Post-transplantation Infections in Bolivia

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ABSTRACT

Over 26 years, we found 46 infectious episodes in 350 kidney transplant recipients. Fifteen were urinary tract infections, recurrent in 4 patients. There were 8 cytomegalovirus infections, three of them fatal when intravenous (IV) ganciclovir was not available. Seven patients had a reactivation of tuberculosis (TB) in the pleura, cervical spine, lumbar spine, knee, ankle, skin and peritoneum, respectively, and were all resolved satisfactorily with conventional anti-TB therapy. Three patients transplanted before routine prophylaxis with the use of acyclovir developed an extensive herpes zoster infection in the 1st 6 months after transplantation, which was resolved with the use of oral acyclovir, and 1 had a disseminated herpes simplex infection resolved with the use of IV acyclovir. Three patients transplanted before routine prophylaxis with trimethoprim sulfa developed *Pneumocystis carinii* pneumonia in the 1st 6 months after transplantation, which was fatal in one of them. In 2 patients, we found a *Nocardia* infection, confined to the lung, which was cured in one of the cases and systemic and fatal in the other. Two patients transplanted before routine prophylaxis with the use of nystatin developed esophageal candidiasis in the 1st 6 months after transplantation. One patient developed infective endocarditis in a stenotic bicuspid aortic valve and died 10 years later after another incident of infective endocarditis at the prosthetic aortic valve. Two patients developed an extensive condyloma at the penis, perianal region, and perineum owing to human papillomavirus, requiring extensive surgical resection and podophyllin applications. Another patient developed fatal post-transplantation lymphoproliferative disease due to Epstein-Barr virus infection 15 years after transplantation. One patient developed a severe and fatal mucocutaneous leishmaniasis with no response to conventional antimonial therapy. It is interesting to note that despite Chagas disease being endemic in Bolivia, we had no patients with reactivation or transmission through the graft even though many of the patients and donors were serologically positive for Chagas disease.

INFECTIONS are an important cause of morbidity and mortality in renal transplant recipients [1–3]. Successful management is complicated by factors related to immune function in the host and the epidemiology of infection [1–3]. Transplant recipients are susceptible to a broad spectrum of infectious pathogens, manifest diminished signs and symptoms of invasive infection, and may develop systemic signs, such as fever, in response to noninfectious processes, such as graft rejection or drug toxicity, with multiple processes often present [1–3]. Immunocompromised patients tolerate invasive established infection poorly with high morbidity and mortality, lending urgency to the need for an early specific diagnosis to guide antimicrobial therapy [4]. Given the T-lymphocyte dysfunction inherent to transplantation immunosuppression, viral infections in particular are increased [1–3].

Early infections that are typical of the 1st month stem from catheters, surgical wound, the lungs, the urinary tract, or sepsis without a determined origin. Infections of the surgical wound show few signs of local inflammation. Early infections are caused by gram-positive bacteria

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(*Staphylococcus* and *Enterococcus*) and gram-negative bacteria (*Enterobacter*, *Pseudomonas*, *Acinetobacter*), followed by fungi (*Candida*, *Mucor*, *Aspergillus*) [3].

Infections that occur starting in the 1st month and in the 1st 6 months are generally produced by opportunistic microorganisms during the period of maximum immunosuppression. After the 1st 6 months, when immunosuppression has been reduced to the minimum, the infections are those usually observed in the general population.

The purpose of the present paper is to describe and comment on the infections found in our patients that have undergone renal transplantation.

METHODS

As of 1987, when we performed our first renal transplantation, we designed a pilot study protocol to document the infections observed in our patients. The accumulated data over 26 years in 350 performed transplantations were assessed retrospectively and are the basis for this publication.

RESULTS

Over 26 years, we found 46 infectious episodes in 350 kidney transplant recipients. Table 1 presents the demographic features in our renal transplant population. Table 2 presents the most relevant clinical features in our 350 renal transplant recipients. And Table 3 presents the incidence of post-transplantation infections by site.

Fifteen were urinary tract infections. Two occurred in the early postoperative period and were related to the urinary catheter. Four young female patients with chronic constipation and frequent genital infections had recurrent urinary tract infections that were eventually resolved.

Table 1. Demographic Features in 350 Renal Transplantations Performed

Characteristic	*n*	%
No of transplants	350	100
Age: 8–68 y, mean 43 y		
<30 y	80	23
30–60 y	241	69
>60 y	28	8
Sex		
Male	180	51
Female	170	49
Donor source		
Living family related	289	83
Living affectively related	56	16
Deceased brain death	5	1
Primary disease		
Glomerular	126	36
Hypertensive	70	20
Unknown	62	18
Tubulointerstitial	42	12
Lupus nephritis	28	8
Diabetic	14	4
Polycystic	8	2

Table 2. Clinical Features–350 Renal Transplantations

Pre-transplantation CMV status		
IgG+/IgM−	344	
IgG−/IgM−	6*	
Pre-transplantation EBV status		
IgG+/IgM−	340	
IgG−/IgM−	10†	
Early postoperative complications		
Delayed graft function	14	4
Urinary leak	14	4
Lymphocele	12	3
Acute rejection	6	2‡
Urinary tract infection	2	1
Others	4	1
Long-term complications		
Acute rejection	10	3§
Chronic rejection	20	6‖
Infections	44	13

Abbreviations: CMV, cytomegalovirus; EBV, Epstein-Barr virus.
*One patient received intravenous ganciclovir for 2 weeks and the other 5 oral valgancyclovir for 1 year.
†All received oral acyclovir 400 mg twice a day for 1 year.
‡1 patient died after OKT3 therapy, 2 patients lost the graft, and 3 responded well to plasma exchange and intravenous immunoglobulin.
§All were related to nonadherence to inmunosupressive medications (prednisone, azathioprine and cyclosporine) and partially responded to intravenous methylprednisolone.
‖All delayed progression after switch to mycophenolate and tacrolimus.

There were 8 cytomegalovirus (CMV) infections, all presenting with a prolonged mononucleosis-like syndrome, 1 associated with allograft dysfunction, 1 with a severe inflammatory bowel disease, and 1 with severe CNS compromise. Three were fatal when intravenous (IV)

Table 3. Incidence of Post-transplantation Infections by Site

Site	*n*
Systemic	
Nocardiosis	1
Leishmaniosis	1
CMV	4
Urinary tract	15
Pulmonary	
Nocardiosis	1
Pneumocystis	3
Pleural TB	1
Mucocutaneous	
Skin TB	1
Mucocutaneous leishmaniosis	1
Herpes zoster	3
Disseminated herpes simplex	1
Digestive	
Esophageal candidiasis	2
CMV-colitis	1
CNS-CMV encephalitis	1
Lymph nodes EBV-PTLD	1
Osteoarticular TB	4
Peritoneal TB	1
Genital warts–HPV	2

Abbreviations: TB, tuberculosis; CNS, central nervous system; PTLD, post-transplantation lymphoproliferative disease; HPV, human papillomavirus; other abbreviations as in Table 2.

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