

Infective Endocarditis and The Pacemaker: Cardiac Implantable Electronic Device Infection

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SUMMARY

We are seeing more implantation of cardiac device such as pacemakers and defibrillators and also cardiac implantable electronic device infection. The infection may affect just the pocket site or progress to deeper infection and bacteraemia. Inadequately treated infection may lead to right sided endocarditis, cardiomyopathy for pacemaker explantation and increased cost and length of stay in the hospital. We report a Staphylococcal infection of a pacemaker system, its successful medical and surgical management

KEY WORDS:

Infective Endocarditis; Tricuspid Valve; Cephazolin Prophylaxis; Pulmonary Embolisation; Pacemaker Infection; Staphylococcus aureus; Pacemaker Explantation

INTRODUCTION

Infective endocarditis (IE) is a form of infection and inflammation of the endocardial segment of the heart in the setting of predisposing cardiac lesions. It is usually of bacterial origin and entails long duration of intravenous (IV) antimicrobial therapy. If IE develops in the setting of a cardiac implantable electronic device (CIED) such as a pacemaker, infection may be difficult to treat without explantation of the pacemaker system.

CASE REPORT

A 47 year old lady suffered from symptomatic sick sinus syndrome and had a dual chamber rate responsive Prodigy® (Model DR 7860, Medtronic, Inc., Minneapolis, MN, USA) pacemaker implanted in 1998. The pacemaker leads were placed in the right atria and right ventricle via the left subclavian vein with the pacemaker generator secured subcutaneously in the left deltopectoral area. As the pacemaker generator was almost at end of life, she underwent a new generator change with a single chamber rate responsive Sensia® (Model SESR01, Medtronic, Inc., Minneapolis, MN, USA) in 2009. The previous atrial lead header was capped. Intravenous cefoperazone 1g was given 30min before the procedure and another dose 6 hours after the procedure.

She developed fever with pus discharge from the swollen pacemaker pocket site one week later. Blood culture was negative but pus swab from the wound site grew *Pseudomonas aeruginosa*. IV cloxacillin 2g 4hourly and

ceftazidime 2g 8 hourly were initiated. The infected pacemaker pocket area was explored, irrigated and resutured during the fourth day of hospitalization. As the second pus swab from the wound site still grew *Pseudomonas aeruginosa*, IV ceftazidime was changed to IV Tazodin® 4.5gm 8 hourly. After 26 days of IV cloxacillin, 14 days of IV ceftazidime and 11 days of IV Tazodin®, she was discharged home with 1 week course of oral cloxacillin and ciprofloxacin as she was already clinically afebrile with C-Reactive Protein (CRP) 6mg/L and white cell count $4.38 \times 10^9/L$. 3 weeks after the second hospital discharge, she became feverish again and the pacemaker pocket site was still tender but with no pus discharge. Unfortunately, CRP increased to 96mg/L. She was treated with one week oral Augmentin® 625mg 12 hourly and cloxacillin 1g 6 hourly. Four months after pacemaker implant, she presented to the emergency department with atypical chest pain, cough and fever. The chest radiograph showed right middle lobe consolidation and multiple abscesses. Blood culture grew *Staph. aureus*. ECHO showed mild tricuspid regurgitation, and vegetations on the tricuspid valve and pacemaker leads. IV Cloxacillin 2g 4 hourly was initiated.

She underwent open heart surgery during this third hospitalization whereby modified De Vega tricuspid annuloplasty, tricuspid valve repair with artificial chordae reimplantation, excision of vegetation from anterior leaflet of tricuspid valve, explantation of the whole permanent Sensia® (Model SESR01, Medtronic, Inc., Minneapolis, MN, USA) pacing system and insertion of temporary pacing wire were performed. Postoperatively, she completed another 2 weeks of IV cloxacillin. 18 days after the surgery, single chamber rate responsive Sensia® (Model SESR01, Medtronic, Inc., Minneapolis, MN, USA) was implanted on the opposite site. Upon discharge, repeated blood culture yielded no growth and inflammatory markers were not raised. 30 months after the surgery, her pacemaker parameters were normal and she remained well.

DISCUSSION

IE is not easily diagnosed solely just from history, clinical, blood or imaging tests. Hence, a set of criteria called the modified Duke criteria¹ has been developed to guide the diagnosis of IE using a combination of major and minor criteria. Intravenous drug use, prosthetic heart valves, structural heart disease such as rheumatic valvular heart disease and ventricular septal defect, hemodialysis,

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immunocompromised status and prior infective endocarditis episode have been identified as predisposing risk factors for IE. A pacemaker is a foreign body and can potentially become a nidus for the formation of IE.

Generator exchange^{2,4} has been identified as a risk factor for pacemaker-related infection and there was no evidence so far to suggest that downgrading of dual chamber to single chamber generator with capped atrial lead per se would lead to higher risk of infection. Pacemaker-related infection usually starts with generator pocket infection due to perioperative contamination of the surgical field with skin flora and may spread deeper along the lead and forms vegetations on the lead and valve. The most commonly implicated pathogens are *Staphylococcus* species^{2,3} in CIED infection.

Hence, it is imperative that the appropriate prophylactic antibiotic of choice must have anti-staphylococcal property such as cephazolin⁵ to reduce the risk of pacemaker infection. Based on de Oliveira JC *et.al*⁵, the American Heart Association(AHA) in 2010² recommended cephazolin as the prophylactic antibiotic of choice. Cefoperazone is a third generation cephalosporin antibiotic with less activity against gram-positive organisms. Generator change procedure and inappropriate prophylactic antibiotic could have contributed to the initial pocket infection. At that stage, the options would be watchful waiting with antibiotic coverage and hope for full infection eradication or explantation of the whole pacemaker system. The initial finding of *Pseudomonas* organisms suggested polymicrobial or mixed infection with *Staphylococcal* organism. The mainstay of anti-staphylococcal cloxacillin with antipseudomonal ceftazidime and Tazosin® were appropriate to cover for possible mixed infection. The inadequate duration of cloxacillin and the retained hardware caused it to be partially treated. The smoldering pocket infection later relapsed, progressed and became right sided endocarditis with pulmonary emboli.

An infected pacemaker system with full blown *Staphylococcal* bacteraemia must be removed surgically or percutaneously. Removal of the lead with direct

traction,telescoping sheath and excimer laser have been attempted. These percutaneous techniques may dislodge large vegetations, causing embolisation and infarction to the lung. Thus, open heart surgery was more appropriate to remove the whole infected pacemaker system, debride the vegetations on the valves and offered the option to repair or replace the damaged valve. As the patient was pacemaker dependent, temporary pacing would buy some time for control of infection. Optimal timing for reimplantation can be difficult and not well defined in the literature². If the infection was well controlled with adequate duration of proper antibiotic and the patient was pacemaker dependent, reimplantation after 2 weeks of surgery and antibiotic duration seemed sensible. After surgery and pacemaker implantation, the patient ought to be follow up to detect any recurrence of infection.

The first step in reducing CIED infection is scrupulous sterile implantation technique. Any pacemaker-related infection must be detected early and treated with the most appropriate antibiotic and duration to avoid the dreaded extreme complications such as highlighted in this case. Early explantation should be considered in established CIED infection.

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