Case Report

Klebsiella endocarditis in the early post-operative period after renal transplantation

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Introduction

Infective complications are among the leading causes of morbidity and mortality among renal transplant recipients. The risk of infection depends on the state of immunosuppression and other predisposing factors. Infective endocarditis has been reported as a rare complication in renal transplant recipients [1,2]. The majority of patients had aortic valve and prosthetic valve endocarditis [3–6]. Here we report a patient with acute bacterial endocarditis of the mitral valve 1 month after renal transplantation.

Case

A 37-year-old male patient who was hypertensive for 6 years developed end-stage renal disease and, after 2 months of haemodialysis using an arterio-venous fistula, underwent a live related renal transplantation in June 1997. He was on a triple immunosuppressive regimen with cyclosporin 150 mg twice daily, azathioprine 150 mg and prednisolone 60 mg daily along with trimethoprim (160 mg) and sulfamethoxasole (800 mg) once a day for prophylaxis against Pneumocystis carinii. He developed an acute rejection episode at the end of the first week after transplantation, which was treated successfully with three 1 g pulses of methylprednisolone. Four days after the rejection episode, he developed a urinary tract infection with Klebsiella pneumoniae which was treated with oral ciprofloxacin 500 mg twice daily for 7 days.

Three weeks after the transplant, the patient developed a wound dehiscence, and the chemistry of the fluid was consistent with extracellular fluid. One month after the transplant, he developed high grade fever with chills and leucopenia with the total white blood cell (WBC) counts declining to 1000/mm³ which necessitated the withdrawal of azathioprine and institution of barrier nursing. Physical examination revealed a grade 3/5 ejection systolic murmur at the left sternal border and haemorrhages in the optic fundus of the left eye. Blood cultures grew K. pneumoniae, which was sensitive to ceftazidime. The patient was started on ceftazidime 1 g twice a day as well as oral fluconazole 100 mg a day for oral thrush.

Investigations were as follows. Haemoglobin 7.1 g/dl; WBC counts 0.8 × 10^9/µl and 0.5 × 10^9/µl during the course of the illness; platelets 161 × 10^9/µl; BUN and serum creatinine were 17.3 µmol/l and 233 µmol/l respectively; sodium 135 mmol/l, potassium 3.6 mmol/l, chlorides 99 mmol/l and bicarbonates 22 mmol/l; ALT 171 IU/l, AST 51 IU/l; total protein 62 g/l, albumin 3 g/dl, bilirubin 29 µmol/l.

He was HbsAg, HCV and HIV negative. Cytomegalovirus IgM titres were normal. C3 and C4 levels were 900 mg/l (normal: 700–2000 mg/l) and 350 mg/l (normal: 50–500 mg/l) respectively. Rheumatoid factor was negative.

A pre-operative echocardiogram showed concentric left ventricular hypertrophy. An echocardiogram done during the febrile episode showed small vegetations on the anterior leaflet of the mitral valve, with a small pericardial effusion with normal ventricular function. Chest X-ray showed multiple non-homogenous opacities in both lung fields. Ultrasound showed the transplant kidney measuring 10.1 × 5.2 cm, with no evidence of any perinephric collection of mild splenomegaly.

The patient became afebrile 4 days after starting ceftazidime, which was continued for 4 weeks. A renal biopsy was deferred in view of the wound dehiscence. An echocardiogram was repeated 4 weeks later and it showed no evidence of any vegetations. An ophthalmologic examination of the optic fundus showed clearing of the haemorrhagic areas. On follow up 4 months after renal transplantation, his serum creatinine was 1.8 mg/dl. The patient developed an asymptomatic...
urinary tract infection with *K. pneumoniae* and was treated with appropriate antibiotics.

**Discussion**

Infections occur in 26–32% of renal transplant patients [7]. They are common causes of morbidity and mortality, and bacterial infections are the most common pathogens. This is due to marked impairment of cellular immunity while on immunosuppressive therapy as in organ transplantation.

However, as the population of transplant recipients continues to grow, infective complications including perhaps bacterial endocarditis will increasingly be detected. Gram-negative bacilli account for 7–15% of all cases of infective endocarditis, of which *K. pneumoniae* has been implicated in 9% of cases [8,9]. The mortality rates in these patients can be high as 83%, and in the post-transplant setting this can be much greater [9]. In previous reports on endocarditis in post-transplant patients, the organisms isolated were Cornybacteriaceae and *Nocardia asteroides*, and fungi such as *Aspergillus fumigatus* and *Exophilla castellani* [3–5,10]. Endocarditis in these patients followed odontectomy, subcutaneous infection with *Exophilla castellani*, skin nodule due to *N. asteroides* and in an HIV-infected recipient who received a kidney from an intravenous drug user [3,4,6].

Urinary tract infections occur in 30–80% of renal transplant recipients [11]. Only 5% of catheter-associated bacteruria will be identified with bacteremia due to the organisms in urine [12]. We believe that in our patient the urinary tract infection with *K. pneumoniae* and severe bone marrow suppression created the ideal milieu for dissemination of the septic process to the mitral valve and deterioration of allograft function which improved with appropriate antibiotic therapy. To our knowledge, this is the first report of *K. pneumoniae* endocarditis in a renal transplant recipient with successful outcome. In some of the patients, combined surgical therapy including valve replacement along with appropriate antibiotics was necessary to cure the infection [7].

The therapy for bacterial endocarditis should be guided by appropriate sensitivity testing, adequate dosage and duration for a complete cure. Under these circumstances, one should consider lowering the immunosuppressive medications especially in the presence of serious infections.

In conclusion we have presented the case of an unusual patient with urinary tract infection due to *K. pneumoniae* who developed severe bone marrow suppression leading on to infective endocarditis which was diagnosed and treated successfully.

**References**


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