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Infective Endocarditis: A Frequent Disease in Chronic Kidney Disease Patients

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Abstract: Infective endocarditis (IE) is the one of the most important causes of increased mortality and morbidity among haemodialysis patients. The reason for this increasing prevalence of infection among these patients is the use of haemodialysis catheters during dialysis, as these patients are highly susceptible to infections that are easily transmitted via blood access points. In our case report, a patient of chronic kidney disease presented with pyrexia of unknows origin was found to have Infective endocarditis. After 21 days of aggressive treatment with antibiotics, patients were sent home successfully. Strict hygiene, cleaning the site and sterile techniques when accessing arteriovenous fistulas or vascular catheters can minimize the potential for infection.
Keywords: Infective endocarditis, Haemodialysis, Chronic kidney disease.

INTRODUCTION

The end-stage renal disease (ESRD) population is increasing rapidly.Infective endocarditis (IE) in patients receiving HD has been reported for the first time in 1966[1]. Dialysis catheter-related infection is a major cause of morbidity and mortality in patients on dialysis.

Patients on chronic hemodialysis (HD) are particularly vulnerable to bacteremia and to infective endocarditis (IE) due, on one hand, to the vascular access needed for dialysis (arteriovenous fistula or central catheter) and, on the other hand, increased susceptibility to infection caused by end-stage renal failure.

Antibiotic treatment should take into consideration the specificity of endocardial damage, its kinetics during the inter-dialysis and the dialysis periods and its toxicity. The seriousness of the disease makes it mandatory to choose the best antibiotic to minimize toxicity without compromising the efficacy of treatment. Also, a specific preventive measure must be implemented.

The purpose of our study is to evaluate pyrexia of unknown origin in chronic kidney disease.

CASE REPORT

A 34-year-old male patient was admitted to hospital two days after his scheduled haemodialysis session. Patient presented with a high grade fever (39.2°C) associated with chills and rigor, severe recurrent non-productive cough and shortness of breath. During admission, his blood pressure was 180/90 mmHg with a pulse rate of 120 beats/min and a blood sugar level of 11 mmol/L.

Premorbidly, he had ESRD secondary to Hypertension nephropathy. He had been on regular haemodialysis for the past ten years.

Physical examination revealed presence of bilateral lower limb swelling and abdominal ascites. A respiratory examination revealed bibasal fine crepitations. First (S1) and second (S2) heart sounds were heard with systolic murmurs and hyperdynamic apex beat.

Renal function profile was as follows: serum creatinine (Cr): 560 µmol/L, creatinine clearance (CrCl): 6.92 ml/min, and urea: 11.3 mmol/L. His hemoglobin (Hb) was 6.0 g/dL, hematocrit (Hct): 22.6%, and white blood cell (WBC) count was 11.63 x 109/L.

An echocardiography revealed two vegetations.First mass at sub aortic region 0f 2.44 sqcm and second pedunculate mass of 1.24 sqcm at AML oscillating through mitral orifice. It also revealed patent foramen ovale with left to right shunt. Mild AS with grade 2 AR with grade 1 TR also seen.

Culture and sensitivity test was carried out to confirm the causative agent. In the meantime, empirical treatment for IE was initiated by administration of intravenous (I.V.) ceftazidime 1 g once daily (cephalosporin) and I.V. cloxacillin 2 g four times daily.

For the management of comorbidities, other medications were prescribed.

On the second day of admission, patient still complained of persistent fever. Further, due to low hemoglobin level, tablet folate 5 mg OD and injectable iron 200 mg OD were added to patients' medical chart for treatment of anaemia. Tablet nifedipine 20 mg OD was added to his current regime for hypertension and tablet cardipin 100 mg OD was added for prophylactic management of stroke. At the end of day 2, results of the culture and sensitivity test showed Enterococcus faecalis at the central catheter and coagulase negative staphylococcus at the peripheral line as the causative agents of infection. Empirical antibiotics were then switched to intravenous penicillin V potassium 2.4 megaunit 4 hourly and intravenous cloxacillin 2 g four times daily for the coverage of gram positive cocci that caused infection.

Despite being on nifedipine (a calcium channel blocker), patient was still hypertensive, therefore another antihypertensive agent, tablet metaprolol (a beta blocker) 75 mg BD, was added to his treatment regime.

DISCUSSION

While Sir William Osler is credited for his initial clinical description of IE, Blagg and his associates [2] were the first to report this complication in HD patients. According to Friedberg [3] the most important factor in the diagnosis of endocarditis is the physician's index of suspicion. This is especially true in patients receiving regular dialysis, whose clinical manifestations may be misleading.

Various factors increase the susceptibility to infection in patients on HD. It may be the causative factors such as diabetes, lupus and hematological diseases, immunosuppressive treatment and malnutrition. Additionally, there is suppressed immune response (decrease of B and T cell response to antigenic or mitogen stimuli and functional abnormalities of monocytes/ macrophages and neutrophils) in patients on HD, as shown by Goldman in 1990 [4].

Clinical symptoms of IE are not typical, especially in HD patients. Therefore, the physicians responsible for the care of these patients have a difficult task and must, indeed, maintain a high index of suspicion if endocarditis is to be detected [5]. In our study patients, symptoms were dominated by the association of fever and systolic heart murmur, especially of mitral and aortic valves and signs of congestive heart failure, explaining the repeated episodes of APO. Although none of our patients had any neurological manifestations, the presences of central nervous system symptoms provide additional diagnostic clues [6]. Peripheral embolic phenomena are not pathognomonic of endocarditis [7, 8], although their presence surely directs the physician's attention to this entity.

The endocardial lesions have an anatomical specificity, which dictates the requirements of the antibiotic. It necessitates the use of a combination of two bactericidal antibiotics with a synergistic action. The antibiotic of choice is decided by the culture and sensitivity reports. It is necessary to get a good penetration of the antibiotic to the site of infection and, therefore, administration of intravenous antibiotics whose protein binding is low and whose diffusion is maximal on the vegetation is required. The antibiotics should be administered for four to six weeks [9,10].

CONCLUSION

End stage renal patients are prone to various types of infections. Presence of co-morbidities and issues related to dose adjustment based on creatinine clearance creates difficulties for the physician for designing an appropriate therapeutic regimen for the patient. IE is one such infection in ESRD patients that is associated with high mortality. Goal of therapy is to manage comorbidities and infection by provision of appropriate treatment based on close monitoring of renal function and electrolytes.

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