Case of invasive adenocarcinoma of the cervix in a human immunodeficiency virus and schistosome co-infected patient

I read with interest the case report on invasive adenocarcinoma of the cervix in a human immunodeficiency virus (HIV) and schistosome co-infected patient. I would like to make some comments. Schistosomiasis is endemic in Limpopo, Mpumalanga and KwaZulu-Natal. Prevention rests on two legs: water sanitation and the screening of schoolchildren. Because of the high cost of water sanitation, the main preventive measure is screening and treatment at school level. Unfortunately, no systematic prevention programme has been implemented in South Africa.

Treatment with praziquantel kills the adult helminths, but has no effect on the ova. Therefore, early treatment before the ova are deposited in the tissue is essential. There is an inflammatory reaction in the early stage in the Splendore-Hoeppli granuloma that surround the ova which translates clinically into the so-called “sandy patches” of the anal and urogenital tract. Like any breach in the mucosae resulting from any sexually transmitted infection, including HIV and human papillomavirus (HPV), those created by schistosomes may promote the transmission of HIV and HPV. However, the two publications on the topic have been cross-sectional, i.e. showing an association, but no firm causal relation.

The link between HPV and anogenital pre-invasive and invasive lesions is firmly established. The contribution of HIV co-infection to the transition of pre-invasive into invasive lesions remains elusive. The influence of urogenital schistosomiasis (UGS) on cervical carcinogenesis, with or without HIV infection, seems weak at best.

Over a period of six years, a total of 706 cases of schistosomiasis were diagnosed histologically (Figure 1). The female to male ratio was 3.7. Fifty five (10%) of females were HIV-infected. Four fifths were cases of UGS in women, with close to half affecting the cervix. Schistosoma haematobium was found in 94.8% and S. mansoni in 5.2% of the cervical specimens.

Table I: Distribution according to cervical pathology and human immunodeficiency virus status

<table>
<thead>
<tr>
<th>Pathology</th>
<th>HIV-uninfected, n (%)</th>
<th>HIV-infected, n (%)</th>
<th>Statistical significance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cervicitis*</td>
<td>56 (31.3)</td>
<td>9 (17)</td>
<td>NS**</td>
</tr>
<tr>
<td>Low-grade intraepithelial lesion</td>
<td>17 (9.5)</td>
<td>6 (11.3)</td>
<td>NS</td>
</tr>
<tr>
<td>High-grade intraepithelial lesion</td>
<td>49 (27.4)</td>
<td>20 (37.7)</td>
<td>NS</td>
</tr>
<tr>
<td>Invasive cancer</td>
<td>57 (31.8)***</td>
<td>18 (34)****</td>
<td>NS</td>
</tr>
<tr>
<td>Total</td>
<td>179/232 (77.2)</td>
<td>53/232 (22.8)</td>
<td></td>
</tr>
</tbody>
</table>

*Schistosomal ova with inflammatory reaction
**Not significant (95% confidence interval)
***3 adenocarcinomas (5.4%)
****1 adenocarcinoma (5.6%)
Table I illustrates the distribution according to different types of cervical pathology and HIV status. There is no statistical significance between the proportions. There is no convincing evidence that cervical schistosomiasis promotes cervical carcinogenesis. The view that the transmission of HIV could be favoured by breaches in the genital mucosa supports the need for the early detection and treatment of schistosomiasis in schoolchildren.

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**References**


