Retrovirology

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Oral presentation

Kaposi (KS) pathogenesis: cell origin, HHV-8 permissiveness, proliferation, genomic instability

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Background

AKS a frequent AIDS tumor is associated with HHV8, but is controversial whether clonal with recurrent genomic changes or mostly reactive proliferation.

Materials and methods

KS biopsies were compared by IFA for LANA, CD34, Ki67, lymphatic endothelial (LEC) marker (LYVE-1) and DNA damage markers (Chk2). After microdissection DNA was also analyzed by CGH for cytogenetic changes.

Results

LANA+ cells varied with frequency of CD34+ cells at all stages of KS. However about 25% of CD34+ cells were LANA- and LYVE-1-. All LANA+ SC were LYVE-1+, but only 75% were CD34+. About 18% of LANA+ SC in early KS were CD34- but LYVE-1+. This LANA+/LYVE-1+/CD34- (resident LEC) cell's decreased from early to late KS. Ki67+ cells were moderate (4.5–11.5%) at all KS stages, but usually higher in early KS. CGH showed Y chrom. deletion in most KS and was the only aberration in early KS, whereas late KS also showed deletions of chrom. 16 and 17, confirmed by FISH. Chk2 which is upregulated at early stage of DNA damage was more frequent in early than late KS.

Conclusion

The presence of CD34+/LANA- cells suggests a continuous recruitment of non infected cells. Some LANA+ cells were CD34-, but LYVE-1+ indicating that resident LEC's are targeted by HHV-8 and transformed to SC. The Y deletion and upregulation of Chk2 in early KS indicates genomic instability from early stage of KS.

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