A case of a false positive result on a home HIV test kit obtained on the internet

There are two major reasons to diagnose asymptomatic HIV infection: to facilitate timely initiation of antiretroviral therapy, and to reduce the chance of onward transmission. A negative test offers an opportunity for preventive health promotion. All these aspects of testing require follow up by suitably trained personnel. We describe a case illustrating the hazards of self testing for HIV.

A 31 year old British heterosexual man attended the genitourinary medicine clinic requesting an HIV test. His last sexual contact was 3 weeks earlier with a female partner of 3 months. He had recently learnt that she had had a previous male partner who had had African sexual partners and therefore may be at higher risk of having HIV infection. He obtained a home HIV test kit ("Discreet" HIV Home Test Kit, Seville Marketing Ltd) from a Canadian based internet site and this result was positive. On further inquiry he gave a history of sore throat and swollen cervical lymph nodes 2 months previously, although these symptoms had largely resolved. He had never tested for HIV before and had no other significant risk factors.

We requested an HIV test on the patient; the result was negative. We repeated the test after 3 months and again it was negative, confirming that the patient was not infected at the time he performed the home HIV test. The current HIV screening test used by our centre uses both HIV antibody and p24 antigen detection and is known to detect HIV infection 3–12 weeks after infection. Given that he was now symptom free with HIV infection 3–12 weeks after infection.

Confirming that the patient was not infected and had no other significant risk factors.

Resolved. He had never tested for HIV before although these symptoms had largely

Case 2

A 39 year old homosexual man presented to the accident and emergency department with fever, ulcerative gingivitis, and maculopapular rash, claiming to have been diagnosed as HIV positive at another hospital 2 years previously. A third generation HIV test, Abbott AXSYM HIV 1/2 gO (antibody only), was negative but reactive with the fourth generation assay, Abbott HIV Ag/Ab Combo (antibody and p24 antigen combined).

Comment

The ability to diagnose PHI has always required a high index of suspicion and a keenly taken history, and if missed the next opportunity for testing may not be until years later when the patient presents in ill health, with symptomatic HIV or even AIDS. Clearly, a missed diagnosis of PHI may have a deleterious effect on the individual’s prognosis, but there may also be significant public health consequences, as early infection is a core factor in the propagation of an epidemic because of high viral burden and de facto risk taking sexual behaviour. Indeed, early detection of PHI probably represents the
single most important method of slowing the spread of HIV within populations, with mathematical modelling indicating that eliminating high infectivity in early infection has more effect than at any other disease stage. Thus, the diagnosis of PHI at-risk individuals has considerable advantages in both individual and public health terms. These two cases demonstrate how easy it can be to disregard such patients as having factitious HIV infection and are a gentle reminder that a negative antibody test does not necessarily exclude PHI. Healthcare professionals must continue to be alert to the less common clinical manifestations of PHI, be aware of the particular assays used in their own laboratory, and because no combination of symptoms is 100% sensitive or specific, diagnostic procedure must be broad and inclusive.

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References


Chlamydia trachomatis PCR positivity and inflammatory changes on cervical cytology

The presence of genital infection does not increase the likelihood of an inadequate Papanicolaou (Pap) test. Conversely, testing for Chlamydia trachomatis at the time of routine cytological screening presents an opportunity to detect asymptomatic genital tract infection. The PreservCyt fixative fluid (Cytac Corporation, Boxborough, MA, USA) used for the ThinPrep Pap test (Cytac Corporation) can be used for detection by the polymerase chain reaction (PCR) of C. trachomatis. This presents an opportunity to study the correlation between the chlamydia result and the Pap test finding.

We retrospectively reviewed all routine requests for chlamydia PCR from the ThinPrep samples sent to our laboratory over a year. Data were collected on the woman’s age, chlamydia PCR result, result of genital tract cultures if performed on the same date, and Pap test result. Data on the Pap test included presence or absence of an epithelial cell abnormality either high grade (HGEA) or low grade (LGEA), whether the Pap was inflammatory and the presence or absence of an inflammatory cell count. Cervical cultures collected in PreservCyt transport medium were processed for C. trachomatis using the automated Cobas Amplicor (Roche Diagnostic Systems) and the method by Bianchi et al. Over the study period, 733 samples were received, of which 23 (3.1%) had C. trachomatis DNA detected by PCR. Comparison of the women with chlamydia infection and those without chlamydia infection is shown in table 1. There was no statistical difference in the presence of high or low grade epithelial abnormalities, recognition of other pathogens, or age of the women; however, 26% of women with chlamydia had an inflammatory Pap test compared to 9% of women without chlamydia (p<0.01).
The association of inflammation on Pap testing and chlamydial infection has been previously examined with variable methodologies and findings. We utilised the same sample (ThinPrep) for determining both the presence of inflammatory changes on Pap test and chlamydial infection and found a positive association between the two despite a low prevalence population. Our study confirms the feasibility of performing chlamydial PCR from liquid based cytology samples in a routine diagnostic setting. Testing for chlamydia should be considered in women with inflammatory Pap tests for which there is no other explanation.

### Table 1: Comparison of women with and without chlamydial infection

<table>
<thead>
<tr>
<th>Positive C trachomatis</th>
<th>Negative C trachomatis</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>PCR</td>
<td>PCR</td>
<td></td>
</tr>
<tr>
<td>Median age</td>
<td>Median age</td>
<td></td>
</tr>
<tr>
<td>24 (range 19–40)</td>
<td>28 (range 15–68)</td>
<td>0.182</td>
</tr>
<tr>
<td>LGEA/HGEA</td>
<td>LGEA/HGEA</td>
<td>0.37</td>
</tr>
<tr>
<td>5 (22%)</td>
<td>106 (15%)</td>
<td></td>
</tr>
<tr>
<td>Other pathogens</td>
<td>Other pathogens</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>1 (17%)</td>
<td>12 (18%)</td>
<td></td>
</tr>
<tr>
<td>Inflammation on Pap test</td>
<td>Inflammation on Pap test</td>
<td></td>
</tr>
<tr>
<td>6 (26%)</td>
<td>65 (9%)</td>
<td></td>
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<tr>
<td>LGEA, low grade epithelial abnormalities; HGEA, high grade epithelial abnormalities.</td>
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Cardiovascular syphilis in HIV infection: a case-control study at the Institute of Sexually Transmitted Diseases, Chennai, India

It is known that HIV co-infection with syphilis may accelerate the onset of gummata and neurosyphilis and increase their severity. However, this has only been reported for cardiovascular syphilis in two previous cases. This case-control study deals with a total of 14 HIV seropositive and 100 HIV 1 and 2 seronegative individuals with syphilis, who were seen in our clinic between June 2000 and May 2001. Of the 14 HIV seropositive individuals, 12 were reactive for VDRL (venereal disease reference laboratory) and TPHA (Treponema pallidum haemagglutination assay) and two had primary syphilis confirmed by dark field examination for T pallidum. Of the 100 HIV seronegative individuals, 85 had reactive VDRL and TPHA and 15 had primary syphilis confirmed by dark field examination. The prevalence of cardiovascular syphilis in the HIV seropositive and seronegative groups was 14.3% and 2%, respectively (OR 8.2, 95% CI 1.1 to 61.5).

Two HIV seropositive individuals with cardiovascular syphilis had aortic root dilatation while the two HIV seronegative individuals had aortic aneurysm. The HIV seropositive individuals were asymptomatic with regard to cardiac status but one HIV seronegative individual had chest pain and the other was asymptomatic. None in the HIV seropositive group had aortic root dilatation (p<0.01). There was a theoretical possibility that aortic root dilatation could be a manifestation of HIV or opportunistic infections involving the heart. A parallel study done on cardiovascular involvement in HIV seropositive individuals from the same institute during the same time interval had revealed that none of the 61 non-syphilitic HIV seropositive individuals had aortic root dilatation, compared with 2 out of 14 with syphilis (p<0.01; paper in preparation).

The mean duration of diagnosing cardiovascular syphilis from the time of acquiring syphilis was 40 months (27 and 53) in the HIV seropositive group and 102 months (p=0.053) in the HIV seronegative group. The mean age of the HIV seropositive individuals who had cardiovascular syphilis was 31.5 years (29 and 34) and that of HIV seronegative individuals was 45.5 years (44 and 47). The shorter duration for diagnosing cardiovascular syphilis from the time of acquiring syphilis for the HIV seropositive group (40 months) compared with the HIV seronegative group (102 months) could be explained by the fact that HIV hastens the progression to late syphilis, which might be due to an alteration to the immune system. It could also be possible that HIV infected individuals seek medical attention because of opportunistic infections, which might have led to the earlier diagnosis of cardiac lesions because the two individuals with aortic root dilatation were asymptomatic with regard to cardiac status. The difference in the clinical manifestation of cardiovascular syphilis between these two groups could not be explained at this point of time.

### Contributions

MM designed the study, collected the data, interpreted the results, and analysed the results and statistics; SKG contributed to collecting data, interpretation of results and laboratory collaboration.

### Acknowledgements

We thank M Muthu, retired Director and Professor of Anatomy, for his valuable guidance. We also thank B Muthukumar, Associate Professor of Cardiology, Madras Medical College, Chennai, for his active participation and guidance in performing and interpreting ECHO and ECG.

### References

NNRTIs was only confirmed after weeks of unsuccessful therapy by further resistance testing.

My patient has alerted all her friends in Nigeria as to the real nature of combivir and advised them to stop using it for breast enhancement. She has also told me that she believes the doctor in Nigeria who prescribed these drugs may have had this activity terminated.

Because no one I have spoken to has come across this particular misuse of antiretroviral therapy I felt it was worth highlighting to a wider audience in the hope that such practises may be addressed.

Acknowledgements
This patient has agreed to publish details of her case to help prevent recurrent misuse of this drug.

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CORRECTION
The order of the authors of the paper by Götz et al on page 24 of the February 2005 issue (HM Götz et al. A prediction rule for selective screening of Chlamydia trachomatis infection. Sex Transm Infect 2005;81:24–30) were wrong. The order should have been as follows: HM Götz, JEAM van Bergen, Ik Veldhuijzen, J Broer, CJPA Hoebe, EW Steyerberg, AJJ Coenen, F de Groot, MJC Verhooren, DT van Schaik, and JH Richardus.

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