Fatal *Brucella melitensis* Endocarditis in a Hemodialysis Patient

C.	Cem	Sungur ^a
A.	Arzu	Sungur ^b
G.	Gokhan	Gedikoglu ^b
A.	Alp	Usubutun ^b
U.	Unal	Yasavul ^a
C.	Cetin	Turgan ^a
S.	Sali	Caglar ^a

Department of ^aNephrology and ^bPathology, Hacettepe University School of Medicine, Ankara, Turkey

Cem Sungur, MD, PK (PO Box) 272, TR-06693 Kavaklıdere-Ankara (Turkey)

Dear Sir,

Several mechanisms are implicated in the immunosuppression of regular hemodialysis patients and they are prone to well-defined infectious complications [1, 2]. On the other hand, regional endemic infectious agents should be considered in the differential diagnosis of febrile hemodialysis patients. Here we describe a fatal *Brucella melitensis* infection in a regular dialysis patient. A postmortem examination revealed an undiagnosed infective endocarditis as the cause of death.

The patient was a 27-year-old man who had received a diagnosis of glomerulonephritis when he was 6 years old. The disease was characterized by recurrent episodes of hematuria and edema, but renal biopsy was not performed. His disease progressed to chronic renal failure in 1980 and was admitted to a regular dialysis program in 1986, in another medical center. In 1988 he underwent a successful renal transplant from his mother and his immunosuppressive regimen included azathioprine, cyclosporine A and prednisone. He was in good health till January 1992 when he started to experience fever and malaise and this infection was followed by a decline in urinary output and edema. He complained of night sweats and lost 11 kg during the following 4 months. Because of progressive renal failure, hemodialysis was prescribed 3 times weekly in March 1992 and his immunosuppressive drugs were discontinued. His febrile episodes continued and he became progressively bedridden and began to experience arthralgias involving his metacarpophalangeal, proximal interphalangeal joints. During the last 2 weeks he developed progressive dyspnea, orthopnea and hemoptysis. He complained of chest pain during the ensuing week. Because of deterioration in his clinical condition he was transferred to our hospital on April 28, 1992.

A physical examination revealed a lethargic man who weighed 42 kg. His blood pressure was 150/50 mm Hg, a regular pulse of 96/min and body temperature 39.0°C. A brief systolic murmur was heard at the apex and aortic valve area. Spleen was palpated 4 cm below the left costal margin and a slightly tender liver could be felt 3 cm below the right costal margin. Ascites

was detected on percussion. A mass compatible with a renal graft was palpated in the left lower quadrant. There was a motor deficit involving the right arm and the patient could not flex his right hand from the wrist.

A laboratory work-up disclosed the following findings: Hb 4.5 g/dl, Hct 14.3%, WBC 3,700/mm³, differential count 60% PNLs, 6% monocytes, 34% lymphocytes, CRP 80, Na 128 mmol/l, K 4.7 mmol/l, serum creatinine 568 mmol/l, plasma calcium 2.9 mmol/l, serum phosphorus 1.2 mmol/l, ALT 194 U/l, AST 43 U/l, alkaline phosphatase 65 U/l, serum globulins 22 g/l, serum albumin 32 g/l. HBS antigen was negative, anti-HBS antibody was positive, anti-HCV antibody (Abbott) was positive and HIV antibody (ELISA) was negative. A standard tube agglutination test for brucella was negative. Blood cultures drawn at the day of admittance grew *B. melitensis* on the fifth day of hospitalization.

He was referred to the Department of Infectious Diseases and was prescribed doxycycline (100 mg/day) and rifampicin (600

Fig. 1. Macroscopic appearance of the destroyed aortic valve cusps with multiple vegetations.

Fig. 2. Hypocellular and shrunken glomeruli, tubular atrophy and prominent hyperplasia and thickening in vascular walls. HE × 115.

mg/day) for brucellosis apart from hemodialysis, blood transfusions. On the third day of his antibiotic treatment he developed sudden cardiac death and did not respond to cardio-pulmonary resuscitation.

A postmortem examination disclosed endocarditis involving the aortic valve with multiple vegetations (fig. 1). On gross examination the graft was small in size, firm and fibrous. On microscopic examination there

was marked cortical fibrosis, hypocellular and shrunken glomeruli and prominent intimal hyperplasia and thickening of vessel walls were observed (fig. 2). These findings were compatible with chronic rejection of the graft.

This case proves that diagnostic evaluation of febrile hemodialysis patients should include endemic microbiologic agents and the patients should be treated accordingly.

References

Haag-Weber M, Hörl WH: Uremia and infection: Mechanisms of impaired cellular host defence. *Nephron* 1993;63:125-131. Nsouli KA, Lazarus JM, Schoenbaum SC, Gottlieb MN, Lowrie EG, Shocair M: Bacteremic infection in hemodialysis. *Arch Intern Med* 1979;139:1255-1259.