Case Report

Salmonella pericarditis and pericardial effusion in a patient with systemic lupus erythematosus on haemodialysis

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Introduction

Infection in patients on maintenance dialysis therapy remains a common cause of mortality and morbidity. Pericarditis with pericardial effusion is a recognized complication of systemic lupus erythematosus (SLE) and uraemia, but it also occurs in patients on dialysis. Dialysis-associated pericarditis could be a manifestation of inadequate dialytic therapy, hypercatabolism, or viral infection. Although salmonella bacteremia is not rare in SLE patients, salmonella pericarditis has been reported only once in an adult patient with SLE who was not on haemodialysis. We report a case of a young girl with SLE on maintenance haemodialysis who developed salmonella pericarditis with pericardial effusion and adhesions requiring surgical drainage. We are not aware of salmonella pericarditis reported in dialysis patients.

Case report

A 16-year-old girl with renal failure secondary to SLE has been on regular haemodialysis three times per week for the last 2 years. The dialysis was adequate with Kt/V of 1.24 and the patient was on erythropoietin therapy. Her lupus was inactive and she was stable on haemodialysis; however, she is on a daily dose of 10 mg prednisone. In February 1996 she came to the Emergency Room with shortness of breath, fatigue, fever and joint pains of 2 weeks duration. On examination she was not in distress. Blood pressure 140/100 mmHg, pulse rate 90 beats per minute, respiratory rate 24 per minute and temperature of 38.0°C. Heart sounds were faint but no pericardial friction rub. The rest of the examination was unremarkable.

Laboratory studies revealed haemoglobin of 12 g/l, WBC 4100/mm³, sodium 134 mmol/l, potassium 6.5 mmol/l, calcium 2.5 mmol/l, phosphate 2.17 mmol/l, creatinine 839 μmol/l, albumin 37 g/l, and normal liver enzymes. The electrocardiogram was normal.

On the day of admission the echocardiogram showed a moderate pericardial effusion with no evidence of tamponade. Pericardiocentesis was carried out and 600 ml of haemorrhagic fluid was drained. A pig-tail draining catheter was left in the pericardial space. The patient was started empirically on ceftazidime. Salmonella group B was cultured both from blood and pericardial fluid. Treatment was modified, with intravenous ciprofloxacin 400 mg daily replacing ceftazidime on hospital day 4, and the haemodialysis frequency was increased, but she remained sick and febrile. A repeat echocardiogram showed pericardial effusion with two pockets, one posterior to the left ventricle and the other cranial to the right atrium. Percutaneous drain was clogged and surgical drainage with pericardiectomy was performed on hospital day 8. Cultures from the pericardial tissue grew Salmonella group B again.

During the next week, the patient was still unwell and febrile in spite of continued intravenous ciprofloxacin to which the isolated salmonella was sensitive. Computed tomography scan of the chest with contrast revealed a small collection of fluid in front of the heart at the level of the aortic root and main pulmonary artery. The pericardium was re-explored on hospital day 14 and the loculated area drained. Salmonella group B was also isolated from the mediastinal tissue. Salmonella was identified by sugar fermentation, gas production and H₂S positive. Based on slide agglutination with polyvalent antiserum specific for Salmonella O group antigen (Murex; Dartford, England) it was serogrouped as B.

The patient became apyrexial and was maintained on ciprofloxacin for 2 more weeks. Over the next month she gradually improved, became asymptomatic, and she was discharged from hospital. A follow-up echocardiogram 5 months later revealed dilated left ventricle with concentric hypertrophy, but no pericardial effusion.

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Discussion

Salmonella bacteraemia tends to occur in SLE and the prognosis is usually poor [3]. The susceptibility of SLE patients to salmonella sepsis is due mainly to the disease activity and immunosuppressive drug therapy, but salmonella bacteraemia has occurred in SLE patients who have not received immunosuppressive therapy, leading to the suggestion that SLE by itself is associated with a defect in host defence against salmonella bacteria [5].

Lupus activity usually continues to regress over time as patients approach end-stage renal disease [6]. Immune dysregulation occurs in uraemia and bacterial infection is a major cause of mortality and morbidity in the dialysis population [1,7]. Haemodialysis vascular access is the main route for bacteraemia in haemodialysis patients. *Staphylococcus aureus* is the most frequent organism causing blood-stream infection in these patients [7]. In chronic dialysis patients other factors such as malnutrition, uraemic toxins, and iron overload contribute to the impairment of defence against infection [7,8]. Our patient was known to have mild asymptomatic pericardial effusion for several weeks prior to her presentation. Gastroenteritis or diarrhoea did not precede the illness.

Transient salmonella bacteraemia from the gastrointestinal tract before or even without gastrointestinal symptoms is a previously noted phenomenon [9]. Our patient could have seeded salmonella into the pericardial fluid from the blood. Subsequent blood cultures remained sterile but tissues continued to grow salmonella for several days on appropriate antibiotics. Most of the organisms causing purulent pericarditis in SLE patients are Gram-positive organism, *Staphylococcus aureus* mainly. Salmonella was reported to cause pericarditis once in an adult not on haemodialysis [4] and in a child [10]. Other rare causes of purulent pericarditis in SLE patients include *Neisseria gonorrhoeae* [11] and *Candida albicans* [12].

Thus salmonella pericarditis in dialysis patients has not been reported before, and could result in serious complications associated with prolonged morbidity. Drainage by pericardiocentesis or surgery remains an important part of treatment along with proper anti-salmonella therapy.

References


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