Lymphadenopathic Aggressive Kaposi's Sarcoma in a Renal Transplant Recipient with HLA-DR2 and HLA-DR5 Antigens

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Dear Sir,

One of the complications of long-term immunosuppression in renal transplant recipients is the increased incidence of certain neoplasms [1]. The incidence of Kaposi's sarcoma (KS) is 5.6% in organ allograft recipients, whereas its prevalence in the general population before the AIDS epidemic was calculated to be 0.02-0.07% of all cancers [2, 3]. It is usually encountered in populations of Mediterranean, Jewish, or Arabic ancestry. 63% have nonvisceral KS confined to skin, conjunctiva, or oropharyngeal mucosa. 37% have visceral disease involving mainly gastrointestinal tract and lungs [4]. Only 10% of the cases present without cutaneous involvement. Lymphadenopathic aggressive KS, also affecting the gastrointestinal tract, is more commonly observed in patients with AIDS or in African patients. Furthermore, AIDS-related KS is associated with human leukocyte antigens (HLA) DR2 in European patients and DR5 in Jewish and Italian patients [5]. A previous study [6] investigating the association between certain HLAs and KS showed no significant relations.

One of our transplant recipients, a 42-year-old man, developed lymphadenopathic aggressive KS with gastrointestinal involvement 11 months after a successful renal transplantation. He was admitted to our dialysis unit in October 1991 because of end-stage renal disease of unknown etiology. In March 1992, he underwent renal transplantation from his sister. A previous serologic HLA typing revealed that there was a complete

Fig. 1. Neoplastic proliferation of spindle cells and formation of slits with erythrocyte extravasation in the lymph node. HE. × 230.

match between donor and recipient. The recipient's HLAs were typed as follows: A2, A26(10), B62(15, W6), B27(W4), DR15(2, QDQ), DR11(5,52, DQ3) and DQ(QW1). The postoperative course was uneventful, and there was no rejection episodes. He received a triple immunosuppressive regimen including aza-thioprine, prednisolone, and ciclosporin A. After the 8th week of transplantation, his immunosuppressive treatment was maintained by 10 mg/day prednisolone, 200 mg/day ciclosporin A, and 100 mg/day azathioprine. When he developed

painless left submandibular and preauricular lymphadenopathies in February 1993, the creatinine level was 125 $\mu\text{mol/l}$ and blood urea nitrogen

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6.3 mmol/l, and there was no evidence of proteinuria and active urinary sediment. A lymph node biopsy revealed KS (fig. 1). A physical examination to find previously overlooked skin lesions was negative. Human immunodeficiency virus screening tests were negative. Upper gastrointestinal endoscopy with biopsy revealed visceral involvement of the fundus of the stomach, and rectosigmoidoscopy disclosed internal hemorrhoids. Ultrasonographic examination revealed three para-aortic lymph nodes not exceeding 3 cm in diameter. Computerized tomography of the chest yielded normal findings and abdominal computerized tomography confirmed the presence of abdominal lymphadenopathies.

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denopathies. He was referred to the oncology department, and his immunosuppressive drugs were discontinued with the exception of prednisolone 10 mg/day. Because of the aggressive nature of the tumor and visceral involvement, he also received combined chemotherapy with bleomycin and vincristine. This therapy resulted in a prompt clinical response with regression of submandibular, preauricular, and abdominal lymph nodes. He is well 8 months after the diagnosis of KS and cessation of the immunosuppressive treatment, with no rejection episodes and good renal function. His lymphadenopathies have regressed completely, and a repeat endoscopy has revealed that the fundic lesion has also regressed.

This 1st case with KS in our series of 128 renal transplants presented with an aggressive form of the disease without cutaneous lesions. This form is rare in solid-organ recipients and is usually encountered in patients with AIDS or in African patients. Although previous studies have failed to demonstrate an association of KS with certain HLA types, there is a clear relation between HLA-DR2 and HLA-DR5 in patients with AIDS. We suggest that HLA-DR2 and HLA-DR5 may also be important determinants of the aggressive form of KS in solid-organ recipients. Further studies with larger patient groups may reveal a particular group of transplant recipients prone to this unusual form of KS.

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