Case Report

Visceral leishmaniasis and renal tuberculosis in a patient on maintenance haemodialysis

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Introduction

Infection in patients receiving maintenance haemodialysis therapy remains a common cause of morbidity and mortality. Alterations in immune response induced by the uraemic state increase the susceptibility to infection and appear to play an important role in determining the type, incidence and outcome of these infectious complications. In this setting, opportunistic infection depends upon the epidemiological exposure of the patients and the state of immunosuppression.

We report on a case of visceral leishmaniasis associated with renal tuberculosis in a patient living in an endemic country who had been treated with haemodialysis for 19 months. Both the opportunistic infections and their clinical courses are remarkable, and a review of previously reported cases is provided. Antimonial derivatives and antituberculous therapy induced a rapid and complete remission.

Case report

A 44-year-old man with end-stage renal failure due to chronic pyelonephritis of tuberculous origin had been treated with haemodialysis for 19 months in another unit. His residual diuresis was about 1000 ml/day. He complained of a frequent need to urinate, dysuria and pain referable to epididymides. Piuria steril was present in several urine samples. The patient presented with a 3-day history of intermittent fever and malaise. He was a hunter and had several dogs at home.

At admission to the hospital, the patient was febrile (38°C), pale and hepatosplenomegaly was palpable. Laboratory tests revealed anaemia (haemoglobin 6.2 g/dl), leukopenia (WBC 1500/mm³) and thrombocytopenia (platelets 102 000/mm³). Immunoglobulin levels were IgG 2140 mg/dl, IgA 254 mg/dl, and IgM 190 mg/dl. The transaminases were normal. The PPD status was negative and serology for HIV was negative. Thoracic radiographs were normal. An abdominal ultrasound study showed a calcified granuloma in the liver and small, irregular kidneys with multiple calcifications and irregular parenchymal cavities. These findings were confirmed with computerized tomographs. A Ziehl–Neelsen of the urine was positive.

A bone-marrow examination was negative for Ziehl–Neelsen staining and direct testing of parasites. The patient was treated with rifampicin 600 mg/day, ethambutol 400 mg/day, and isoniazid 150 mg/day. After 8 days of the antituberculous treatment, the fever and the pancytopenia persisted.

A new bone-marrow examination carefully evaluated by an experienced observer showed invasion of macrophages by amastigote Leishmania donovani. The parasite was also found in the first bone-marrow aspiration. Anti-leishmanial antibodies were positive to titres of 1/120. The patient was given intramuscular pentavalent antimonials (Glucantime) 425 mg/day (6 mg/kg per day) for 5 days. Afterwards, because of the onset of nausea and vomiting, the dose was reduced to half (3 mg/kg per day) and continued for 25 consecutive days with excellent tolerance. On the days the patient underwent haemodialysis, the dose was given 6 h after the end of the dialysis session. The QT interval and amylase serum levels were not modified. The fever subsided after a few days of treatment with Glucantime and afterwards, the pancytopenia recovered, and the hepatosplenomegalgy disappeared. Twenty seven days after admission, the patient was discharged in an excellent clinical condition. Ethambutol was discontinued 2 months later and rifampicin and isoniazid maintained for 9 months. The patient has been followed up for 2 years without recurrence.

Discussion

Mycobacterium tuberculosis infection is more than 10–15 times more frequent in haemodialysis patients...
than in the normal population. Its clinical manifestation in these patients is more varied than in the general population and includes miliary disease, pulmonary cavitation, intestinal involvement, and bone disease [1]. Reactivation generally results from old, caseous foci located principally in the lungs, lymph nodes, bone and genitourinary tract.

Visceral leishmaniasis is an acute, or subacute, disease characterized by fever, chronic consumption, hepatosplenomegaly, and hypergammaglobulinemia, which is almost invariably fatal if not treated. The disease is endemic in many underdeveloped countries around the world and is also present in some Mediterranean countries such Spain, southern France, Italy, and Greece. Leishmaniasis is recognized as a serious opportunistic infection in individuals infected with HIV, those receiving immunosuppressive therapy after organ transplantation, or during the administration of high doses of corticosteroids [2–12].

This is the first case of visceral leishmaniasis complicating mycobacterial tuberculosis infection in a haemodialysis patient and, to the best of our knowledge, only one case of visceral leishmaniasis in a haemodialysis patient has been reported [13]. It has been suggested that impaired cell-mediated immunity and inhibition of macrophage function, which are important in the elimination of intracellular micro-organisms, may be predisposing factors for this kind of infection. The most common underlying diseases in immunocompromised hosts with visceral leishmaniasis are haematological malignancies, renal transplantation, systemic lupus erythematosus, and HIV infection [3–13]. There is a tendency for a relapse of infection to occur in these patients and treatment has to be continued for several weeks or months to sterilize the infected tissues or to prevent relapse [6,11]. These data suggest that a deficiency in the host’s immune system rather than micro-organism virulence factors are responsible for the infection and that an impairment of the mononuclear phagocyte function produced by uraemia and by concomitant tuberculosis may have predisposed our patient to leishmanial infection.

Although antimony derivatives are the drugs of choice for leishmaniala [14], these must be handled with caution in immunocompromised patients. When current antiparasitic therapy fails to eradicate leishmaniala from infected tissues, amphotericin B has been proposed as an alternative treatment [11]. In renal transplant patients, good results have been obtained with doses of meglumine antimoniate ranging from 10 to 100 mg/kg/day [6,8,10,11]. However, little is known about the pharmacokinetics of antimonial compounds in renal insufficiency [15], and there are no data concerning dosage adjustment in haemodialysis. It has been suggested that continuous treatment with small doses of antimony may give rise to accumulation of antimony in the body because of its long biological half-life. Our patient tolerated a full course of meglumine antimoniate despite poor renal function, probably because a low dose was administered. This low dose proved to be sufficient to eradicate the organism. Concomitant treatment with rifampicin is another possible therapeutic regime [16–18].

This case illustrates that infectious disease remains a principal cause of morbidity in haemodialysis patients. The possibility that more than one organism can simultaneously infect immunodepressed patients should always be taken into account. We believe that aggressive diagnostic approaches are indicated if the source of infection is not discovered promptly. It appears evident that in patients with renal insufficiency, the dosage of antimonials should be adjusted.

References


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