Retrovirology

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Oral presentation

Virus (KSHV/HHV8) Infection and Genomic Aberrations in Developing AIDS Kaposi's Sarcoma (KS)

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Background

AKS is the most frequent AIDS tumor and like endemic (EKS) always associated with HHV8/KSHV but it is still controversial whether KS represents a monoclonal cell proliferation with distinct recurrent genomic changes or a polyclonal, hyperplastic, reactive process.

Material and Methods

Biopsies of AKS and EKS were compared by triple immunoflourscence for possible stage related phenotypic differences in HHV8 (LANA) infected tumor spindle cells (CD34+SC) and proliferating (Ki67+) cells. Histological tumor areas were alsolaser microdissected and the DNA analyzed by CGH and interphase FISH for cytogenetic changes in early and late stages of tumor development.

Results

LANA and CD34 tumor spindle cells (SC) varied concordantly with stage of AKS and EKS. However among CD34+SC approximately 30–40% were LANA negative, but onlya minor population of LANA cells were CD34- (3–4%) in all KS forms and stages Cell proliferation (Ki67+) was relative constant (4.5–11.5%) at all KS stages but usually more frequent in early AKS and EKS. Most Ki67+ cells were LANA+/CD34+ but a few were LANA+/CD34-. CGH analysis of KS tumors (n = 27) showed mostly apparent random but no recurrent chromosomal aberrations. Fewer chromosomal aberrations were observed in AKS compared to EKS.

Conclusion

Apparently there is either a heterogeneity among SC for HHV8 infection or a continuous recruitment of non-infected precursor SC to the lesions. The LANA+ SC appeared to have a proliferative advantage compared to LANA- SC. Comparison of random chromosomal aberrations in AKS and EKS appears to indicate that genomic instability could be a more important factor for the development of EKS than for AKS. Most likely AKS development is also promoted by various cytokines and growth factors produced during HIV infection and by the compromised state of host immune response in HIV infection.

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