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# Lead-dependent infective endocarditis complicated by septic emboli

## Infekcyjne zapalenie wsierdzia związane z obecnością elektrody, powikłane zatorami septycznymi

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**Abstract**

Lead-dependent infective endocarditis (LDIE) has emerged as a serious complication of electrotherapy in the era of advanced medical technology and is a growing problem due to greater patient longevity, limited electrode life-time, an increasing number of abandoned leads, and subclinical symptoms. We present a case of dramatic course of LDIE in a 26 year-old patient in whom standard management had failed to cure endocarditis. This case was complicated by extensive pulmonary septic emboli and required cardio-thoracic intervention.

**Key words:** lead-dependent infective endocarditis, electrotherapy complications, pulmonary septic emboli, transvenous lead extraction, Byrd dilatators

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## CASE REPORT

A 26 year-old Caucasian male presented with fever of a duration of a few months. Past medical history included corrected transposition of the great vessels and implantation of a DDD pacing system 11 years previously due to congenital complete heart block. At that time, two active electrodes Pacesetter Tendril and pacemaker Pacesetter Trilogy DR+ were implanted, with longer loops of leads left in the right atrium due to the anticipated growth of the body at the conclusion of adolescence.

After ruling out potential sites of infection, transthoracic/ /transoesophageal echocardiography (TTE/TEE) revealed a vegetation of 1.3 × 3.1 cm attached to the ventricular lead and a diagnosis of lead-dependent infective endocarditis (LDIE) was made. At this time, laboratory signs of inflammation were subtle: WBC 11.000/$\mu$L (N: 4.0–10.5), CRP 28.2 mg/L (N: 0.0–5.0) with negative blood cultures. Because difficulties regarding lead extraction were foreseen, the patient was transferred to the reference centre.

Diagnostic imaging at the reference centre, in the form of angiographic multislice computed tomography (angio--MSCT) (Fig. 1), showed spontaneous dislocation of the vegetation into the pulmonary artery, with complete occlusion of the left inferior lobar artery. TTE/TEE confirmed the presence of two smaller vegetations, one in the superior vena cava (SVC) and the second attached to the ventricular lead. Continuous dynamic contact between the electrodes and mutual friction was observed on the fluoroscopy scan. Blood gasometry showed a deficit in oxygenation ($pO_2$ 56 mm Hg) with borderline $O_2$ saturation of 90.3% in room air.

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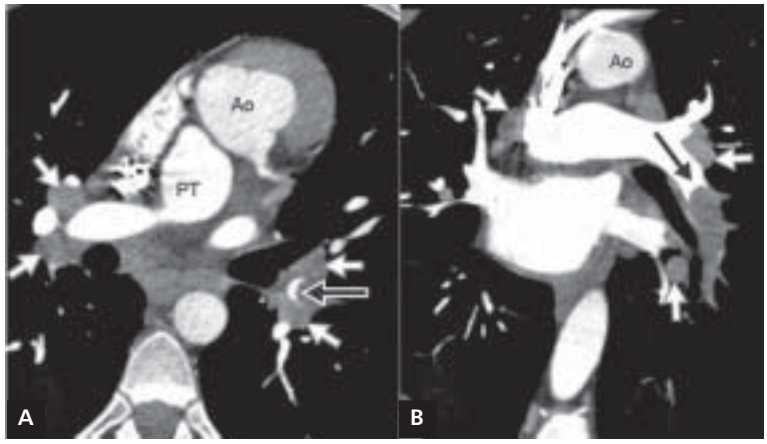


**Figure 1.** Angio-MSCT — arterial phase. In axial view (**A**) and oblique reconstruction (**B**) visible embolus totally occluding the lumen of left low-lobar artery (black long arrow), presence of numerous enlarged mediastinal lymph nodes (short white arrows); Ao — aorta; PT — pulmonary trunk

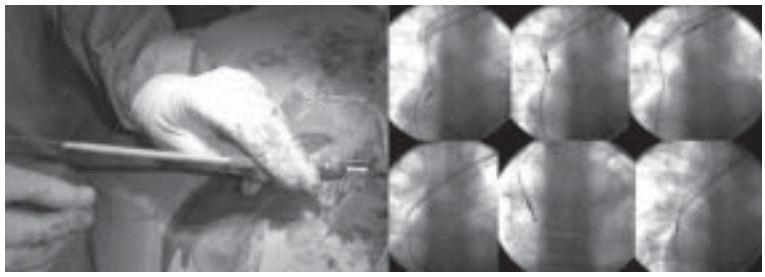

**Figure 2.** Transvenous lead extraction using Byrd dilatators. The system is used for separation of connective tissue adhesions from electrodes

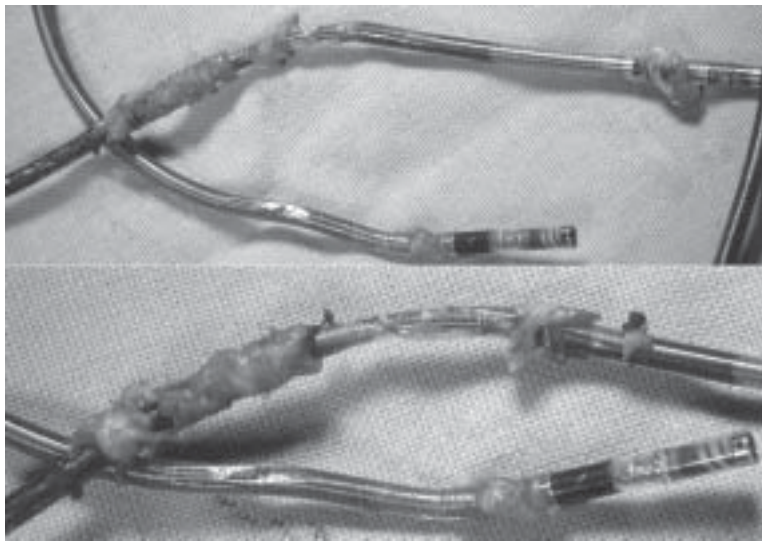

**Figure 3.** Removed leads. Connective tissue and abrasions of external silicone sheaths with exposure of metal coil due to mutual friction are visible

Total percutaneous pacing system extraction was performed successfully using a mechanical system of Byrd dilatators (Cook Co.) (Fig. 2). There was an outflow of pus from the lumen leads during penetration by guidewire inside the leads, indicating local infection. The removed electrodes had multiple lead abrasions (Fig. 3) related to their dynamic contact, as seen earlier on the fluoroscopy scan. Lead cultures were taken and these showed no growth.

Efforts to transvenously recanalise a huge embolus in the left pulmonary artery were unsuccessful. Control angio-MSCT

**Figure 4.** Embolic masses removed during surgery from left pulmonary artery

confirmed that the embolus remained. Despite total pacing system extraction, intensive antibiotic therapy and negative results of all cultures (blood × 2, skin × 5, pocket × 2, electrode × 4), values of inflammatory parameters were increasing — CRP: 42.7; 100.9; 121.6; 79.4 mg/L; as well as values of D-dimer: 3,702.0; 662.0; 943.0; 1,088.0 μg/L (N: < 500 μg/L). Control TEE showed again vegetation or thrombus at the ostium of the SVC with acceleration of flow. Anticoagulation with intravenous heparin and subsequent warfarin was started. The decision as to implantation of a new pacing system was postponed indefinitely due to stable ventricular escape rhythm.

For the next three weeks, the patient remained febrile with laboratory signs of inflammation. Serial TEE and angio-CT confirmed enlargement of the thrombus/vegetation at the SVC and total obliteration of the left pulmonary artery (5.3 × 1.6 × × 1.8 cm). The described findings were associated with enlargement of the mediastinal lymph nodes up to 2.6 × 1.2 cm.

Considering that the clinical picture was complicated by septic emboli and the ineffectiveness of subsequent antibiotic regimens: vancomycin and gentamycin; rifampicin, ceftazidime and teicoplanin; linezolid, meropenem and ketoconazole, a final decision was taken for cardio-thoracic intervention to remove septic emboli from the SVC and the left inferior lobar artery.

The surgery was performed from a sternal approach. Opening of the pulmonary trunk and left pulmonary artery allowed for the removal of the huge embolus, which looked like a waxy cast of the artery and was 9 cm long (Fig. 4). Successful surgery ended with epicardial permanent pacing system implantation.

In the post-operative period, we observed spectacular clinical improvement. The fever resolved and the patient was discharged home in good condition after a 14-day course of linezolid and meropenem therapy. A follow-up two weeks later showed no signs of infection and effective VVI pacing. The patient returned to normal active living.

## DISCUSSION

In this case, some facts need to be empasised. The fundamental pathogenetic factor for development of complicated LDIE was mutual friction of exceedingly long loops of electrodes implanted in childhood. Multiple abrasions of lead sheaths with subsequent exposure of metal coil constituted an excellent milieu for the growth of bacteria [1–3]. This local infection developing under damaged sheaths goes a long way to explaining the ineffectiveness of antibiotic therapy and the formation of septic emboli.

Awareness of subclinical LDIE symptoms is low. The diagnosis can be easily missed based on TEE, as the vegetation may be dislocated into the pulmonary artery.

Transvenous lead extraction is a safe and effective procedure of LDIE management at experienced centres. At our centre, of 80 patients with LDIE, 97.5% were treated successfully with a zero mortality rate. Only two cases (2.5%), including this patient, required subsequent open-heart surgery [4–8].

Based on this experience, a conclusion can be made that although TEE constitutes the gold diagnostic standard in LDIE, angio-MSCT is an important tool, as it allows not only visualisation of the vegetation, which may disappear from the echocardiographic field, but also the evaluation of other potential embolic material and the patency of the SVC [4–8].

We suggest the inclusion of high resolution computed tomography (angio-MSCT) in association with TEE as gold standards in the diagnosis of LDIE. It is vital to appreciate the significance of LDIE as an underestimated problem of 21st century medicine.

***Conflict of interest:*** *none declared*

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