Pacemaker Endocarditis Caused by *Pseudomonas aeruginosa* Treated Successfully


**Abstract**

Infective endocarditis (IE) is a rare but serious complication of permanent cardiac pacemaker placement. Endocarditis in the presence of prosthetic valves and pacemakers is usually due to staphylococci. We present a case of pacemaker endocarditis caused by *Pseudomonas aeruginosa* that was successfully treated with a combination of antimicrobial therapy and percutaneous removal of the colonized lead.

**INTRODUCTION**

The diagnosis and management of infective endocarditis (IE) poses major challenge even to experienced clinicians. Although heart valves are most commonly affected, the disease may also occur on septal defects, mural endocardium, prosthetic valves and other intravascular devices. A plethora of microorganisms has been implicated in IE, but streptococci and staphylococci account for 80-90% of cases. The increasing use of prosthetic valves and intravascular devices and poor infection control practices have resulted in increased incidence of endocarditis caused by indwelling devices by Gram negative organisms. As such devices are inherently more susceptible to bacterial colonization, infection established upon them is often difficult to eradicate, necessitating their removal and replacement.

**CASE REPORT**

A 50 years male had a permanent pacemaker inserted in November 2000 for treatment of a symptomatic bi-fascicular block. Two weeks later, he developed pacemaker pocket infection that improved with oral antimicrobials. In March 2001, he had 10 days of high fever with *Pseudomonas* bacteremia for which he received intravenous ceftazidime and amikacin for two weeks. A complete removal of the pacemaker lead wire was not possible then as the pacemaker lead tip was embedded deeply in the right ventricular myocardium. The generator however was removed completely and fever subsided.

As fever recurred despite this therapy that lasted for five months, he sought referral to our medical centre in August 2001. On examination, he had an oral temperature of 103˚F and a normal general examination with no peripheral (vascular or immunologic) manifestations of IE. There was no palpable hepatosplenomegaly. Examination of the cardiovascular, respiratory, and neurological systems including optic fundi were normal.

Investigations revealed haemoglobin of 9.4 gm%, total leukocyte count 11,100/cumm with a differential of neutrophils 92%, band forms 3%, eosinophils 1%, and lymphocytes 4%, elevated erythrocyte sedimentation rate (ESR) (65 mm at one hour), urine showing numerous RBCs, 4+ urine albumin, and a 24 hour urinary protein of 10 gm, and elevated creatinine (3.2 mg%). Trans-thoracic echocardiography (TTE) showed the tip of the pacemaker lead wire embedded in the right ventricular myocardium and the proximal cut-end floating freely in the pulmonary trunk but no intramural or valvular vegetations.

All of five blood cultures drawn at separate intervals within a 24-hour period grew *Pseudomonas aeruginosa* that was susceptible to amikacin, gentamicin, ciprofloxacin and ceftazidime.

A diagnosis of pacemaker lead wire infection was made based on the following evidence - prolonged fever occurring in a patient with an indwelling intracardiac foreign device, *P. aeruginosa* bacteremia, presence of intracardiac foreign device, and immune-complex glomerulonephritis.

Intravenous antibiotics, ceftazidime (1 gm q8h) and gentamicin (1 mg/kg q8h) was started and the pacemaker lead wire was successfully removed percutaneous using a snare introduced through a 14-French sheath placed in the right internal jugular vein within a few days. The removed pacemaker lead wire on culture yielded *P. aeruginosa* with the same antibiogram as well. Fever defervesced by day 10 and the patient was discharged afebrile after four weeks of
The commonest type of pacemaker infection is restricted to the generator unit and the lead wires in their subcutaneous position. These infections commonly arise in close proximity to the pacemaker placement. S. aureus and S. epidermidis are isolated most often (63-75%) from generator pocket infections. Streptococci, Corynebacterium, Propionibacterium, Candida spp., and Enterobacteriaceae are the other pathogens reported to cause generator pocket infections.

Infection of the intravascular portion of the transvenous electrode, which occurs primarily at an intracardiac site, most commonly arises from infection of the subcutaneous portion of the pacing apparatus that has tracked intravascularly. Among patients with pacemaker endocarditis, up to a third are symptomatic within 6-12 of the procedure with fever and chills as the most common symptoms. However, a fever of low grade or failure to thrive may be the only symptom in 25-35%, and septic shock may be the initial presentation in 9%. Pulmonary symptoms consistent with pneumonia, bronchitis, lung abscess, or embolism are seen in 20-45%, and with a more common occurrence among those who have a later onset of infection. New or changing heart murmurs and splenomegaly are uncommon. An elevated ESR and leucocytosis are uncommon. Trans-esophageal echocardiogram has demonstrated vegetations on the electrodes, tricuspid valve, or ventricular endocardium in 90-96% of patients studied. In contrast, TTE has been diagnostic or suggestive of endocarditis in only 22-54% of patients.

Although staphylococci [coagulase negative staphylococci (56%), S. aureus (27%)] is the commonest cause, other microorganisms that can cause pacemaker endocarditis include Enterobacteriaceae (6%), P. aeruginosa (3%), C. albicans (2%) and enterococci, Corynebacterium, Listeria, streptococci, and Aspergillus spp are rare (each < 1%).

_Pseudomonas_ species causing right-sided endocarditis is mostly seen in IV drug users-afflicting the structurally normal heart valves and is associated with septic pulmonary embolism and right heart failure. Left-sided valvular _Pseudomonas_ endocarditis is associated with systemic embolization that is relatively refractory to therapy and less common. _P. aeruginosa_ is however reported only rarely to cause pacemaker endocarditis.

While treating pacemaker infections, in patients with bacteremic pacemaker endocarditis when pacemakers are in-situ, consensus approach favor the removal of the generator and the electrode(s) as an integral part of the treatment, in addition to the use of appropriate antibiotics. In a large review of nine retrospective series that included 190 cases of pacemaker endocarditis, the mortality rate in those treated with antibiotics was 41% compared to 9% among those treated with antibiotics plus removal of the entire pacing system. In the treatment of initial and relapsed episodes of pacemaker endocarditis, Camus and associates, noted cure and survival in 91% (10 of 11 patients) in whom the electrodes were removed, compared with 9% cure (1 in 11) in patients where the electrode was not removed.

In summary, accurate identification of the microbial etiology, prompt antimicrobial therapy and removal of the infected lead contributed to a successful outcome in our patient with a rare complication of permanent pacemaker insertion.