All that glitters is not gold: positron emission tomography/computed tomography detection failure in a patient with implanted pacemaker and relapsing Corynebacterium striatum sepsis

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Over the last several years the number of implanted cardiac electronic devices has increased. This fact has been accompanied by a higher prevalence of cardiac device-related infective endocarditis (CDRIE), which is usually diagnosed with the use of echocardiography. However, in echocardiographically negative cases with a high index of suspicion for CDRIE the fluorodeoxyglucose labelled with fluorine-18 (18F-FDG) positron emission tomography (PET) coregistered with computed tomography (CT) appears to be a useful technique for further evaluation [1].

We present a very rare case of a patient with implanted pacemaker with recurring Corynebacterium striatum bacteraemia and negative advanced imaging tests. A 79-year-old white woman seven years after pacemaker implantation was admitted to the Department of Cardiology due to two-day history of fever, dizziness, and progressive weakness. Laboratory tests revealed elevated serum levels of C-reactive protein (up to 114 mg/L), procalcitonin (up to 1.64 ng/mL) and mildly elevated white blood cell count (12.5 × 10⁹/µL). Because of three episodes of C. striatum sepsis of unknown origin within the preceding six months, the empiric followed by antibiotic-guided treatment with vancomycin and gentamycin IV was implemented. Three blood cultures yielded C. striatum. Transthoracic (TEE) and transoesophageal (TEE) echocardiography as well as chest X-ray findings were normal. Abdominal ultrasound also revealed no abnormalities. Pacemaker interrogation showed increased pacing threshold of the ventricular lead. Because of the strong suspicion of CDRIE, PET/CT fusion imaging was performed on a BIOGRAPH 64 (Siemens, Munich, Germany) PET/CT scanner (Fig. 1). The examination was conducted after overnight fasting. PET/CT showed metabolically activated enlarged mediastinal nodes and pathological glucose spleen uptake, but no pathological 18F-FDG uptake in the heart. Despite negative results of the imaging tests, due to a high probability of CDRIE we decided to continue antibiotic therapy. Control laboratory tests revealed normal serum levels of inflammatory markers. Due to pacemaker dysfunction, percutaneous device extraction was performed. Postprocedural blood and urine cultures were negative, but the lead culture grew C. striatum. Monotherapy with vancomycin was continued for a subsequent four to six weeks. Because there was no strong indication for device reimplantation, the procedure was delayed. The patient remained in good clinical condition and was discharged home. Corynebacterium striatum is a gram-positive, multidrug-resistant bacterium, mostly nosocomial, causing major infections, and it is associated with the presence of implantable materials [2]. The patient reported here had been hospitalised three times within the preceding six months due to Corynebacterium striatum sepsis; however, imaging tests such as TTE, TEE, or even PET/CT were unable to detect the origin of infection. False negative result of 18F-FDG PET/CT might be associated with the low spatial resolution of this modality, especially when intracardiac leads are evaluated. Antibiotic therapy may also affect the sensitivity of diagnostic imaging. 18-FDG PET/CT is a useful imaging tool in the detection and localisation of infection, but its reliability in CDRIE diagnosis is limited, which should be taken into account when device removal is considered.

References

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