

Poster presentation

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## A retrospective analysis of HIV-associated Kaposi's sarcoma in patients with non-detectable HIV viral loads and CD4+ counts greater than 300 cells/ $\mu$ L

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In the HAART era, the incidence of HIV-associated Kaposi's sarcoma (KS) has declined precipitously for countries with ready access to anti-retrovirals. Patients with newly diagnosed KS typically have low CD4+ counts and high HIV viral loads (VLs). Several recently published reports (*NEJM* 2007;357:1352–3, *NEJM* 2008;358:535–6, *AIDS* 2008;22:551–2) suggest that patients with KS may require treatment despite effective HAART and that KS may progress even in the setting of suppressed HIV VLs and adequate CD4+ counts.

To investigate this further, we examined the demographic and HIV-related clinical data of 91 KS patients attending our HIV outpatient clinic between 1996 and 2008. Twenty of the 91 patients (22%) had either newly diagnosed KS or persistent or progressive disease despite CD4+ counts >300 cells/ $\mu$ L and undetectable HIV VLs. Nineteen of these 20 patients were males. The median age was 43 years (range 25 to 59), the median time since onset of HIV infection was 156 months (range 24 to 276) and the median duration of HAART was 60 months (range 12 to 144). Nine (45%) patients had a KS stage of T0S0, five (25%) had T1S0, three (15%) had T0S1, three (15%) had T1S1; no patient had KS visceral involvement. All 20 patients received specific anti-KS treatment in addition to HAART including liposomal doxorubicin (65%), paclitaxel (15%), radiation (20%) and novel clinical agents (25%). After a median follow up of 52 months (range 6 to 120), eight (40%) had complete regression of KS, six

(30%) had partial regression, one (5%) had stable disease and five (25%) had KS progression. In this retrospective analysis, a substantial proportion of KS patients had undetectable HIV VLs and CD4+ counts greater than the level typically associated with opportunistic infections and required systemic anti-ks therapy. This raises important questions regarding the mechanisms of KS progression and the management of KS in such patients.