Methicillin resistant *Staphylococcus aureus* (MRSA) balanoposthitis in an insulin dependent diabetic male

**Editor,—**Balanoposthitis is a common condition affecting 11% of the male attendees at GUM clinics. It is an inflammation of the glans penis and the prepuce, and its causes include bacterial and yeast infections, parasitic infestations, trauma, and irritants. However, to our knowledge, no case has been reported to be caused by MRSA.

A 49 year old insulin dependent diabetic man was an inpatient for repair of an upper jaw fracture developed a penile itch with swollen foreskin, which was difficult to retract, together with longitudinal fissures on the upper jaw fracture developed a penile itch with swollen foreskin, which was difficult to retract, together with longitudinal fissures on the prepuce and subpreputial discharge. In his recent past he had had two incidents of unprotected sexual intercourse with two known females. He was clinically diagnosed as having candida balanitis and was commenced on clotrimazole cream, which did not produce a clinical response over the course of a week. The swabs taken before the commencement of clotrimazole cream failed to grow candida; however, MRSA resistant to erythromycin, penicillin, and fluocxinolone benzyl ether was isolated.

Screening tests for chlamydia, gonorrhoea, and trichomonas were negative. A 10 day course of mupirocin 2% ointment completely resolved his symptoms. Subpreputial swab after treatment was negative.

MRSA has been a well recognised cause of hospital acquired infections worldwide since it was first detected in Europe in the 1960s. The organism can survive for long periods in both the hospital and the home environment and can colonise the skin, nose, or throat of patients and healthcare staff. Several reports have suggested that diabetic patients are more susceptible to *Staphylococcus aureus* bacteremia MRSA has been isolated from different sites in diabetic patients but not the genitalia. MRSA rarely invades intact skin; however, it can give rise to severe infections—for example, wound infection, bacteraemia, endocarditis, and osteomyelitis.

This case illustrates the fact that MRSA is an organism to consider in patients who develop balanoposthitis while in hospital or shortly after discharge especially those whose immune system is incompetent.

There may be implications of spread of MRSA in the community for sexual contacts of patients carrying MRSA in the genital area.

**Contributors:** Both authors managed the patient and wrote the manuscript.

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**Letters to the Editor**

Chlamydia trachomatis reinfection rate: a forgotten aspect of female genital chlamydial management

**Editor,—**Hillis et al reported that repeated episodes of infection of female genital tract with *Chlamydia trachomatis* increase the risk of hospital admission for pelvic inflammatory disease and ectopic pregnancy. The first diagnosed attack of genital infection with chlamydia presents the clinician with a unique opportunity to implement measures to minimise the risk of reinfection—that is, health promotion and contact tracing.

During April–June 1998 we reviewed the case notes of female patients who were diagnosed with genital chlamydia at Leicester Royal Infirmary and Derbyshire Royal Infirmary GUM clinics in the year 1996 for evidence of repeat episode of genital chlamydia. We also noted the following data: age at presentation with the first episode of infection, time for presentation with reinfection, test of cure if performed, co-infection with gonorrhoea, review by health adviser, contact(s) traced, and treatment in the first 3 months after diagnosis. For the purpose of the study we defined reinfection as a patient testing positive for genital chlamydia 30 days or more after the completion of treatment. We also looked at the genital chlamydia treatment protocols in both clinics. A total of 540 female patients were diagnosed with chlamydia (311 at Leicester and 229 at Derby). The patients’ mean age at first episode was 22.6 years for Leicester and 23.4 years for Derby. The health advisers had made contact with 94.5% (294) in Leicester and 97.8% (224) in Derby; 85.2% (265) of the patients diagnosed at Leicester returned at 30 days or more and were retested for chlamydia compared with 87.3% (200) at Derby; 9% (24) episodes of repeat infection were identified in Leicester group compared to 17% (34) episodes in the Derby cohort. The mean period for presentation with reinfection was 9.4 months (range 3–25) at Leicester and 9.8 months (range 2–24) at Derby. At Leicester the contacts of 66.5% (207) patients were traced and treated compared to 64.6% (148) at Derby. A test of cure was performed on 282 patient in Leicester (where it was routine practice); 2.5% (seven) were found to be positive for chlamydial infection, while the test of cure was performed on 22 patients in Derby (where it was performed selectively) revealed no positive cases.

Of the reininfected patients 58.3% (14) at Leicester were reininfected because of failure to trace and treat their partner(s) compared to 35.5% (12) at the Derby clinic.

Both clinics manage genital chlamydia with what was considered standard treatment and perform contact tracing wherever possible. Two reininfected patients from each clinic were also co-infected with gonorrhoea.

Other risk factors for reinfection—for example, ethnic origin, number of sexual partners, were not analysed as these data was not discernible from the notes.

This retrospective study highlights the fact that a substantial number of patients get reininfected with chlamydia despite health education and counselling by health advisers. Though the figures (66.5% and 64.6%) for partner notification and treatment were close to that proposed by the Central Audit Group (70%), the proportion of cases traced and treated still too high. Does the message that repeat episodes of genital chlamydia are more damaging get through to our patients or do we need a new health education strategy?

Currently, as the success of management of genital chlamydia is evaluated by the level of contact tracing, the number of patients referred to health advisers, and number of contacts per index patient seen and treated, it is vital that we become even more skilled in the near future as all specialist registrars and probably
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Detection of chlamydia on meatal swabs

EDITOR,—The advent of ligase chain reaction (LCR) and other DNA technologies and further interventional technology has allowed risk reduction strategies to be considered by HIV status. A total of 792 homosexual men (median age 35 years) completed a confidential questionnaire (estimated response rate 50–60%8); 126 (16.0%) were HIV positive, 477 (60.2%) HIV negative, while 169 (21.3%) had never had an HIV test (data missing for 20 men). Just over half the men (54.7%) reported they were currently in a relationship with another man; this did not differ significantly by HIV status (p=0.1).

Our analysis focused on how sexual risk behaviour varied both by HIV as well as by relationship status. For HIV negative and positive men, UAI was classified as either discordant (UAI with a partner of the same HIV status) or non-discordant (UAI with a partner of unknown or discordant HIV status). Men reporting more than one UAI partner were classified as discordant only if all UAI partners were of the same HIV status as themselves. Men also indicated whether they had had UAI with a main partner only. By way of comparison, just over half the men (54.7%) reported they were currently in a relationship with another man; this did not differ significantly by HIV status (p=0.1).

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Men reporting UAI with a partner of unknown or discordant HIV status. Non-concordant UAI was more likely to report discordant HIV status. Non-concordant UAI was less likely to report concordant HIV status.

Table 1

<table>
<thead>
<tr>
<th>Type of partner for UAI</th>
<th>HIV negative men (n=477)</th>
<th>HIV positive men (n=126)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Main only</td>
<td>Casual†</td>
</tr>
<tr>
<td>Men in a relationship reporting</td>
<td>n=276</td>
<td>n=63</td>
</tr>
<tr>
<td>Concordant UAI‡</td>
<td>27.1 (75)</td>
<td>1.5 (4)</td>
</tr>
<tr>
<td>Non-concordant UAI</td>
<td>8.0 (22)</td>
<td>6.5 (18)</td>
</tr>
<tr>
<td>Total</td>
<td>35.1 (97)</td>
<td>8.0 (22)</td>
</tr>
<tr>
<td>Men not in a relationship reporting</td>
<td>n=199</td>
<td>n=63</td>
</tr>
<tr>
<td>Concordant UAI</td>
<td>2.5 (5)</td>
<td>2.5 (5)</td>
</tr>
<tr>
<td>Non-concordant UAI</td>
<td>1.5 (3)</td>
<td>16.1 (32)</td>
</tr>
<tr>
<td>Total</td>
<td>4.0 (8)</td>
<td>18.6 (37)</td>
</tr>
</tbody>
</table>

*Data on UAI or relationship status missing for two HIV negative men.
†Men reporting IAU with a partner of unknown or predominantly with a partner of unknown HIV status.
‡Concordant UAI whether they were in a relationship or not. Concordant UAI was less likely to report concordant HIV status. Non-concordant UAI was more likely to report discordant HIV status.

Concordant UAI among negative men may have been only established with confidence if both men were tested for HIV together. For this reason it is difficult for HIV negative men to establish concordance with a casual partner. On the other hand, HIV positive men can establish concordance with it whether they were in a relationship or not was confirmed in a multivariate model. With HIV status and relationship as independent variables and concordant UAI as the dependent variable, the interaction between HIV status and relationship was highly significant (p<0.001).

Seroconcordance among negative men can only be established with confidence if both men are tested for HIV together. The observation that HIV negative men were more likely to report concordant UAI whether they were in a relationship or not was confirmed in a multivariate model. With HIV status and relationship as independent variables and concordant UAI as the dependent variable, the interaction between HIV status and relationship was highly significant (p<0.001).

These data provide further evidence that HIV positive and negative homosexual men have both adopted HIV risk reduction strategies, including the less, high risk sexual behaviour (that is, non-concordant UAI) was reported. Overall, non-concordant UAI was reported by 15.8% (75) of HIV negative and 27.6% (266) of HIV positive men (p<0.001). No significant differences were seen when stratified by either relationship or HIV status (table 1). In the multivariate model there was no significant association between non-concordant UAI and either HIV status (p=0.4) or being in a relationship (p=0.7).

Non-concordant UAI was usually reported with a casual partner with one notable exception. HIV negative men in a relationship were equally likely to report non-concordant UAI with a main partner alone (8.0%) as with a casual partner (6.5%) highlighting the continuing risk for HIV transmission between regular partners. However, for most men the risk of HIV transmission occurred in the context of a casual sexual encounter. Surveys conducted in the gyms in 1998 and 1999 revealed similar patterns of sexual risk behaviour (data available from authors).

Conclusions: HIV negative and positive homosexual men have adopted different strategies for reducing the risk of HIV transmission with their sexual partners. HIV negative men predominantly reported concordant UAI with a main partner in the context of a relationship while HIV positive men were more likely to report discordant UAI with a casual partner. HIV prevention programmes need to reinforce risk reduction strategies, tailored to a person’s HIV status, while simultaneously addressing high risk sexual behaviour.

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A mobile phone text message and Trichomonas vaginalis

EDITOR—Over the past decade vast numbers of the general population have accepted the use of mobile phones and text messaging as the mechanism for contact tracing as far as I am aware has not been reported previously.

A 26 year old African-Caribbean man presented to our clinic and informed us that his girlfriend had attended a GUM clinic but unfortunately he did not know why. However, we informed him that he had a test message on his mobile. He duly brought up the message, which gave the woman’s clinic number and the KC60 diagnosis of C6A.

On examination there were no abnormalities seen, there were no polymorphs on microscopy, swabs for gonorrhoea, chlamydia, and trichomonas were all clear. He was treated with a 5 day course of metronidazole as per MSSVD guidelines.

If this patient had turned up without a contact slip, epidemiological treatment of chlamydia is unlikely to have been instituted and contact tracing would have been impossible. Thanks to the use of text messaging on this man’s mobile phone, appropriate treatment was initiated. Certainly patients and health advisers appreciate the security offered by mobile phones (no other family members can take the calls), the instant access, and it avoids additional paperwork. The use of text messaging and mobile phones for contact tracing may be considered as an adjunct to contact slips in GU clinics.

A NEWELL

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Chaperoning male patients

EDITOR—It was delightful to see the letter by Fisk et al in the journal. My staff and I were becoming alarmed at the suggestion that male patients should have a chaperone when they are being examined by a male doctor. Was common sense finally leaving the specialty? There are thousands of consultations taking place throughout the country, in both primary and secondary care, where sex issues are discussed. These often include a genital examination, and just because there is a problem found with one or two individual patients or doctors it doesn’t mean the whole national service has to be turned upside down. Surely, the last thing an overworked, under pressure, genitourinary medicine service needs is to have another section of its skilled staff standing idly by in a room, while either a consultation or examination is taking place. I have never found any difficulty in taking the swabs on my own, and labelling the stuff myself, and have never felt the need for another person handing me things during a male examination. Indeed, I could easily see that interfering with the process at times, as there are some issues patients feel more comfortable discussing on a one to one basis, and they can feel embarrassed and hindered if there is a chaperone present.

An occasional complaint is a small price to pay for the 99.9% otherwise effective consultations that occur. It’s lovely to see work like this published, as it becomes more widely visible in the evidence base that says we don’t need this
Lichen sclerosus of the glans is significantly associated with penile carcinoma

EDITOR,—We read with interest the article by Riddell et al on 66 men with penile lichen sclerosus (PLS) attending a department of genitourinary medicine.1 In this study, the authors found no cases of malignancy. We have previously reported a retrospective study on the incidence of cancer on 86 cases of PLS retrieved from our histopathological files over a 10 year period (1987–97).2 In that study, five cases showed malignant transformation—namely, squamous cell carcinoma (SCC) (three cases), in situ carcinoma (one case), and verrucous carcinoma (one case).

Since that report, we decided to interview all PLS patients in order to rule out any further malignancy that occurred over time. Of 86 patients identified, 60 were evaluated at our clinic. Among these, we found three malignant degeneration in 3–6% of women,3 a likely malignant evolution of PLS should be considered. Careful and systematic histopathological evaluation of any ulcerated or indurated plaques developing within PLS is therefore strongly recommended. The association between PLS and cancer may very well be underestimated and there is a need for further investigation that includes long term follow up and routine PCR analysis for HPV infection.

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Table 1 Clinical and histopathological features of eight cases of carcinoma on penile lichen sclerosus

<table>
<thead>
<tr>
<th>Patient No</th>
<th>Age of onset of PLS (years)</th>
<th>Age of onset of Ca (years)</th>
<th>Lag time (years)</th>
<th>Site</th>
<th>Clinical aspect of malignancy on PLS</th>
<th>Histopathology</th>
<th>PCR testing for HPV</th>
</tr>
</thead>
<tbody>
<tr>
<td>1*</td>
<td>41</td>
<td>62</td>
<td>21</td>
<td>glans</td>
<td>fungating heretatic nodule with a white-yellowish hue slightly elevated verrucous papules</td>
<td>SCC well differentiated</td>
<td>negative</td>
</tr>
<tr>
<td>2*</td>
<td>36</td>
<td>59</td>
<td>23</td>
<td>glans</td>
<td>multiple erythematous, indurated, and ulcerated plaques sharply circumscribed, erythematous, and ulcerated plaque</td>
<td>SCC well differentiated</td>
<td>HPV 16</td>
</tr>
<tr>
<td>3*</td>
<td>41</td>
<td>55</td>
<td>14</td>
<td>glans, coronary sulcus glans, coronary sulcus, inner aspect of the foreskin glans</td>
<td>sharply circumscribed, verrucous whitish nodule sharply circumscribed, erythematous, and ulcerated plaque</td>
<td>SCC well differentiated</td>
<td>HPV 16</td>
</tr>
<tr>
<td>4*</td>
<td>39</td>
<td>49</td>
<td>10</td>
<td>glans</td>
<td>sharply circumscribed, erythematous, and ulcerated plaque</td>
<td>SCC well differentiated</td>
<td>HPV 16</td>
</tr>
<tr>
<td>5*</td>
<td>29</td>
<td>47</td>
<td>18</td>
<td>glans</td>
<td>exoplythic verrucous whitish nodule sharply circumscribed, erythematous, and ulcerated plaque</td>
<td>SCC well differentiated</td>
<td>HPV 16</td>
</tr>
<tr>
<td>6</td>
<td>75</td>
<td>85</td>
<td>10</td>
<td>glans</td>
<td>exoplythic whitish and indurated plaque</td>
<td>SCC undifferentiated</td>
<td>negative</td>
</tr>
<tr>
<td>7</td>
<td>66</td>
<td>70</td>
<td>15</td>
<td>glans, coronary sulcus</td>
<td>sharply circumscribed, erythematous, eroded, crustated, and indurated plaque</td>
<td>SCC undifferentiated</td>
<td>negative</td>
</tr>
<tr>
<td>8</td>
<td>33</td>
<td>67</td>
<td>34</td>
<td>glans, coronary sulcus</td>
<td>sharply circumscribed, erythematous, and ulcerated plaque</td>
<td>SCC undifferentiated</td>
<td>negative</td>
</tr>
</tbody>
</table>

*Previously reported cases.1

PLS = penile lichen sclerosus; Ca = carcinoma; PCR = polymerase chain reaction; HPV = human papillomavirus; SCC = squamous cell carcinoma; VC = verrucous carcinoma.


Accepted for publication 15 February 2001

Third trimester screening or safer sex to prevent mother to child transmission of HIV

EDITOR,—Since 1992 Department of Health guidelines have recommended that HIV screening be offered to all pregnant women in areas of high seroprevalence6 but implementation and uptake has been poor. In 1998 an intercollegiate working party recommended that HIV testing be integrated with antenatal screening for other infections assuming that the test should be offered and recommended to all pregnant women in high seroprevalence areas.7 In 1999 the Department of Health extended these recommendations to all regions aiming to reduce neonatal HIV infection by 80% by 2002.8 We present the case of an infant with symptomatic HIV infection, whose mother's antenatal HIV test was negative and discuss the implications.

A 3 month old female, born at term by spontaneous vaginal delivery and breastfed, presented with a 1 week history of increasing respiratory difficulty. Following further deterioration, she was transferred to St Mary's Hospital and ventilated. Pneumocystis carinii pneumonia (PCP) was diagnosed on bronchoalveolar lavage. Anti-HIV antibodies were present in serum and HIV infection was confirmed by the detection of HIV-DNA in peripheral blood mononuclear cells (PBMC) by PCR amplification. HIV-1 infection was confirmed in both parents. Her asymptomatic mother had received antenatal care from the 12th week of gestation and was HIV seronegative at 29 weeks. To investigate a
The HIV antibody test is usually performed at the booking visit with other routine antenatal screens. This allows the parents time to adjust to the diagnosis before delivery, to consider family planning issues and to prepare for the anticipated birth. A practical approach, which may be adopted for safe sibling transmission, is highly prevalent and recognised as a major threat to public health.

Table 1 Peripartum HIV test results

<table>
<thead>
<tr>
<th>Time (in weeks of gestation)</th>
<th>1 T = 12 weeks (“Booking blood”)</th>
<th>2 T = 29 weeks</th>
<th>3 T = 33 weeks (“Booking blood”)</th>
<th>4 T = 13 weeks post partum (child presents)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hospital where blood taken</td>
<td>X Blood was stored and retrospectively tested</td>
<td>Y Index antenatal test (serum not available for repeat retrospective testing)</td>
<td>Y Blood was stored and retrospectively tested</td>
<td>St Mary’s Postnatal test. Blood stored</td>
</tr>
<tr>
<td>HIV antibody screening tests</td>
<td>Clear negative: Detect-HIV®</td>
<td>Weak positive: Murex HIV 1+2®</td>
<td>Strong positive: Abbott Asym HIV 1/2®</td>
<td>Strong positives for IgG and IgA: weak positive IgM (OD/CO) HIV IgG=15.41, IgM=3.14, IgA=4.18. *Note decreasing values for IgM and IgA compared to previous</td>
</tr>
<tr>
<td>HIV specific antibodies tests (CPhL in-house EIA/As)</td>
<td>Clear negatives, (OD/CO) HIV IgG=0.49, IgM=0.36, IgA&gt;0.44</td>
<td>Strong positives, (OD/CO) HIV IgG=12.34, IgM=10.94, IgA&gt;5.28</td>
<td>HIV1 pag p17+, p24++, p55++; pol p31++, p51++, p66++, env gpl40−, gpl120+, gpl160++; HIV2 gp38−</td>
<td></td>
</tr>
<tr>
<td>HIV western blot®</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>41377 Quaniplex HIV-1 RNA 3.0®</td>
</tr>
<tr>
<td>HIV RNA (copies/ml)</td>
<td>Not detected (&lt; Limit of detection)</td>
<td>Cobas Amplicor HIV-1 Monitor v1.5®</td>
<td>—</td>
<td>82400 Cobas Amplicor HIV-1 Monitor v1.5®</td>
</tr>
</tbody>
</table>

*Enzyme immunoassay (EIA) for detection of antibody to HIV-1 and 2. Biochem Immunosystems Inc, Montreal, Quebec, Canada.

1EIA for detection of antibody to HIV-1 (Abbott Murex), Murex Biotech Ltd, Dartford, UK.

2EIA for detection of antibodies to HIV-1 and 2. Abbott Laboratories, IL, USA.

3EIA for detection of antibodies to HIV-1 and 2 (Abbott Murex), Murex Biotech Ltd, Dartford, UK.

4Passive particle agglutination test for detection of antibodies to HIV-1 and 2. Fujirebio Inc, Tokyo, Japan.

5Western blot for detection of antibodies to HIV-1 antigens. Genelabs Diagnostics, Singapore.

6Polymerase chain reaction (PCR) for quantitative detection of HIV-1 RNA. Roche Diagnostics, Branchburg, NJ, USA.

7Signal amplification nucleic acid probe assay for quantitative detection of HIV-1 RNA. Chiron Corp Emeryville, CA, USA.

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<tr>
<td>HIV antibody screening tests</td>
<td>Clear negative: Detect-HIV®</td>
<td>Weak positive: Murex HIV 1+2®</td>
<td>Strong positive: Abbott Asym HIV 1/2®</td>
<td>Strong positives for IgG and IgA: weak positive IgM (OD/CO) HIV IgG=15.41, IgM=3.14, IgA=4.18. *Note decreasing values for IgM and IgA compared to previous</td>
</tr>
<tr>
<td>HIV specific antibodies tests (CPhL in-house EIA/As)</td>
<td>Clear negatives, (OD/CO) HIV IgG=0.49, IgM=0.36, IgA&gt;0.44</td>
<td>Strong positives, (OD/CO) HIV IgG=12.34, IgM=10.94, IgA&gt;5.28</td>
<td>HIV1 pag p17+, p24++, p55++; pol p31++, p51++, p66++, env gpl40−, gpl120+, gpl160++; HIV2 gp38−</td>
<td></td>
</tr>
<tr>
<td>HIV western blot®</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>41377 Quaniplex HIV-1 RNA 3.0®</td>
</tr>
<tr>
<td>HIV RNA (copies/ml)</td>
<td>Not detected (&lt; Limit of detection)</td>
<td>Cobas Amplicor HIV-1 Monitor v1.5®</td>
<td>—</td>
<td>82400 Cobas Amplicor HIV-1 Monitor v1.5®</td>
</tr>
</tbody>
</table>

*Enzyme immunoassay (EIA) for detection of antibody to HIV-1 and 2. Biochem Immunosystems Inc, Montreal, Quebec, Canada.

1EIA for detection of antibody to HIV-1 (Abbott Murex), Murex Biotech Ltd, Dartford, UK.

2EIA for detection of antibody to HIV-1 and 2. Abbott Laboratories, IL, USA.

3EIA for detection of antibodies to HIV-1 and 2 (Abbott Murex), Murex Biotech Ltd, Dartford, UK.

4Passive particle agglutination test for detection of antibodies to HIV-1 and 2. Fujirebio Inc, Tokyo, Japan.

5Western blot for detection of antibodies to HIV-1 antigens. Genelabs Diagnostics, Singapore.

6Polymerase chain reaction (PCR) for quantitative detection of HIV-1 RNA. Roche Diagnostics, Branchburg, NJ, USA.

7Signal amplification nucleic acid probe assay for quantitative detection of HIV-1 RNA. Chiron Corp Emeryville, CA, USA.
A total of 148 male and 153 female patients were tested; 23/148 (16%) swabs from male patients were positive for Chlamydia trachomatis. A sensitivity, specificity, and positive predictive values of 100%, 99%, and 59%, respectively, were observed, compared with LCR (see fig 1). Although EIA tests cost less than LCR, the inferior detection rate for EIA (17 patients screened per case detected) compared with LCR (nine patients screened per case detected) compared with LCR would cost £564 and detect all 11 cases. EIA had been used alone.

In a hypothetical cohort of 100 GUM attendees, with an 11% prevalence of chlamydial infection (as in the present study), testing with EIA would cost £650 and would detect 6.4 of the 11 cases. Testing the cohort with LCR would cost £564 and detect all 11 cases. The additional cost of LCR is thus £199. The additional benefit is 4.6 additional cases detected. The additional cost of LCR per additional case detected is £34.

The clinic in which the study was conducted sees 6000 new attendees annually. Had EIA been used alone, 276 cases of chlamydial infection would have been missed in a one year period, at an estimated cost of over £82 000. A full economic evaluation would require that these long term health and economic benefits be more thoroughly quantified and compared with other uses of NHS resources.

In summary, this study demonstrates that the overall sensitivity of LCR was double that of EIA, the previous standard diagnostic test used. Because of its improved sensitivity and increased case detection rate, the cost of LCR per case detected is equivalent to that of EIA in an urban UK GUM clinic population. Use of LCR as the diagnostic test of choice for both screening and clinical diagnosis in this setting thus represents a cost effective strategy.

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NOTICES

International Herpes Alliance and International Herpes Management Forum

The International Herpes Alliance has introduced a website (www.herpesalliance.org) from which you can download patient information leaflets. Its sister organisation the International Herpes Management Forum (website: www.IHFM.org) has launched new guidelines on the management of herpesvirus infections in pregnancy at the 9th International Congress on Infectious Disease (ICID) in Buenos Aires.

Pan-American Health Organization, regional office of the World Health Organization

A catalogue of publications is available online (www.paho.org). The monthly journal of PAHO, the Pan American Journal of Public Health, is also available (subscriptions: pubsvc@tsp.sheridan.com).


Further details: ECEAR 2001 Conference Secretary, Division of Virology, NEBSC, Bianche Lane, South Mimms, Potters Bar, Herts, EN6 3QG, UK.

International Congress of Sexually Transmitted Infections, 24–27 June 2001, Berlin, Germany

Further details: Congress Partner GmbH, Krausenstrasse 63, D-10117, Berlin, Germany (tel: +49-30-204 500 41; fax: +49-30-204 500 42; e-mail: berlin@cpb.de).

1st Asia Pacific Forum on Quality Improvement in Health Care

The 1st Asia Pacific Forum on Quality Improvement in Health Care will be held from 19–23 September 2001 in Singapore, Australia. Presented by the BMJ Publishing Group (London, UK) and Institute for Healthcare Improvement (Boston, USA), with the support of the Commonwealth Department of Health and Aged Care (Australia), Safety and Quality Council (Australia), NSW Health (Australia) and Ministry of Health (New Zealand).

Further details: quality@bma.org.uk; fax +44 (0) 7383 8609.

41st St Andrew's Day Festival Symposium on Therapeutics

The 41st St Andrew's Day Festival Symposium on Therapeutics will be held on 6–7 December 2001 at the Royal College of Physicians of Edinburgh. Further details: Ms Eileen Srawn, Symposium Co-ordinator (tel: 0131 225 7324; fax: 0131 220 4393; email: e.srawn@rcpe.ac.uk; website: www.rcpe.ac.uk).

10th International Congress on Behchet's Disease will be held in Berlin 27–29 June 2002

Further details: Professor Ch Zouboulis (email: zoubbere@zedat.fu-berlin.de).

5th World Congress of Perinatal Medicine, 23–27 September 2001, Palau de Congressos de Barcelona - Avda Maria Cristina s/n, Barcelona, Spain

Further details: Dr Francesc Figueras, Congress Promotion Secretary (fax: +34 93 451 74 38; www.perinatology2001.com).

Second International Conference on Sexual Health, to be held in Bangkok, Thailand on 23–28 February 2002. Calls for abstracts deadline 1 September 2001

Further details: European Secretariat, Dr Richard Burack (tel: +44 (0) 20 8599 8029; email: siancare@aol.com).

International Conference on HIV/AIDS 16–19 December 2001, Mumbai, India

Further details: Dr Chander P Puri, President, Indian Society for Study of Reproduction and Fertility, Institute for Research in Reproduction, Janghanj Merwanji Street, Parel, Mumbai 400012, India (Tel: 413770 (Direct); 413211–2-4-7; fax: 091-022-4964853 or 091-022-4139412; e-mail: vinchin@bom4.vsnl.net.in OR dirrr@vsnl.com).

10th International Symposium on Human Chlamydial Infection, 16–21 June 2002, in Antalya, Turkey

The scientific programme will encompass the breadth of chlamydial research from clinical and epidemiological studies to molecular and cell biology of all species of Chlamydia. Further details: Professor A Demir Serter, Department of Clinical Microbiology and Infectious Diseases, Ege University, Faculty of Medicine, 35100 Bornova, Izmir, Turkey (Fax: 90 232 343 71 30; e-mail: isHICIX@itsa.ucsf.edu).

20th World Congress of Dermatology, Paris, 1–5 July 2002

Further details: P Fournier, Colloquium, 12 rue de la Croix St Faubin, 75011 Paris, France (ref: +33 1 44 64 15 15; fax: +33 1 44 64 15 16; email: p.fournier@colloquium.fr; website: www.derm-wcd-2002.com).
A mobile phone text message and *Trichomonas vaginalis*

A Newell

*Sex Transm Infect* 2001 77: 225
doi: 10.1136/sti.77.3.225

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