Acute Renal Failure Due to Leptospirosis in a Renal Transplant Patient

R.C.	Roberto C.	Manfro
M.V.	Marta V.	Boger
J.	Jaime	Kopstein
L.F.	Luis F.	Gonçalves
C.A.	Carlos A.	Prompt

Renal Transplant Unit, Hospital de Clínicas de Porto Alegre, Federal University of Rio Grande do Sul, Porto Alegre, Brazil

Roberto C. Manfro, MD, Hospital de Clínicas de Porto Alegre, Unidade de Transplante Renal, Porto Alegre, RS. 90210 (Brazil)

Dear Sir,

Acute renal dysfunction in the late course of renal transplantation is an unusual finding. The present report describes a case of acute renal failure (ARF), secondary to an episode of leptospirosis, in a renal transplant patient 20 years after transplantation.

A 20-year-old man received a haplotype-matched kidney allograft in 1971. In September 1990, his serum creatinine was $97.3 \mu\text{mol/l}$ (1.1 mg/dl) and he was receiving 150 mg of azathioprine q.d. since he had discontinued prednisone on his own 2 years before. Four months later, he presented to the emergency room with a 5-day history of muscle pain, fever, vomiting and diarrhea. He reported he had had contact with rat tissues 10 days prior to admission. The physical examination displayed an acutely ill patient with a blood pressure of $120/70 \text{ mm Hg}$, heart rate of 140 and a temperature of 38°C . He also presented jaundice, bilateral conjunctival suffusion, a tender liver, splenomegaly and petechiae in the medial aspect of the right thigh. The allograft was not tender. Numerous warts were present in the patient's hands. At that moment, his serum creatinine and BUN were $530.4 \mu\text{mol/l}$ ($6.0 \text{ mg\%}$) and 26 mmol/dl ($73 \text{ mg\%}$), respectively, SGOT 38 and SGPT 42 U/l. Total and direct bilirubin increased up to 9.8 and 7.0, respectively, 2 days later. Leukocytes were $15,600/\text{mm}^3$, and platelet count was $15,000/\text{mm}^3$. Urinalysis displayed 3 + proteinuria, and the urinary sediment had 5 erythrocytes and 20 leukocytes per high-power field with granular and hyaline casts. He was treated with intravenous fluids, penicillin, vitamin K, cimetidine and loperamide. The azathioprine dosage was halved in the next 2 days and discontinued by the 3 day when peritoneal dialysis was started due to worsening of uremia and continued for 48 h. Within 24 h. of admission, the urinary volume was 450 ml. Afterwards, he presented improvement of diuresis and renal function. Prednisone 10 mg/day was started by the 10th day and azathioprine 50 mg/day 2 weeks later. Antileptospira IgM antibodies tested positive (macroagglutination). The patient was discharged 14 days after admission with normal renal function.

This report describes a case of ARF secondary to leptospirosis in a renal transplant patient 20 years after transplantation. We think that leptospira transmission may have occurred through

the wart lesions in the patient's hands, although transmission through unabraded skin has been reported as possible [1]. Leptospirae are carried to the glomeruli and peritubular capillaries from which sites they migrate to the renal interstitium. ARF occurs through a variety of mechanisms including nonspecific effects of infection such as hypovolemia, intravascular coagulopathy, hemolysis, severe jaundice and increased viscosity, direct nephrotoxicity due to a toxic byproduct of leptospirae and a possible immunologic mechanism since leptospiral antigens have been demonstrated in the renal interstitium. The lesion is primarily tubulointerstitial with mononuclear cell infiltration and tubular degeneration. Glomerular changes are limited to mild mesangial hypercellularity [2]. A renal biopsy would have been most helpful, marked thrombocytopenia prevented us to perform it. In this particular case, we do not feel that the course of disease was modified by immunosuppressive therapy. Renal transplantation is increasing in Brazil [3] and some infections, although not opportunistic, are much more prevalent in the third world so that the transplant physician in these areas must be aware that intriguing infections can occur in this setting and be responsible for temporary renal dysfunction.

References

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