, long tubular mass with multiple offshoots floating into the right atrium (*Figure 2A–C*), attached to the superior vena cava (SVC). The inspection of both leads revealed that proximal coils were completely uncovered (*Figure 3*). We hypothesized that the floating mass in the right atrium could be a 'ghost', the residual fibrous sheath tear-out from the proximal coil (Supplementary material online, *Movies S1* and *S2*).

The post-operative observation in the intensive care unit was uneventful, but in the following days ECG monitoring highlighted episodes of symptomatic marked sinus bradycardia (heart rate 35 b.p.m.) with 2nd degree sino-atrial exit block and junctional escape rhythm treated with isoprenaline intravenous infusion, with re-implantation of a dual-chamber pacemaker (2 days later).

The patient was discharged from hospital in improved clinical conditions 5 days after re-implantation (with both passive fixation leads), in therapy with enoxaparin 3000 IU o.d., colchicine 0.5 mg o.d., and ibuprofen 400 mg t.i.d. We opted for a serial follow-up every 30 days. One month after discharge, TOE confirmed the presence of floating

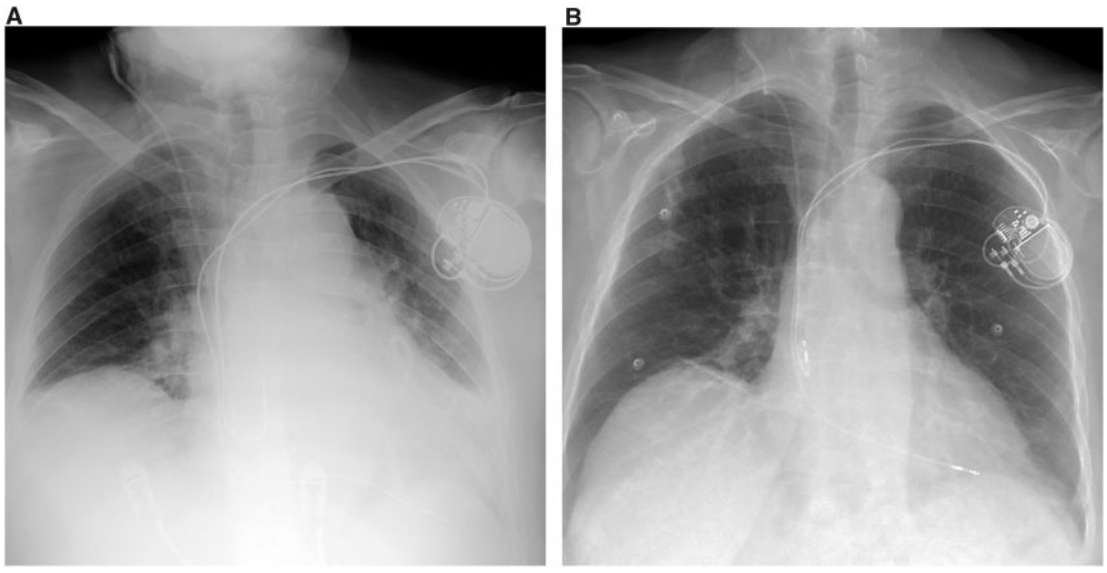

Figure 1 Chest X-rays before pericardiocentesis (*A*) and before lead extraction (*B*).

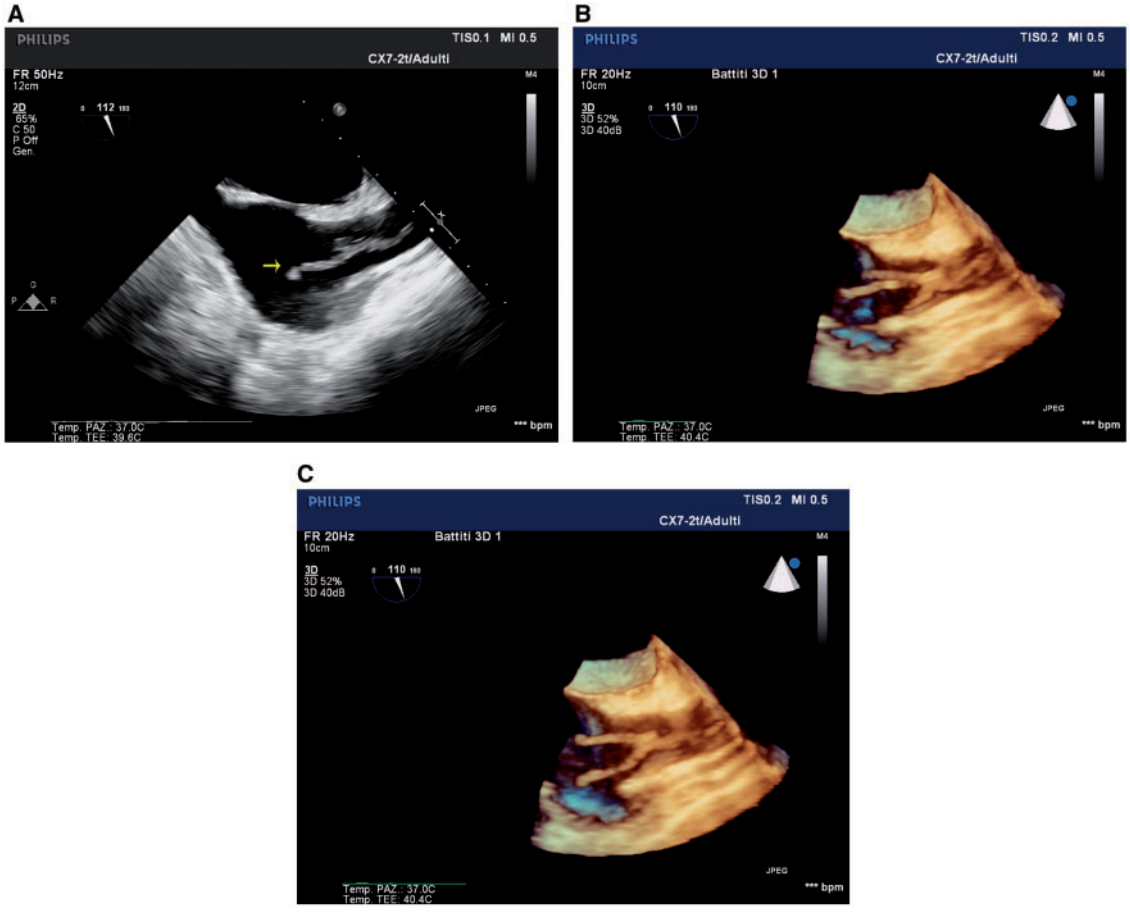

Figure 2 Long floating masses clearly visible (arrow), during extraction, at two-dimensional transoesophageal echocardiography in mid-oesophageal bicaval view (*A*); three-dimensional transoesophageal echocardiography imaging in two successive frames of the same cardiac cycle (*B* and *C*).

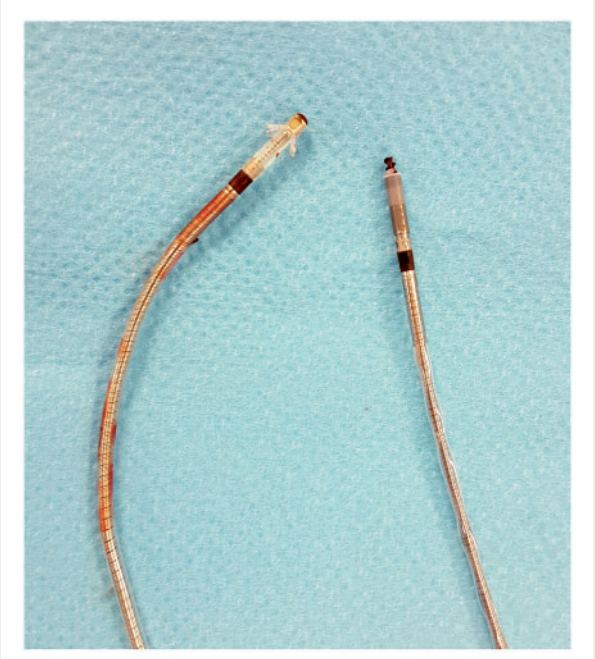

Figure 3 Atrial and ventricular leads after their extraction.

masses in the SVC and small anterior pericardial effusion (both findings significantly reduced compared to discharge), so we opted to continue colchicine 0.5 mg o.d. for another 2 months. Chest CT scan, performed 2 months after discharge, excluded the presence of 'intraluminal filling defects' related to episodes of thromboembolism.

At the last follow-up, 4 months after discharge, the patient was completely asymptomatic with normal pacing parameters and further reduction of the anterior pericardial effusion (2 mm). The next follow-up has been scheduled for 6 months.

Discussion

The number of implanted intracardiac devices, and consequently of lead removal procedures, has considerably increased over the years. The main indications for lead removal are CDRIE, LDI, and lead dysfunction.[4] After implantation, a fibrotic encapsulating process forms around the device leads. That fibrous sheath increases over time and becomes endothelialized as a way to tolerate foreign materials in the bloodstream.[5] After TLE, the presence of persistent fibrous sheath ('ghost') was associated with diagnosis CDRIE or LDI, but 'ghosts' were never observed among the remaining non-infected patients who underwent lead extraction.[1,5,6] The approach for these residual masses is unclear. Given the potential association between 'ghosts' and adverse outcomes, their presence indicates the need for a close clinical and echocardiographic follow-up to detect complications. In a consensus statement on lead extraction, no specific therapy, including mechanical removal, has been indicated for patients with incidental finding of 'ghosts'.[4] For a better patient compliance and considering a moderate chronic kidney disease, we opted for an antithrombotic prophylaxis with enoxaparin for 1 month after discharge.

The incidence of pericardial complications following pacemaker implantation may depend on the design of the lead and its fixation method as well as its location. These complications are not uncommon with active fixation atrial leads; instead the use of passive fixation leads is likely to reduce their incidence.[2]

All findings supported a mechanical irritative mechanism and other possible causes of pericardial effusion have been excluded. A pericardial effusion was absent on the echocardiography, 40 days before admission and a whole body CT excluded a neoplastic pathology. The patient's history, echocardiography, blood tests, clinical signs, and symptoms allowed us to exclude acute myocardial infarction and systemic autoimmune diseases. A moderate renal dysfunction has been present for several years and a significant new-onset of pericardial effusion with a tendency to rapid increase after the first pericardiocentesis, cannot be explained.

Conclusions

This case report describes for the first time the presence of the residual post-extraction tubular mobile mass called 'ghost' in an early lead extraction for non-infectious causes, in the context of reactive pericarditis. We hypothesize that the inflammatory reaction, in addition to the significant pericardial effusion, determined the early formation of 'ghosts'. Transoesophageal echocardiography was essential for early detection and the subsequent monitoring of this complication.

In our case report no major cardiac events or pulmonary embolic complications were observed, but the embolic potential of the 'ghosts' including ideal management has not yet been defined.

Supplementary material

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

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that written consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance.

Conflict of interest: none declared.

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