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HHV8 in renal transplant recipients

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Abstract Human herpevirus 8 (HHV8) DNA sequences have been found in lesions from patients with Kaposi's sarcoma (KS) in several forms including immunosuppressed transplant patients. We wanted to study the transmission of HHV8 in kidney transplant recipients and to assess the risk of development of KS related to the viral infection in this group of patients. We tested sera of 120 renal transplant recipients with serological assay for antibodies to HHV8 antigens before transplantation and then we tested sera of 66 patients of the same group after transplantation. Antibodies were detectable in 27.5 % of the patients before transplantation. In the seropositive population 15.1 % developed KS and in the negative group 1.1 %. Analysing 66 posttransplant sera we noticed that 24 % of the seronegative patients became positive after transplantation. Our data suggest that being positive for HHV8 before transplantation could be an important risk factor for the development of KS.

Key words Human herpesvirus 8 · Kaposi's sarcoma · Kidney transplant

Introduction

In 1994 Chang and colleagues [2] identified DNA sequences homologous to the gene of the gammaherpesvirinae (Epstein Barr virus and herpesvirus Saimiri) in Kaposi's sarcoma (KS) tissues from patients with AIDS. This new herpesvirus was called human herpesvirus 8 (HHV8) and DNA sequences have been found in lesions from patients with KS in several forms including immunosuppressed transplant recipients [4]. These findings suggested that HHV8 might have a causative role in the pathogenesis of KS. In fact, HHV8 DNA was detected in all cases of iatrogenic KS lesions [1] and HHV8 seroprevalence correlates with the incidence and distribution of iatrogenic KS (Mediterranean, Arabic, Jewish and African individuals) [3]. We wanted to study the transmission of HHV8 in kidney transplant recipients and to assess the risk of development of KS related to the viral infection in this group of patients.

Materials and methods

We analysed HHV8 infection in renal transplant recipients by using serological studies. Sera of 120 patients were tested with serological assay for antibodies to HHV8 antigens before and after transplantation. For immunofluorescence assay we used the BC3 cell line (American Type Culture Collection, Rockville, Md., USA) that is latently infected with HHV8 and does not harbour EBV genomes. Specific reactivity at a diluition of 1/40 or more with uninduced and TPA-induced BC3 cells was considered positive for HHV8 antigens.

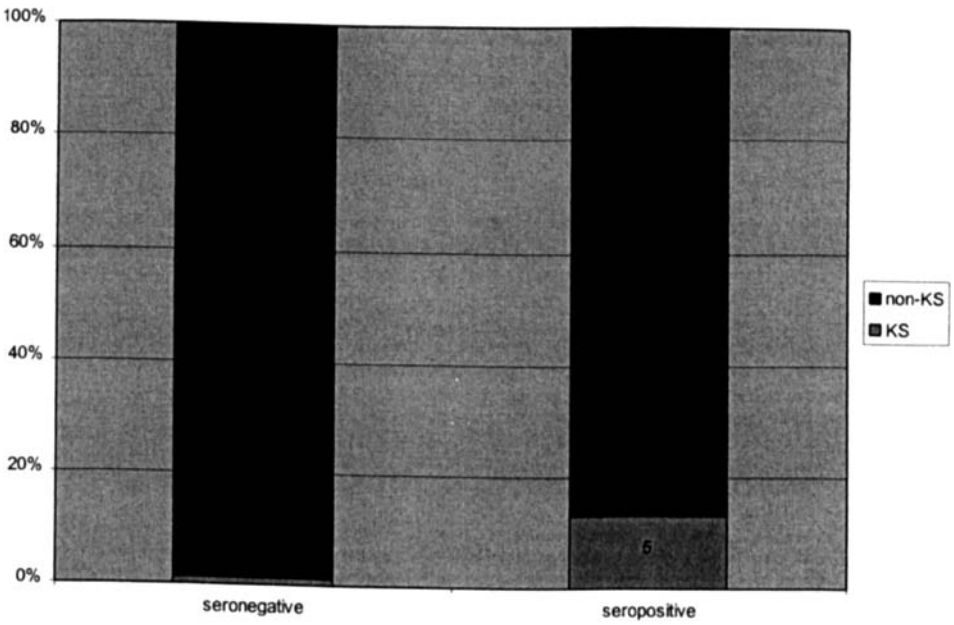

Fig. 1 Percentage of Kaposi's sarcoma (KS) in seronegative and seropositive renal transplant recipients (odd ratio = 15.3)

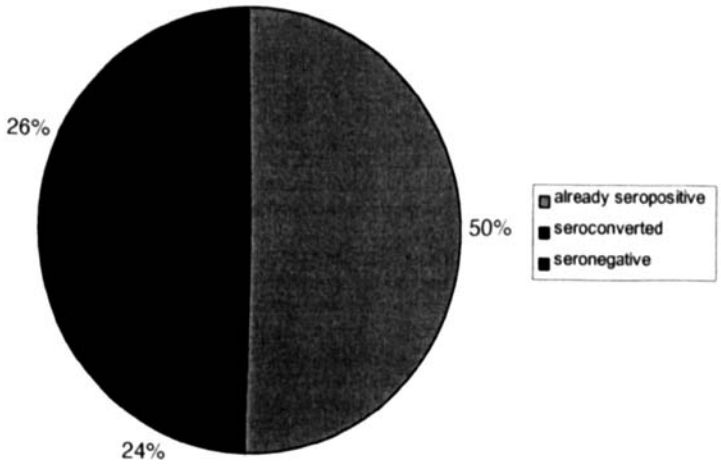

Fig. 2 Seroprevalence of human herpesvirus 8 in 66 renal transplant recipients

Results

Antibodies to HHV8 were detectable in the sera of 33/120 transplant patients (27.5%) before transplantation. In this population we observed six cases of KS (5% of transplant recipients). Five of them were positive before transplantation and 1 was negative. Thus in the seropositive population 5/33 (15.1%) developed KS, and in the negative group only 1/87 (1.1%) developed the disease (odd ratio = 15.3; Fig. 1). Retrospectively we tested posttransplant sera of 66 patients. Thirty-three were negative before transplantation and only 8 of them (24%) were positive at different times after transplantation (Fig. 2).

Discussion

Our data suggest that being seropositive for HHV8 before transplantation could be a risk factor for the development of KS (15.1% of KS in the seropositive population in comparison to 5% of the whole population) and that transplantation does not seem to be a frequent route of transmission of the virus (only 24% of those seronegative before transplantation became positive). Furthermore the only patient with KS who was seronegative during dialysis became seropositive after transplantation. These findings confirm the strong correlation between the onset of KS and the viral infection as already described in other studies in renal transplant recipients [5]. Even though we cannot quantify the risk of development of KS in the seroconverted patients from this preliminary analysis, viral reactivation after the on-

set of immunosuppressive therapy seems to play an important role in the pathogenesis of KS in renal transplant recipients.

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References

1. Cathomas G, Tamm M, McGandy CE et al. (1997) Transplantation-associated malignancies: restriction of human herpes virus 8 to Kaposi's sarcoma. Transplantation 15: 175–178
2. Chang Y, Cesarman E, Pessin MS et al. (1994) Identification of a novel herpervirus-like DNA sequence in AIDS-associated Kaposi's sarcoma. Science 266: 1865–1869
3. Gao SJ, KingsleyL, Li M et al. (1996) KSHV antibodies among Americans, Italians, and Ugandans with and without Kaposi's sarcoma. Nat Med 2: 925–928
4. Huang YQ, Li JJ, Kaplan MH et al. (1995) Human herpesvirus-like nucleid acid in various forms of Kaposi's sarcoma. Lancet 345: 759–761
5. Qunibi W, Al-Furayh O, Almeshari K et al. (1998) Serologic association of human herpesvirus eight with posttransplant Kaposi's sarcoma in Saudi Arabia. Transplantation 65: 583–585