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SUMMARY OF THESIS

PIERROTTI, Lígia Camera **- Fatores de risco para a ocorrência de viremia pelo herpesvírus 8 humano (HHV-8) em pacientes com AIDS e sarcoma de Kaposi.** São Paulo, 2004. (Tese de Doutoramento - Faculdade de Medicina da Universidade de São Paulo).

RISK FACTORS FOR HUMAN HERPESVIRUS 8 (KSHV/HHV-8) VIREMIA IN AIDS PATIENTS WITH KAPOSI'S SARCOMA

There are several epidemiological, virological and serological lines of evidence suggesting that human herpesvirus 8 (HHV-8) is the cause of Kaposi's sarcoma (KS) and previous studies have provided evidence for a link between HHV-8 replication and KS pathogenesis. However few prospective studies investigating the relationship between HHV-8 replication and possible risk factors have been published.

The present study intended to evaluate the HHV-8 replication and humoral host response to HHV-8 infection in a cohort of AIDS patients with KS assisted at AIDS Outpatient Clinic - Fundação Zerbini, and estimates the prevalence and the risk factors for HHV-8 viremia.

The patients were follow-up periodically along the study for blood collection and clinical data information about HIV/AIDS evolution and KS evolution. Positive HHV-8 viremia was defined by the presence of HHV-8 DNA in peripheral blood mononuclear cells (PBMC) in at least one of two nested-PCR amplifying fragments of 170 bp. or 233 bp. HHV-8 antibodies were detect by either indirect immunofluorescence assay (IFA) for lytic or latency-associated nuclear antigen (LANA) antigens or ELISA for lytic phase-ORF 65 recombinant antigen. To analyze the effects of various factors on the risk of HHV-8 viremia the generalized estimating equation models for cluster data were used.

From March 1998 to July 2000 419 clinical and laboratorial evaluations of 42 AIDS patients with KS were available. HHV-8 viremia was detected in 146 (35.6%) evaluations, seropositivity was higher for antibodies to lytic HHV-8 antigens than antibodies to latent HHV-8 antigens in the population studied (99.0%, 86.3% and 80.1% of seropositivity by IFA-LYTIC, ELISA-LYTIC and IFA-LANA, respectively). The seropositivity for antibodies to lytic HHV-8 antigens preceded the seropositivity for antibodies against the latent antigens.

In the univariate analysis, the positive HHV-8 viremia was associated with KS clinical stage at the study admission (OR 1.93 for visceral KS, $p = 0.009$) and opportunistic infection occurrence (OR 3.04, $p = 0.02$) variables. In the multivariate analysis, the positive HHV-8 viremia was associated with category of exposure to HIV (OR 1.61 for homosexual; OR 3.64 for bisexual; $p = 0.03$), KS clinical stage at "the study admission (OR 2.22 for visceral KS, $p = 0.01$), and specific therapies against KS (OR 3.82 for expectation therapy, $p = 0.01$) variables. There were no association between HHV-8 viremia and the other studied variables (sociodemographic informations, number of cutaneous KS lesions at the study admission, time from KS diagnosis to study admission, CD4+ lymphocyte T count, plasma HIV load, IP, ITRN and ITRNN use, SK clinical evolution, and titres of antibodies against HHV-8).

The largest rate of the HHV-8 DNA detection in PBMC in patients with KS visceral in relationship those with non visceral KS support the use of HHV-8 viremia as a prognostic marker of the tumor in AIDS patients with KS.

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