A Case of Pacemaker Endocarditis Caused by Aerococcus urinae

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Background. Aerococcus urinae has lately been acknowledged as a cause of infective endocarditis (IE) especially in older males with underlying urinary tract disorders. In this population, cardiac implanted electronic devices (CIED) are not uncommon, but despite the capacity of A. urinae to form biofilm in vitro, no cases of aerococcal CIED infections have been reported to date. Case Presentation. An 84-year-old male with pacemaker was admitted with dysuria one month after a transurethral procedure for urinary bladder cancer. A. urinae was isolated from urine and blood. Transesophageal echocardiography (TEE) was without signs of vegetation on valves or pacing cables. The patient was treated with a twelve-day course of β-lactam antibiotics. Forty days after the initial admission, the patient was readmitted due to malaise, general pain of the joints, chills, and renewed blood cultures grew A. urinae. TEE demonstrated a 10 × 5 mm vegetation on either the tricuspid valve or one of the pacing cables. The pacemaker system was completely removed and demonstrated macroscopic signs of infection. A temporary system was implanted, and after 14 days of penicillin G treatment, a new system permanent system was implanted. Total treatment time was 40 days. Recovery was uneventful. Conclusion. This report demonstrates that A. urinae can cause CIED infection. In patients with A. urinae bacteremia and a CIED, this risk must be considered, especially if bacteremia reoccurs.

1. Introduction

Aerococcus urinae is a Gram-positive bacterium which can cause urinary tract infections (UTIs), bacteremia, and infective endocarditis (IE) [1–3]. After the introduction of MALDI-TOF MS for species determination in medical microbiology, A. urinae has been increasingly identified as a relatively common cause of both UTIs and bacteremia [4, 5]. Many cases of IE caused by A. urinae have been reported [6], but in a larger case series, this complication to bacteremia was found to occur only in around 10% of cases [5]. IE caused by A. urinae mainly affects older males with underlying urinary tract conditions, and the prognosis is relatively favourable despite the advanced age and many comorbidities of the patients [3, 7]. A. urinae has been described to induce aggregation of human platelets in vitro and is able to form biofilms on plastic surfaces, two potential mechanisms of importance for the pathogenesis of IE [8, 9]. A particular form of IE is an infection where bacteria adhere to the leads of transvenously coronary implanted electronic devices (CIED), such as a pacemaker. In this type of infection, adherence of bacteria to the foreign material and a subsequent formation of biofilm are believed to be important [10]. CIED infections are treated with antibiotics and device removal, a procedure not without risks. CIED infection with A. urinae has to our knowledge not been reported previously.

2. Case Presentation

An 84-year-old male presented with a medical history of sarcoma in 1999 located on the right flank (treated with surgical removal, chemotherapy, and radiation), ischemic heart disease, atrial fibrillation, and a pacemaker (St. Jude 5816,
atrial lead Medtronic 4076, and ventricular lead Medtronic 4074) since 2010 due to sick sinus syndrome and AV block II Mobitz type 2. Three months prior to the present episode, he was diagnosed with urinary bladder cancer (pTaG1), and one month prior to the present admission, he underwent a diagnostic transurethral resection of bladder tissue (TUR-B).

On admission, the patient reported malaise and dysuria since the TUR-B. He was afebrile and respiratory and circulatory stable and had macroscopic haematuria, C-reactive protein (CRP) of 204 mg/L, and leukocytes of $12 \times 10^9$/L. After cultures had been taken from blood and urine, he was admitted to the Infectious Diseases Clinic. On the third day, blood (4/4 bottles) and urine cultures grew A. urinae ($>10^5$ cfu/mL). Species determination was performed with MALDI-TOF MS (Bruker Microflex) with a score of 2.23. Antibiotic treatment with ampicillin $2 \times 3$ was initiated. Transesophageal echocardiography (TEE) was performed, and no signs of vegetation on valves or pacing leads could be demonstrated. After five days, the patient’s condition had improved, CRP was 124 mg/L, and he was discharged with amoxicillin 500 mg three times daily for another seven days.

Forty days after the initial admission, the patient was readmitted due to a two-week history of malaise and general pain of the joints. Two days before, he experienced chills but was on admission afebrile and respiratory and circulatory stable, CRP was 250 mg/L, and leukocytes were $21 \times 10^9$/L. Treatment with intravenous penicillin G 3 g four times daily was initiated, and renewed blood cultures grew A. urinae (6/6 bottles), whereas the urine culture was negative. Minimum inhibitory concentrations were 0.008 mg/L for penicillin G, 0.25 mg/L for ceftriaxone, and 256 mg/L for tobramycin. A renewed TEE demonstrated a 10 $\times$ 5 mm vegetation on either the tricuspid valve or one of the pacing leads (Figure 1). After 19 days of treatment using penicillin G 3 g four times daily, the patient was referred to a tertiary centre for pacemaker system extraction. The extraction was performed using transvenous techniques, the device and leads were completely removed without complications, and a temporary VVI pacemaker was implanted on the ipsilateral side. The subsequent delay to extraction was due to a regional limitation in the capacity for device extraction. Lead extraction was without complications and a temporary pacemaker on the ipsilateral side was in place for two weeks before a new permanent pacemaker was implanted.

IE with A. urinae is typically treated with a β-lactam antibiotic, and we choose penicillin to which the bacterium had very low MIC [11]. Aminoglycosides are often added to the treatment despite that synergism between β-lactams and aminoglycosides is not always present [7, 11, 12]. In our case, MIC towards aminoglycosides was high, and cure of the patient was dependent on lead extraction rather than on antibiotic treatment. Therefore, aminoglycosides were not given.

The patient described here is prototypic for invasive infections with A. urinae. He is male, above 80 years old, and has urinary bladder cancer, all factors associated with A. urinae bacteremia [3, 5, 13]. In the first episode, the urinary tract was the likely focus of infection demonstrated by both the symptoms and bacteriuria of the patient. In the recurring episode, however, the focus of infection was rather the pacemaker system demonstrated by the vegetations on leads and the lack of bacteriuria. Cultures from the tip remained negative, but after a long course of antibiotic treatment this was the expected. In this case, the infection was macroscopically evident, but in less evident cases, 16S rRNA gene PCR and sequencing of material from leads might provide additional information.

The incidence of bacteremia with A. urinae has been estimated to be 1.4 per 100,000 inhabitants per year [5] and is certainly much higher among older people. The prevalence of CIED in the elderly population, at risk for A. urinae bacteremia, is not insignificant. Given the lack of reports about A. urinae CIED infection and the presumed significant number of episodes of A. urinae bacteremia in CIED carriers, the risk for CIED infection in A. urinae bacteremia is likely to be relatively low.

This case report demonstrates that A. urinae can cause CIED infection. In patients with A. urinae bacteremia and a CIED, this risk must be considered, especially if bacteremia recurs.

Figure 1: TEE demonstrating the lead (long arrow) and the vegetation (short arrow) attached to the lead.
Consent
Written informed consent was obtained from the patient for publication of this case report.

Conflicts of Interest
The authors declare that they have no conflicts of interest.

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