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MEETING ABSTRACT Open Access

A role for activatory KIR/HLA complexes in HIV-associated Kaposi's sarcoma development

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Kaposi's Sarcoma is a vascular tumour caused by the Kaposi's sarcoma-associated herpesvirus (KSHV). Although KSHV and HIV co-infection is often observed in HIV disease, the majority of patients nevertheless do not develop KS. Innate immunity, and in particular human leukocyte antigen alleles (HLA) and killer cell immunoglobulin-like receptor (KIR) are suspected to play a role in the development of viral associated carcinomas. We verified possible correlations between KIR/HLA complexes and KS by analyzing 82 HIV subjects: 15 with KS (KSpos), 32 KSHV-infected without KS (KSHVpos/KSneg) and 35 KSHV-uninfected (KSHVneg/KSneg); results were compared to those of 103 age-and sex-matched KHSV-seronegative individuals (HC).

Molecular genotyped was performed by Single Specific Primer (SSP) method for HLA class I, and KIR alleles. Chi-square analysis Yates corrected (py) and Fisher exact test (pf) were evaluated when appropriated. Results showed a statistically higher frequency of the activating KIR2DS2 allele in KSpos subjects (93.3%) than either in KSHVpos/KSneg (46.8%, py=0.02), or KSHVneg/KSneg (51.4%,py=0.008) patients, and HC (42.7%, py=0.02). Moreover, the homozygous genotype KIR2DS2+/2DL2- was present in 3/15 KSpos, but in none KSHVpos/KSneg (pf=0.03) or KSHVneg/KSneg (pf=0.02) and only in 1/103 HC (pf=0.006).

Finally there was an higher frequency of KIR2DS2/HLAC-1 functional complex in KSpos (66.7%) than in all the other groups (40.6%, 40.0%, 43.7%, respectively), though not statistically significant.

Results herein, although stemming from analyses performed on a small number of individuals, suggest that HIV and KSHV co-infected subjects in whom activating KIRs and activating KIR-HLA ligands functional complexes are detected, are more prone to develop KS.

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