Dear Sir,

Tuberculosis is a relatively common infectious disease among hemodialysis patients and is usually related to Mycobacterium tuberculosis [1, 2]. Infections due to other mycobacteria are exceptional in these patients [3]. We observed a disseminated mycobacterial infection due to M. kansasii in a patient dialyzed for 4 years.

A 63-year-old Caucasian man suffered from psychosis and type II diabetes with hypertension and end-stage renal failure. During the past 4 years, he had been dialyzed three times weekly for 4 h without major complications. Then he developed fever up to 39°C with poor appetite and loss of weight and was hospitalized. On admission, he had high grade fever with cardiac insufficiency and edemas. The liver was enlarged. There was no peripheral lymphadenopathy. Widespread cutaneous infection linked to severe pruritus was prominent. A tuberculin test was negative. Chest X-ray showed a reticular infiltrate of the right subclavian region with a frank cardiomegaly.

Laboratory data showed a white cell count of 9 × 10^9/L with 76% polymorphonuclear leukocytes. The erythrocyte sedimentation rate was 105 mm/h. Total serum protein was 6.4 g/dl, serum albumin was 3.2 g/dl, and serum γ-globulin was 1.6 g/dl. Serum aminotransferase and bilirubin were normal. Serum γ-glutamyltransferase and alkaline phosphatase were twice the upper limit of normal values. Tests for HBsAg, hepatitis C, and HIV were negative. Sputum and gastric juice were negative on smear for acid-fast bacilli (AFB), and cultures were set up. Blood cultures yielded no growths. Echocardiography showed a non-obstructive cardiomyopathy without valvular disease. An abdominal CT-scan was normal.

The patient received isoniazid 350 mg, rifampicin 600 mg and ethambutol 600 mg. Because of the cutaneous infection, vancomycin was added to this regimen for 15 days. Two months later, two cultures of gastric juice gave growths of AFB identified as M. kansasii; this microorganism was sensitive to the antibiotics prescribed. Treatment was continued with isoniazid 250 mg after each dialysis session, rifampicin 600 mg daily and ethambutol 600 mg after dialysis and 400 mg daily between the dialysis sessions. However, the patient remained severely ill and febrile, and cardiac failure persisted. Chest X-ray did not improve. He
developed cardiac tamponade with a left pleural effusion, both requiring a drainage. Pleural and pericardial fluid showed 15% polymorphs with 40% lymphocytes and 45% mesothelial cells, proteins were 1.2 g/dl, bacterial culture showed no growth, and AFB smear was negative. Blood cultures remained negative.

One month later, the patient died. Cultures, including mycobacteria, of pleural, pericardial and gastric fluid yielded no growths. Autopsy disclosed multiple lumbar, preaortic and mediastinal lymphadenitis. On pathologic examination, liver and lymph nodes contained numerous giant cells, and noncaseating granulomas were present in the spleen. No specific pulmonary, pleural, pericardial and peritoneal lesions were seen. There was no evidence of bacterial endocarditis.

The incidence of tuberculosis in hemodialyzed patients is 10-12 times higher than that in the general population [1, 2]. Most of these patients have miliary or extrapulmonary tuberculosis [4]. Presenting symptoms are nonspecific, including anorexia, weight loss and fever. Isolation of mycobacteria is often difficult, and the diagnosis may rely upon a biopsy specimen of an involved organ [1]. Mortality varies from 0 [4] to 75% in some series [5].

Nontuberculous mycobacteria are rarely isolated in fluid cultures. In patients with chronic renal failure, Rutsky and Rostand [3] observed 2 cases with M. avium intracellulare and 1 case with M. fortuitum. Osteomyelitis related to M. kansasii has been once reported in a 38-year-old woman 21 months after receiving a renal graft [6]. Pulmonary involvement with cavitations or infiltrates is rarely present in nondialyzed and nonimmunocompromised patients infected with M. kansasii [7, 8]; two thirds of these cases have preexisting lung disease [7]. Recently, disseminated infections due to M. kansasii have been mostly described in HIV patients [9, 10]. Even in HIV patients, antituberculous therapy is usually effective [9].

To our knowledge, no dialysis patient infected with M. kansasii has been reported. This opportunistic infection may have been favored in our patient by reduced host defenses and an impaired cellular immunity, due both to diabetes and chronic renal failure. No other underlying disease was found. Hematophagic [11] or silicone histiocytosis [12] could be excluded by pathological examination. Pleuropericarditis was not directly related to mycobacteriosis and was mainly attributed to chronic volume overload. Dissemination of the mycobacteriosis and severe undernutrition could have favored the fatal outcome, despite 3 months of adequate antituberculous therapy and the absence of AFB on repeated smears. This case reminds that nontuberculous, as well as tuberculous, mycobacterial infection should be systematically suspected in any dialysis patient with persistent fever and anorexia of unknown origin. Prompt treatment by antituberculous drugs should then be instituted, but recovery is not an invariable rule.

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