

Diagnosis and treatment of lead-dependent infective endocarditis

Diagnostyka i leczenie infekcyjnego zapalenia wsierdza związanego z elektrodą

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Abstract

We report a typical case of lead-dependent infective endocarditis in an 84 year-old patient, occurring two years after pacemaker implantation. Before the correct diagnosis was reached, the patient was repeatedly hospitalised in several centres due to fever of unknown origin. Eventually, the diagnosis was confirmed when the patient was referred for transoesophageal echocardiography by a consulting cardiologist from the pacemaker implantation centre. The successful treatment included the removal of the whole pacing system with endocardial leads and adequate antibiotic therapy.

Key words: lead-dependent infective endocarditis, lead extraction

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INTRODUCTION

The incidence of the late complication of pacemaker/cardioverter-defibrillator (PM/ICD) implantation increases with the growing number of implantation procedures [1]. Infective complications, including lead-dependent infective endocarditis (LDIE) in the right heart, are among the most serious [2], and occur in 1.9 of 1,000 implants per year [3]. The LDIE can have obscure clinical course and symptoms, leading to a delayed, or even no, diagnosis [2, 4]. We describe a case of typical course of LDIE in a patient implanted with a DDD pacemaker.

CASE REPORT

An 84 year-old female with a history of moderate arterial hypertension, chronic renal failure and implantation of cardiac PM was referred to our centre with a diagnosis of severe lead-dependent infective endocarditis. Two years earlier, due to highly symptomatic brady-tachycardia syndrome with recurrent syncope, she had been implanted with dual-chamber PM (two bipolar screw-in leads in right atrial appendage and right ventricular apex, connected to a Medtronic Sensia DR pacemaker) in another centre. The first symptoms occurred three months before referral to our centre, and included re-

current fever (up to 40°C), chills, malaise, muscle pain, nausea, and loss of body weight. Initially, the patient was admitted to the pulmonology ward in a district hospital with a diagnosis of upper respiratory tract infection, and empiric antibiotic treatment was launched. The chest X-ray revealed slight cardiomegaly with clear lungs; no blood cultures were taken. Blood analysis revealed increased WBC and ESR rate.

One month after discharge, she was admitted for the second time to the hospital due to recurrence of the previous symptoms. Laboratory findings included elevated inflammatory markers (CRP, ESR, WBC), slight anaemia and slight renal insufficiency. This time, blood cultures were taken, of which one was negative and the second yielded *Staphylococcus lugdunensis* sp. Antibiotic therapy was administered according to susceptibility tests. Chest X-ray and abdominal ultrasonography yielded no specific findings. Transthoracic echocardiography (TTE) revealed no vegetations, but transoesophageal echocardiography (TEE) was not performed. After the antibiotic treatment, the symptoms diminished and the patient was discharged.

A month later, she was admitted to hospital for the third time due to recurrence of symptoms (fever and chills), this time accompanied by relentless cough. Laboratory findings

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Figure 1. Vegetations on ventricular lead in TEE examination

revealed persistent elevated inflammatory markers (CRP, ESR, WBC). Bronchoscopy was performed, and due to suspicion of fungal infection, therapy with nystatin was administered. After the computed tomography (CT) scan, which revealed local, non-specific pulmonic consolidations, the patient was transferred to a reference centre (Department of Pulmonology, Oncology and Allergology). At this time (three months from the onset of symptoms and during the fourth hospitalisation), the consulting cardiologist referred the patient for TEE, which revealed vegetations on the ventricular lead (Fig. 1) and a diagnosis of infective endocarditis was made.

Previously described findings in the CT scan (without angiography mode) probably resulted from infected pulmonary emboli in the course of endocarditis. The patient was instantly referred to the Department of Cardiology. Laboratory findings revealed still elevated values of WBC, ESR, CRP and procalcitonin, and D-dimer as well. Blood cultures were taken once more, yielding *Staphylococcus lugdunensis* sp., which was sensitive to vancomycin administered at admission.

We decided to remove both PM and intracardiac leads. Different explanation approaches, including surgical intervention, were discussed. Considering the patient's age and status (prolonged septicaemia, renal failure, anaemia), despite the borderline vegetation size, we decided to select transvenous lead extraction [5]. After confirmation of the presence of sufficient, stable spontaneous rhythm, the PM pocket was opened under local anaesthesia, with no signs of local infection. Both leads were cut off and the PM removed. The insulation of the ventricular lead was found to be broken, with the presence of a purulent secretion.

Swabs were taken for microbiological cultures. We found symptoms of chronic inflammation of lead tunnel in subclavian region. In order to avoid laceration of subclavian vein and spreading the infection, we removed both leads by gen-

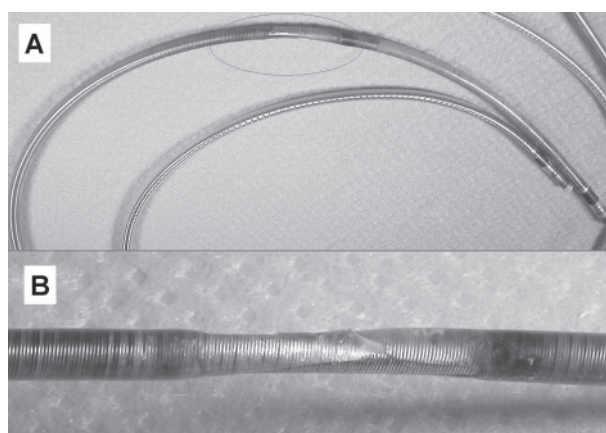


Figure 2. Both leads after extraction (A) evident friction-related insulation damage on ventricular lead (B)



Figure 3. Vegetation on anterior mitral valve leaflet in TEE after lead extraction

tle screw-out and simple manual traction, which was successful. We found complete insulation damage on ventricular lead caused by chronic friction in the vicinity of the tricuspid valve (Fig. 2).

There were no complications of the procedure. The drainage was removed three days after extraction. Interestingly, the swabs taken during the procedure yielded no growth. In the following days, the patient was doing well, with no fever and normalisation of inflammatory markers (WBC, ESR, CRP) and D-dimer at discharge. However, in the control TEE study, we found a vegetation (2.8 × 0.8 cm) on the atrial aspect of the anterior mitral valve leaflet (Fig. 3).

The ongoing antibiotic therapy (vancomycin) was continued for three weeks. Since the patient remained on stable sinus rhythm, with no signs of sinus node incompetence nor atrioventricular conduction dysfunction on 24-hour Holter monitoring, we decided to postpone the reimplantation pro-

cedure. Four weeks after discharge, the patient was admitted to our centre to re-confirm the indications for reimplantation. She remained on stable sinus rhythm, control TEE revealed no vegetations, and inflammatory markers were normal. In 24-hour Holter monitoring, there were no indications for cardiac pacing, and she was discharged for further follow-up in the outpatient clinic.

DISCUSSION

In the reported case, two points in particular merit attention. First, the obscure symptomatology, which is characteristic of LDIE, resulting in delayed diagnosis. In the presented case, it significantly postponed appropriate treatment. The described, prolonged period of diagnostic evaluation demonstrates that many healthcare professionals are unfamiliar with the symptoms of right heart infective endocarditis.

It is our firm opinion that in all patients implanted with PM/ICD with fever of unclear aetiology (particularly with coexisting symptoms suggesting pulmonary infection), LDIE should be suspected. Therefore, they should be referred for TEE examination.

The second interesting observation is the complete insulation damage found on the ventricular lead. This finding seems to be crucial in the pathogenesis of LDIE — bare metal coil predisposes for vegetations [6, 7]. Moreover, this can also explain resistance to antibiotic therapy. The complete remo-

val of the infected leads is essential for successful treatment, according to current guidelines [2, 4, 5]. Of the two available approaches, surgical and transvenous extraction, we strongly advocate the latter as being safer and more effective, even in the presence of lead-dependent vegetations.

Conflict of interest: none declared

References

1. Kutarski A, Małecka B. Późne powikłania stymulacji — gdzie jesteśmy, dokąd idziemy? *Kardiologia po Dyplomie*, 2009; 8: 14–22.
2. Małecka B, Kutarski A. Lead-dependent infective endocarditis: an old problem, a new name. *Cardiol J*, 2010; 17: 1–6.
3. Uslan DZ, Sohail MR, St Sauver JL et al. Permanent pacemaker and implantable cardioverter defibrillator infection: a population-based study. *Arch Intern Med*, 2007; 167: 669–675.
4. Habib G, Hoen B, Tornos P et al. Guidelines on the prevention, diagnosis and treatment of infective endocarditis (new version 2009). *Eur Heart J*, 2009; 30: 2369–2413.
5. Wilkoff BL, Love CJ, Byrd CL et al. Transvenous lead extraction: Heart Rhythm Society expert consensus on facilities, training, indications, and patient management. *Heart Rhythm*, 2009; 6: 1085–1104.
6. Kutarski A, Małecka B. Przetarcie silikonowych izolacji elektrod wewnątrzsercowych — nowo odkryte zjawisko w elektroterapii: obserwacje własne. *Folia Cardiol Exc*, 2009; 4: 126–131.
7. Kutarski A, Małecka B, Ruciński P et al. Percutaneous extraction of endocardial leads: a single centre experience in 120 patients. *Kardiol Pol*, 2009; 67: 149–156.