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Septic Pulmonary Embolism Caused by Infected Pacemaker Leads After Replacement of a Cardiac Resynchronization Therapy Device

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Conflict of interest: None declared

Patient: Male, 70
Final Diagnosis: Pacemaker leads endocarditis
Symptoms: Bacterial lead endocarditis • congestive heart failure • fever • pacemaker dysfunction
Medication: —
Clinical Procedure: Pacemaker box replacement due to end-of-service
Specialty: Cardiology

Objective: Unusual clinical course

Background: Cardiac resynchronization therapy (CRT) has been demonstrated to reduce morbidity and mortality in patients with advanced, drug-refractory heart failure. Procedure-related mortality is less than 1% in larger studies. Approximately 10% of CRT patients have to undergo surgical revision because of infections, dislocations, or unacceptable electrical behavior manifested as high threshold, unstable sensing, or unwanted phrenic nerve stimulation.

Case Report: A 70-year-old man with symptomatic congestive heart failure underwent implantation of a biventricular pacemaker on the left anterior chest wall in 2003 and pulse generator exchange in August 2009. The patient responded well to CRT. At follow-up, the pacing system functioned normally. In September 2009, in the context of a predialysis program, an abdominal computed tomography (CT) scan was performed in another hospital for assessment and evaluation of chronic kidney disease. This procedure was complicated with peripheral thrombophlebitis that was managed appropriately with complete recovery. Eight months later (May 2010), the patient was admitted to our hospital with fever, anemia, and elevated infection parameters. During admission, blood cultures grew *Staphylococcus epidermidis*. The chest X-ray, lung perfusion scintigraphy, and CT scan depicted pulmonary embolism and infarction. The right ventricular lead threshold was found to be increased to 7 volts with unsuccessful capture. Echocardiography demonstrated vegetations on leads. The entire pacing system was explanted, but the patient expired few days later following percutaneous removal due to multiorgan failure.

Conclusions: In heart failure, replacement of the CRT device may be complicated by bacterial endocarditis. As noted from this case report, sudden elevation of the pacing lead threshold should prompt thorough and immediate investigation to unravel its causes, not only the electrical characteristics but also the anatomical features.

MeSH Keywords: Cardiac Resynchronization Therapy Devices • Endocarditis, Bacterial • Heart Failure • Pulmonary Embolism

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Background

Cardiac resynchronization therapy (CRT) has been demonstrated to reduce morbidity and mortality in patients with advanced, drug-refractory heart failure in association with impaired left ventricular function (ejection fraction ≤ 0.35) and prolonged QRS duration (QRS ≥ 150 msec) [1–4]. Procedure-related mortality is less than 1% in larger studies [5,6], and 10% of these patients have to undergo surgical revisions due to infections, dislocations, or unacceptable electrical behavior manifested as high threshold, unstable sensing, or phrenic nerve stimulation. There are hardly any differences reported in the incidence of lead infections in primo implanted CRT systems compared with upgrades to CRT [7]. Lead infections, in general, are often complicated by vegetations at the intracardiac aspect of the leads including valvular leaflets [8]. Several treatment modalities are currently available for pacemaker infections such as conservative measures with intensive antibiotics [9], percutaneous transvenous extraction [10], and surgical removal [11] or a combination thereof. We report a case of infected pacemaker leads with *Staphylococcus epidermidis* causing septic pulmonary embolism. The patient was referred to tertiary hospital for system removal. He died due to multi-organ failure a few days later after explantation.

Case Report

A 70-year-old man with symptomatic chronic heart failure (CHF) with a left ventricle ejection fraction (LVEF) of 27% was treated, after a straightforward procedure, with cardiac resynchronization therapy (CRT) on December 8, 2003. Past medical history was significant for chronic obstructive pulmonary disease, chronic kidney disease, anemia, hypertension, and permanent atrial fibrillation (AF). For AF with rapid ventricular frequency that was not responding well to medical therapy, a redo His bundle ablation was performed after VVIR pacemaker implantation on August 8, 2002. Due to development of congestive heart failure with reduced ejection fraction (LVEF 27%), upgrading to biventricular pacing was undertaken on December 8, 2003. In 2004, he developed intracerebral hemorrhage of the left hemisphere under adequate anticoagulation. He was a good responder to CRT, as evidenced by increment of LVEF from 27% in November 2003 to 42% in June 2010. Because of end-of-service criteria of the pulse generator, an exchange procedure was indicated. A few years later (August 25, 2009), a routine uncomplicated exchange was performed, where the CRT device was replaced and the leads, after electrical evaluation, were kept *in situ*. The lead positions are shown in (Figure 1). The pacing threshold was 0.8 volts with pulse duration of 0.4 msec, and impedance was 480 ohms during pacemaker dependency.

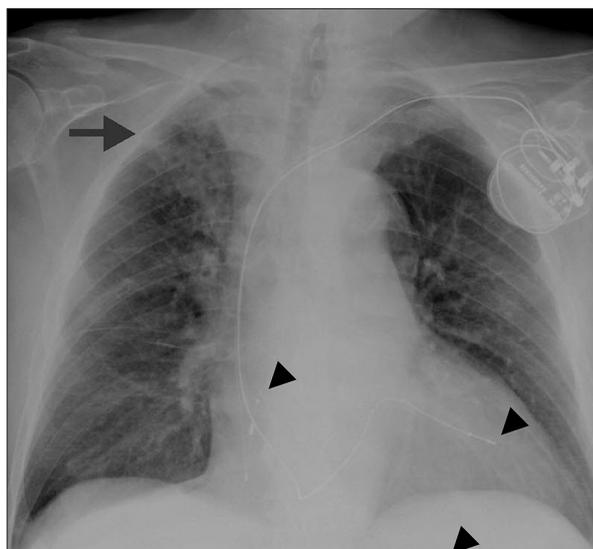


Figure 1. Chest X-ray demonstrating cardiomegaly with cardiac resynchronization therapy pacing leads (arrowheads) and segmental defect of the right upper lobe (arrow).

Because of deterioration of chronic kidney disease, the patient was scheduled for a predialysis outpatient clinic. An abdominal computed tomography (CT) scan with adequate prehydration was performed for further evaluation of chronic kidney disease upon recommendation of his nephrologist (September 30, 2009), which yielded a bilateral renal artery stenosis (50%) with small kidneys and cortical loss, and infrarenal aneurysm of the abdominal aorta (4.5 cm). His hospital stay was complicated, after venous cannulation, with peripheral thrombophlebitis of the upper extremity accompanied with fever that was managed appropriately with complete recovery. Further investigations revealed no other focal inflammation. The pacemaker pocket was not infected, and the echocardiography findings were negative for possible vegetations.

His medical regimen was continued including oral anticoagulants, amiodarone, bumetanide, atorvastatine, long-acting nitrates, barnidipine, and alphacalcidol. His condition remained stable until he was admitted on May 22, 2010, to our hospital with fever (39.2°C), elevated infection parameters, concomitant anemia, and renal function impairment. No redness or swelling of the skin over the pocket was seen. The serial blood cultures were positive for *Staphylococcus epidermidis*. This may be considered as a late complication of the replacement procedure itself. Treatment with vancomycin and rifampicin was initiated. The patient developed severe skin rash with eruptions, necessitating the withdrawal of both antibiotics. The infection parameters remained elevated. Transthoracic and transesophageal echocardiography (TTE and TEE) demonstrated visible vegetations on the pacemaker leads (Figure 2A, 2B) with moderate left ventricular systolic dysfunction. Lung perfusion scintigraphy depicted segmental defect of the right

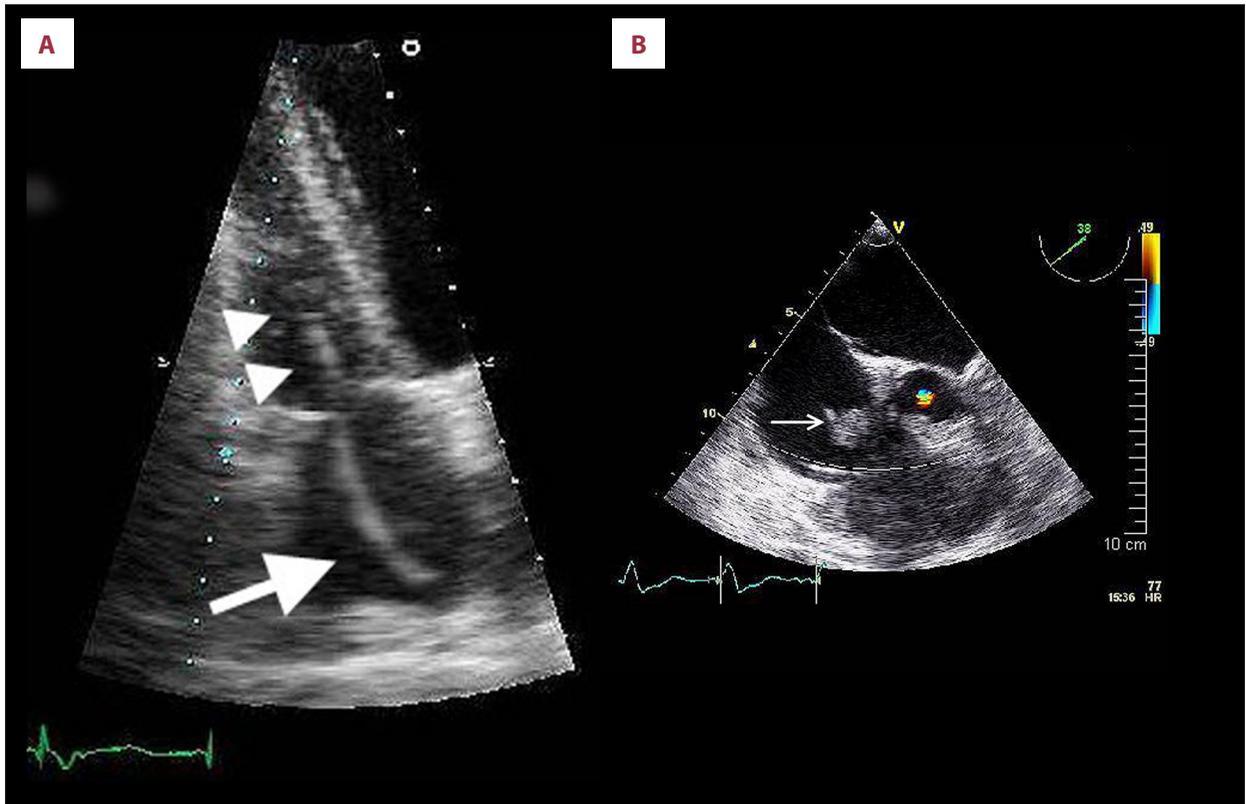


Figure 2. Transthoracic (A) and transesophageal (B) echocardiography demonstrating vegetation of the pacemaker lead at the atrial and ventricular levels (arrows) and an abnormal high-density mass in the right atrium (arrow).

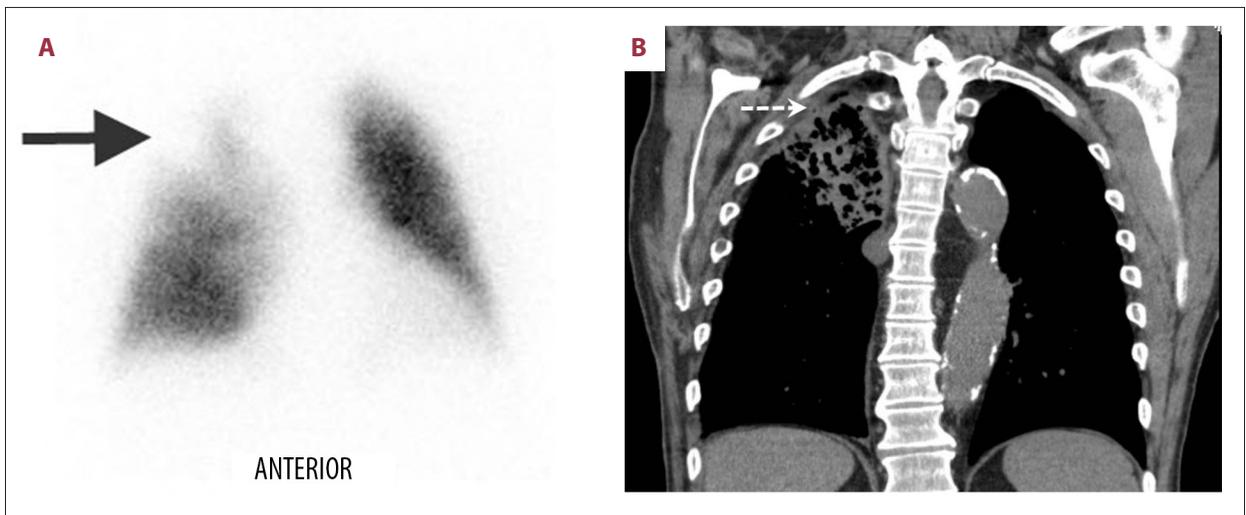


Figure 3. (A) Pulmonary perfusion scintigraphy showing segmental defect of the right upper lobe (arrow) and (B) chest computed tomography scan, coronal view illustrating segmental defect of the right upper lobe (arrow).

upper lobe (Figure 3A). Plain thoracic CT scan revealed pulmonary infarction of the posterior segment of the right upper lobe (Figure 3B), as well as multiple gall bladder stones, splenomegaly, bilateral renal parenchymal loss, and aneurysmal dilatation of the abdominal aorta of 4.8 cm.

During the chronic phase of CRT, threshold measurements were stable for all leads. Also at replacement no deviations in electrical behavior were observed. Follow-up (May 22, 2010) showed an increase in the threshold of the right ventricular lead up to 7.0 volts at 0.5 msec pulse duration accompanied with loss of capture and decrease of right ventricular lead impedance (Figure 4A, 4B).

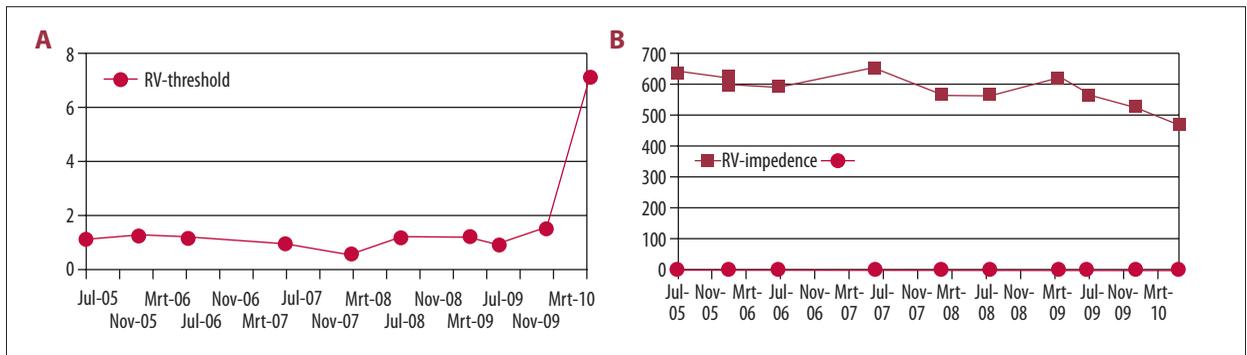


Figure 4. (A) Changes in right ventricular (RV) pacing threshold over time and (B) decrease in RV lead impedance.

Left ventricular pacing was the only available option; there was no capture possible during right ventricular pacing.

A decision was taken to remove the pacemaker system. The transvenous lead extraction was complicated with cardiac, renal, and pulmonary insufficiency necessitating inotropic drug treatment, dialysis, and mechanical ventilation. The condition deteriorated rapidly and the patient succumbed. Permission for autopsy was not given by his family.

Discussion

The present patient developed *Staphylococcus epidermidis* pacing leads endocarditis late after replacement of a CRT device with concomitant deterioration of renal function and development of skin rash as a reaction to antibiotic treatment.

Incidence and prevalence

Bacterial invasion of the cardiac devices is not a rare finding. Pichlmaier et al. found high prevalence of asymptomatic carriers of bacterial colonization of rhythm management devices [12]. In 47.2% of the patients who underwent elective device replacement, bacterial DNA was demonstrated on the device and/or the device pocket. Staphylococci were demonstrated in only 3.7% of the patients who became symptomatic later [12]. In a prospective study of 1744 patients with elective pacemaker or implantable cardioverter-defibrillator generator replacement, Poole et al. found major infectious complications requiring extraction of pulse generator and leads in 0.8% of those without upgrade and 0.8% of those with upgrade to CRT [7]. Sohail et al. reported that cardiac device infections occurred after initial device implant in 42% of patients, and after revision or re-exploration this figure increased up to 58% [13].

Microorganisms

Staphylococcus aureus and *epidermidis* are the most common infecting microorganisms [8]. The early infections of

pacemaker pocket and lead endocarditis are mainly due to perioperative bacterial infiltration; *Staphylococcus aureus* is commonly seen early after implantation [14]. In contrast, in latent delayed pocket infections, *Staphylococcus epidermidis* is the most common infecting microorganism [13,15], as was the case in the current patient. Other isolated microorganisms were coagulase-negative *Staphylococci*, gram-negative bacilli, *Pseudomonas aeruginosa*, and *Corynebacterium* species [13,15]. Not only bacterial infections have been incriminated in pacemaker lead endocarditis, but also fungal *Aspergillus* infection has been reported [13,16]. Moreover, several microorganisms were shown in asymptomatic carriers: *Pseudomonas* (16%), *Staphylococcus* (11%), *Stenotrophomonas* (10%), *Rhizobium* (9%), and *Propionibacterium* (7%) [12]. In patients with endocardial vegetations, *Staphylococcus aureus* was isolated in 60% [8].

Predisposing factors

Predisposing factors for infection include hematoma, erosion, long duration of procedure, operator's inexperience, and re-exploration [17].

Clinical presentations and management

Cardiac device infections present as local discharge, inflammation or abscess formation, erosion of part of the cardiac device system through the skin with subsequent infection, or rarely with endocarditis, sepsis, and positive blood cultures [15]. Blood cultures were positive in only 40% of all subjects [13]. Cardiac device infections may include lead endocarditis or pocket infection. Lead endocarditis may be complicated by secondary dissemination of septic pulmonary embolism [14,18].

In the series of Sohail et al., extraction of the infected system was performed in 94% of subjects [13]. Extraction can be performed percutaneously or after median sternotomy with cardiopulmonary bypass [8,14]. High mortality rate (25%) is associated with retaining the infected system without removal [19,20].

Despite improvement and use of sophisticated lead materials and designs and superior implantation techniques, infections of the pacemaker pocket or leads remain a serious disorder carrying a high death rate. The reported overall mortality rate after long-term follow-up was 26.9% [14]. Device-related endocarditis was one of the most common clinical presentations of cardiac device infections, found in 23% of patients in the series of Sohail et al. [13]. Pacemaker endocarditis is complicated by a high rate of pulmonary embolism varying from 31.2% [14] to 55% [8] of patients, as was found in our present case. There is general consensus that once there is pacemaker pocket or lead infection with or without lead endocarditis, removal of the whole pacemaker system followed by a course of intravenous or oral antibiotics results in a favorable outcome [14]. Percutaneous transvenous extraction of infected permanent endovascular leads is reserved for conditions associated with vegetations less than 10 mm [14]. In case of large vegetations, surgical removal of the infected system is indicated [8].

One of the major advantages of TTE is noninvasive detection of a vegetation, a hallmark of the lead infection. TEE discloses an abnormal appearance on the pacemaker lead in 94% of cases [14]. It has been observed that vegetations >15 mm in diameter are associated with pulmonary embolic events [8].

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Conclusions

In CHF, replacement of the CRT device may be complicated by bacterial endocarditis. Extra caution should be taken to prevent the occurrence of such a complication. As noted from this case report, sudden elevation of the pacing lead threshold should prompt thorough and immediate investigation to unravel its causes, not only the electrical characteristics but also the anatomical features.

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Conflict of interest

None to declare.