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Abstract (poster session)

Evidence of Chlamydophila pneumoniae and human herpesvirus 8 in a patient with primary cutaneous anaplastic large-cell lymphoma

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Objectives. The risk factors for the development of cutaneous lymphomas are unknown. Although an infectious etiology has been suspected, the implication of infectious agents in the pathogenesis of cutaneous lymphoproliferative disorders is still controversial. Accumulating evidence suggests that Chlamydia Spp. may play a role in ocular adnexa lymphomas of MALT-type in some geographic areas, and that Human herpesvirus 8 (HHV8), known as Kaposi's sarcoma associated herpesvirus and also to other malignancies such as Multicentric Castleman's Disease, and Primary Effusion Lymphomas, have been found in the so called tumor spindle cells, and keratinocytes, showing a broad in vivo tropism. We report the case of a HIV-negative patient referred to our hospital with a rapidly enlarging skin tumor on her eyelid region. Methods. Histological analysis on bioptic lesion evidenced a primary cutaneous anaplastic large-cell lymphoma (PCALCL). Tumor cells presented frequent mitotic figures, and were positive for CD3, CD2 and CD30, and negative for CD5, CD56, ALK and CD20, as determined by immunohistochemistry. Considering the histological features of the lesion, its localization, and the high potential of Chlamydia and HHV8 in lymphoma induction, the local and systemic presence of both pathogens was investigated by molecular analyses. Chlamydophila pneumoniae and HHV8 DNA were found in surgical biopsy and in PBMC and both sustained a productive infection. For C. pneumoniae, a fraction of PBMCs was also centrifuged, suspended in RPMI medium and co-cultured with Hep-2 cell line (ATCC CCL-23), to increase bacterial inclusions. Amplification fragments, corresponding to C. pneumoniae 16S rRNA gene, were found both in tumor tissue and in co-cultured PBMCs. The same specimens harbored HHV8 sequences by PCR or real time quantitative PCR (qPCR) specifically designed in three different regions of HHV8 ORF50 and ORF26 genes. C. pneumoniae was no longer detectable in the patient's PBMCs, and HHV8 was no longer sustaining active infection, suggesting that the virus entered the latent phase. Conclusion. Chlamydia Spp and HHV8 may play a role in oncogenesis for their tendency to cause persistent infections, cell proliferation and transformation. Our data present evidence of a concurrent active infection by C. pneumoniae and HHV8 in a PCALCL patient, suggesting for the first time their potential association with the development of the PCALCL in non-immunocompromised subjects.