LETTERS TO THE EDITOR


Sir,

Although genital herpes has been recognised as a major sexually transmissible disease for many years, it is important to monitor closely its changing epidemiology. Although Herpes simplex virus type 2 (HSV-2) remains predominantly a genital pathogen, Herpes simplex virus type 1 (HSV-1) has been variably reported as causing between 4·2% and 60% of genital herpetic episodes.1·4 This is of interest, because it has a better prognosis than genital HSV-2 infection.1·5

We recently reviewed serotyping data on genital isolates of HSV since our laboratory began routinely typing all isolates with type-specific monoclonal antibodies. Between June 1985 and December 1988, 2485 genital specimens were received for HSV culture, originating mainly from genitourinary medicine clinics (77·5%), but also from gynaecology departments (10·9%), other hospital sources (7·4%), general practitioners (2·4%), and family planning clinics (1·1%), predominantly from patients with clinically suspected genital herpes. Specimens were transported to the laboratory in virus transport medium, inoculated onto human fibroblast and Vero cell monolayers and observed daily for cytopathic effect. Positive cultures were typed using fluorescein labelled monoclonal antibodies against HSV-1 and HSV-2 (Syva microtrak). Negative cultures were continued for 10 days before being discarded. Results of typing were grouped according to date of isolation and the incidence of HSV-1 genital isolates analysed by a chi-square test for trend. Our results, shown in the table, indicate an increasing trend in laboratory reports of HSV-1 from young females (<25 years) and, because of the strength of this trend, in females overall.

Table: Typed positive specimens—proportion identified as HSV-1, 1985–88. (chi square test for trend)

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<tbody>
<tr>
<td>All patients</td>
<td>31%</td>
<td>29%</td>
<td>38%</td>
<td>4·63</td>
<td>&gt;0·1</td>
</tr>
<tr>
<td>Females only</td>
<td>41%</td>
<td>47%</td>
<td>54%</td>
<td>4·63</td>
<td>&lt;0·05</td>
</tr>
<tr>
<td>Males only</td>
<td>21%</td>
<td>21%</td>
<td>17%</td>
<td>1·1</td>
<td>&gt;0·25</td>
</tr>
<tr>
<td>Females &lt;25</td>
<td>45%</td>
<td>59%</td>
<td>65%</td>
<td>6·46</td>
<td>&lt;0·025</td>
</tr>
<tr>
<td>Females &gt;125</td>
<td>33%</td>
<td>31%</td>
<td>34%</td>
<td>0·02</td>
<td>&gt;0·5</td>
</tr>
<tr>
<td>Males &lt;25</td>
<td>33%</td>
<td>26%</td>
<td>19%</td>
<td>1·85</td>
<td>&gt;0·1</td>
</tr>
<tr>
<td>Males &gt;25</td>
<td>13%</td>
<td>6%</td>
<td>16%</td>
<td>0·16</td>
<td>&gt;0·5</td>
</tr>
</tbody>
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*Non-compliant patients.

There are obvious limitations in interpretation of retrospective studies, but, nevertheless, HSV-1 is, in Glasgow, effectively replacing HSV-2 as the main cause of genital herpes in young females. The higher frequency of HSV-1 in younger patients can be explained by delayed primary exposure to the virus observed in socially privileged groups in Western countries,1 primary attacks manifesting at a genital site due to sexual activity. The sex bias shown by HSV-1 is, however, unexplained, and although it has been noted by other workers,5·6 no satisfactory explanation has, to our knowledge, been offered. Sexual habits vary greatly, so it seems unlikely that this consistent disparity could be due to the more frequent practice of cunnilingus relative to fellatio. The risk of autoinfection may be higher in females, for anatomical reasons, but this is not a commonly reported complication of symptomatic primary herpes infection, and, again, seems an unlikely explanation.

Whatever the causes of such observations, it is important to monitor the incidence of HSV-1 genital infection; it has a 14–55% recurrence risk, in contrast to a risk of 60–80% for HSV-2; furthermore, any recurrences are fewer, symptomatically less severe and usually temporary.1·5 If HSV-1 is now the main cause of genital herpes in young women, this has important prognostic implications for counselling of patients with initial episodes of genital herpes.

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Penile condylomata acuminata in a male child: a case report

Sir,

Condylomata acuminata is a disease of sexual maturity, though there have been many reports of the disorder occurring in children.1 However, penile warts have not been described in boys.2 Most of the warts in the prepubertal children occur either in the perianal area or the vulvar region, are more common in girls than in boys, and are thought to be due to sexual abuse.

Recently, a six year old boy was seen in the STD clinic with an asymptomatic warty papule on the penis.
present for the past 25 days. No history of sexual assault was forthcoming. There were no genital or extragenital warts either in the parents or any of the close family members seen presently or in the past. Local examination revealed a single, asymptomatic, pedunculated, pink coloured, warty papule, 5 mm in size, situated at the coronal sulcus at 7 o’clock position (fig). There was no regional lymphadenopathy. No other wart was seen on the body. Histology confirmed the diagnosis of condylomata acuminata. Follow up four weeks after the excision biopsy did not show any recurrence.

Genital warts are not uncommon in children. The reported age of onset varies from one day to 13 years. Our patient had exclusive involvement of the genitalia without any evidence of anal involvement or extragenital lesions. The exact mode of contracting the infection is not known. Probably, it was a non-sexual mode, a close intimate contact in the past with a person having warts elsewhere. Although genital and non-genital strains of HPV are different, in some cases the wart virus present on the non-genital sites is responsible for the genital lesion. In most of the reported cases, sexual assault was suspected despite a clear cut history, but there are views that non-sexual transmission is possible. We are reporting this case because of the rarity of presence of warts on the penis of a child, which certainly could not have been used for sexual purposes. To the best of our knowledge, such a case has not been reported previously in the literature.

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Penicillin concentrations in CSF during repository treatment for syphilis

Van der Valk et al detected treponemal levels of penicillin in cerebrospinal fluid (CSF) after the first daily injection in only 23 of 40 patients treated with daily injections of 2.4 MIU procaine penicillin IM and probenecid 500 mg 6 hourly by mouth. The findings indicate the likelihood that accumulation of penicillin in CSF would occur, to produce higher levels following the second injection of the daily treatment. Thus at 24 hours after the first injection three of ten patients had treponemal levels in CSF. In these patients with treponemal levels (and in those with subtreponemal levels), the second injection would produce a concentration of penicillin superimposed upon the existing level from the first injection.

It was for this reason that each penicillin level in our assessment of the regimen for outpatients was measured after the second to the ninth daily IM injection of procaine penicillin with probenecid by mouth. All our 50 patients had treponemal levels in the CSF: 38 after 2.4 MIU daily and 12 after only 1.8 MIU daily: all 50 received probenecid 500 mg 6 hourly by mouth. The lowest concentration of penicillin achieved in CSF was 0.06 mg/l in one of the 12 patients receiving 1.8 MIU procaine penicillin daily, a level providing an appreciable margin. There was no evidence of summation of levels following the second to the ninth injection.

Another difference is that all our patients were ambulant so that the results could be applied to outpatients. Van der Valk et al tested their patients after complete bed rest following the first injection and considered that they had followed "the same regimen" as we had although the differences are material.

It is of interest that van der Valk et al found a serum penicillin range of only 0.3-25 mg/l compared with our 1.5-30.4 in the 38 patients who had received 2.4 MIU of procaine penicillin G IM daily with probenecid by mouth. The average was 9.1 mg/l in serum and 0.28 mg/l in CSF in nine patients over 80 kg in weight, with a CSF concentration of 3.1% of serum concentration.

In 8 patients who weighed less than 60 kg the corresponding levels were appreciably higher at an average 13.8 mg/l, 0.5 mg/l and 5.5%. So weight may also be a factor in the lower levels found by van der Valk et al. We previously had treated 31 patients with an in-patient regimen of 500,000 IU benzyl penicillin G im 6 hourly and probenecid 500 mg 6 hourly by mouth. All were shown to have achieved treponemal levels in CSF with this intramuscular regimen.

The work of Fishman is sometimes quoted against the use of probenecid with penicillin for the treatment of patients with neurosyphilis. But he was considering the use of probenecid in the treatment of “penicillin-sensitive bacterial infections” for which high levels of penicillin are indicated at all infected sites; provided