Pacemaker Lead Endocarditis Due to *Trichosporon* Species

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Abstract

Pacemaker-related fungal endocarditis is an uncommon and unexpected complication. It is associated with high mortality rates. Due to nonspecific clinical symptoms, negative blood culture and delays in obtaining appropriate imaging studies; late diagnosis is common with fungal endocarditis. Hereby we are reporting a rare case of pacemaker lead endocarditis due to *Trichosporon* species. In literature we did not find any case of pacemaker-related endocarditis due to *Trichosporon* species.

Case Report

A 57 year old lady with known history of hypertension, diabetes mellitus and bronchial asthma was admitted with one month history of low grade fever with cough, shortness of breath and bilateral pedal edema. She had history of symptomatic sinus node dysfunction with repeated syncopal attacks for which she underwent single chamber pacemaker implantation 16 years ago, which was changed to dual chamber pacemaker 3 years ago in contralateral site, without the removal the pre-existing intracavitary lead. One month prior to admission she developed low grade fever with cough and shortness of breath, for which she consulted a physician and oral antibiotic therapy was initiated. No peripheral signs of infective endocarditis was observed. Otherwise physical examination was unremarkable. Laboratory analysis showed Hb 7.2 gm/dl, neutrophilic leukocytosis, random blood sugar 154 mg/dl, blood urea 58 mg/dl, serum creatinine 1.1 mg/dl. Urine and 3 sets of blood samples sent for culture were negative for bacterial growth. ECG showed normal functioning pacemaker rhythm.

Chest X-ray showed two cardiac pacemaker leads, mild cardiomegaly and clear lung fields. Ultrasound of whole abdomen showed mild hepatomegaly with uniform echotexture. Transthoracic echo cardiography (TTE) demonstrated no regional wall motion abnormality with good left ventricular systolic function and grade I diastolic dysfunction, right atrium (RA) and right ventricle (RV) were mildly dilated, pulmonary artery pressure was 40 mmHg, pacemaker leads in RA and RV. Tip of lead in RV showed large (2.35x1.49 cm) vegetation with irregular outline, all valves were normal (Figure 1).

Patient was advised for surgical removal of existing hardware and medical management. But she denied surgical removal. After hospital admission intravenous vancomycin, piperacillin and tazobactum. Subsequently within 3 days her renal function deteriorated with thrombocytopenia. By this time blood culture (two out of three sample) showed fungal growth of *Trichosporon* species. Then therapy was switched to injection voriconazole 200 mg twice daily and vancomycin was stopped. Consultation was taken from nephrologist. She was diagnosed as a case of thrombotic microangiopathy with acute renal failure. Dialysis and plasmapheresis was done on the next day and second sitting of dialysis was done on that night. The patient attendants refused further hospital stay, hence injection voriconazole was changed to oral form of 200 mg twice daily and she was discharged. After two week she came for follow up in cardiology outpatient department, that time she was afebrile, BP...
was 130/70 mm of Hg, serum creatinine 9.0, hemoglobin 9.7gm/dl, and platelet count 160000/mm³. She has been on dialysis once a week since her discharge from the hospital. The repeat transthoracic echocardiogram done showed the vegetation size had increased from 2.35x1.49 cm to 2.54x2.36 cm (Figure 2), RV size (42 mm) also increased and PASP (pulmonary artery systolic pressure) 56 mm of Hg.

We advised her again to get admitted in the hospital, to remove the infected pacemaker generator and leads, but she denied surgical intervention and hospital admission.

Discussion

Infections of pacemaker systems have been described in 7% to 15% of patients. Among the cause of infective endocarditis fungal organisms account for only 1% to 10% of cases. Bacterial infections with *Staphylococcus* species constitute the majority of pacemaker lead endocarditis but fungal etiologies comprise an important subgroup. To our knowledge, there are only few reported cases of *Candida albicans* and *Aspergillus fumigatus*. But no case of pacemaker-related infective endocarditis been reported. *Trichosporon* species are fungi that commonly inhabit the soil. They colonize in the skin and gastrointestinal tract of humans. Long known as the cause of superficial infections such as white piedra, a distal infection of the hair shaft, the genus is now the second most commonly reported cause of disseminated yeast infections in humans. The genus *Trichosporon* was once regarded as a single species, *Trichosporon beigeli*. However, more recently, *T. beigeli* has been divided into distinct species, at least 9 potential to cause human disease have been identified. *Trichosporon* species are increasingly recognized as a cause of systemic illness in immunocompromised patients. Hematologic malignancies are the best-described risk factors for trichosporonosis, accounting for 63% of reported cases. Additional risk factors include corticosteroid use, hemochromatosis, other deficiencies of granulocyte function, and end-stage renal disease, solid tumors, HIV/AIDS, and intravascular devices, including catheters and prosthetic heart valves. The diagnosis of trichosporonosis is usually confirmed by a positive blood culture result obtained in the evaluation of a febrile (typically neutropenic) patient. Urine cultures may be the first to grow *Trichosporon* in the setting of disseminated disease. Endocarditis is rarely reported and all were prosthetic valve related, but is associated with high mortality rate (82% in a single series). Among the antifungal drugs, the newer triazoles (eg, voriconazole, posaconazole, ravuconazole) has shown excellent in vitro activity against *Trichosporon*. In particular, Voriconazole seems to have better in vitro activity than Amphotericin B. Indeed, successful clearance of fungemia with voriconazole has been reported when liposomal amphotericin B treatment was failing. Early diagnosis of fungal endocarditis is often elusive because blood cultures are commonly negative (because of delayed growth) and classic physical findings are rare. Overall mortality rate of fungal endocarditis range from 50% to 94% despite of aggressive surgical and antimycotic therapy. Although very few cases of fungal endocarditis have been effectively treated with antimycotic therapy alone, combined medical therapy and surgical removal of infected valve, pacemaker lead and accompanying hardware is the mainstay of treatment. Regarding pacemaker lead endocarditis most authors advocate thoracotomy when vegetations exceed 1.0 cm in size, as the risk of embolization is high with intravascular traction methods. Lifelong antifungal
prophylaxis has been advocated in numerous series, as late recurrent fungal endocarditis is common. In our case, we planned for combined medical and surgical treatment for this patient but the patient denied for surgical intervention. She is now on anti-fungal treatment with voriconazole 200 mg twice daily along with her routine antihypertensive and antidiabetic medications. Patient is still following up.

Conclusions

Pacemaker lead endocarditis itself is a rare complication especially with fungus species, so late diagnosis is common. As the outcome of pacemaker related fungal endocarditis is very poor, a high index of clinical suspicion for fungal endocarditis is required in patients with transvenous pacemaker and fever of an undetermined cause. When blood cultures for microorganisms became negative then suspicion of fungal endocarditis must be considered. Serial echocardiography, especially transoesophageal echocardiography is very important for early detection of vegetations. Appropriate antimycotic therapy along with surgical removal of all the hardware is the mainstay of successful treatment of pacemaker related fungal (Trichosporon) species.

References