Persistence of anal squamous intraepithelial lesions and anal HPV infection in HIV-infected patients despite immune restoration under cART


To cite this version:


HAL Id: inserm-00663882
http://www.hal.inserm.fr/inserm-00663882
Submitted on 27 Jan 2012

HAL is a multi-disciplinary open access archive for the deposit and dissemination of scientific research documents, whether they are published or not. The documents may come from teaching and research institutions in France or abroad, or from public or private research centers.

L’archive ouverte pluridisciplinaire HAL, est destinée au dépôt et à la diffusion de documents scientifiques de niveau recherche, publiés ou non, émanant des établissements d’enseignement et de recherche français ou étrangers, des laboratoires publics ou privés.
Persistence of anal squamous intraepithelial lesions and anal HPV infection in HIV-infected patients despite immune restoration under cART

C Piketty1, E Lanoy2, A Si-Mohamed1, B Cochand-Priolet3, S Trabelsi2, P-M Girard6, R Tubiana5, L Abramowitz6, E Tartour1,8, C Rouzioux7,8, L Weiss1,8, D Costagliola2,5, The Valparaiso Study Group

From 12th International Conference on Malignancies in AIDS and Other Acquired Immunodeficiencies (ICMAOI)
Bethesda, MD, USA. 26-27 April, 2010

Background
A high prevalence of anal squamous intraepithelial lesions (ASIL) and HPV infection have been observed in HIV-infected MSM in the pre-cART era. To date, the impact of cART on the natural history of HPV infection and ASIL is poorly documented.

Methods
94 HIV-infected MSM naïve of cART were enrolled in a longitudinal study before starting a first-line regimen of cART. Each patient provided anal samples for cytology, histology, and HPV DNA testing at baseline, month 12, and month 24 of cART. HPV DNA was detected by real-time PCR and Roche Linear Array assay. Anal cytologic was processed by the Thin Prep™ method (Hologic). CD4+ and CD8+ T cell responses to HPV-16 E6 and E7 proteins were measured in a subgroup of individuals exhibiting HPV-16 anal infection at inclusion.

Results
Prevalence of low-grade SIL, high-grade SIL, and HPV infection was similar at M12 compared to baseline. Among patients with normal cytology and/or histology at baseline, 44% progressed to SIL at M12 whereas 31% of patients with ASIL at baseline exhibited a regression at M12. Specific anti-HPV CD4 T cell responses were mostly undetectable both at baseline and M12. (Table 1 and 2)

At month 12, prevalence of anal HPV DNA detection was similar than at baseline. High-risk HPV was detected at month 12 in 92% of the patients with high-risk HPV infection at baseline. Low-risk HPV was detected at month 12 in 91% of the patients with low-risk HPV infection at baseline. HPV-16 and HPV-18 were detected at month 12 in 13% and 3.7% of patients with no HPV-16 and HPV 18 infection at baseline, respectively. HPV-16 was detected in 100% and 70% of high-grade SIL at baseline and month 12, respectively.

Table 1 The median age of the patients was 39.7 years (33.2-43.5). Baseline and month 12 cytologic and/or histologic results

<table>
<thead>
<tr>
<th>CD4/mm³ median (Q1-Q3)</th>
<th>Plasma HIV RNA log₁₀ copies/mL</th>
<th>VL &lt;50</th>
<th>Prior AIDS event</th>
<th>Visible lesion</th>
<th>Presence of condyloma</th>
<th>Anal SIL</th>
<th>Low-grade SIL; High-grade SIL</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline 299 (242 – 342) 48 (4.17 – 5.26) 1% 4 (4%)</td>
<td>40/94</td>
<td>(43%)</td>
<td>23/94 (25%)</td>
<td>51 (54%)</td>
<td>30 (32%); 8 (9%)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>M12 500 (411 – 575) 1.6 (1.6 – 1.6) 93%</td>
<td>25/71</td>
<td>(35%)</td>
<td>5/71 (7%)</td>
<td>41 (58%)</td>
<td>24 (34%); 10 (14%)</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

*Correspondence: christophe.piketty@egp.aphp.fr
1 Hôpital Européen Georges Pompidou, Paris, France
Full list of author information is available at the end of the article
Conclusion
Our results demonstrate a high prevalence and incidence of ASIL and anal HPV infection in HIV-infected MSM despite CD4 reconstitution under cART. These data suggest that all HIV-positive MSM, even under antiretroviral therapy, remain at risk of anal SIL.

Acknowledgements
This article has been published as part of Infectious Agents and Cancer Volume 5 Supplement 1, 2010: Proceedings of the 12th International Conference on Malignancies in AIDS and Other Acquired Immunodeficiencies (ICMAOI). The full contents of the supplement are available online at http://www.biomedcentral.com/1750-9378/5?issue=S1.

Author details
1 Hôpital Européen Georges Pompidou, Paris, France. 2 INSERM U943 - UPMC UMR 943, Paris, France. 3 Hôpital Lariboisière, Paris, France. 4 Hôpital Saint Antoine, Paris, France. 5 Hôpital Pitié Salpêtrière, Paris, France. 6 Hôpital Bichat-Claude Bernard, Paris, France. 7 Hôpital Necker, Paris, France. 8 Université Paris 5, René Descartes, Paris, France.

Published: 11 October 2010

Table 2 Baseline and month 12 virological results

<table>
<thead>
<tr>
<th></th>
<th>Number of HPV</th>
<th>Number of high-risk and low-risk type</th>
<th>High risk HPV</th>
<th>HPV-16</th>
<th>HPV-18</th>
<th>HPV-16 DNAlog_{10} copies/10^{6} cells</th>
</tr>
</thead>
<tbody>
<tr>
<td>Baseline</td>
<td>5 (2 – 7)</td>
<td>3 (2 – 5); 2 (1 – 4)</td>
<td>83 (90%)</td>
<td>49 (53%)</td>
<td>28 (30%)</td>
<td>6.1 (5.3 – 7.1)</td>
</tr>
<tr>
<td>M12</td>
<td>5 (2 – 6)</td>
<td>3 (1 – 4); 2 (1 – 4)</td>
<td>59 (87%)</td>
<td>28 (41%)</td>
<td>15 (22%)</td>
<td>6.1 (2.0 – 7.2)</td>
</tr>
</tbody>
</table>

Submit your next manuscript to BioMed Central and take full advantage of:

- Convenient online submission
- Thorough peer review
- No space constraints or color figure charges
- Immediate publication on acceptance
- Inclusion in PubMed, CAS, Scopus and Google Scholar
- Research which is freely available for redistribution