LETTERS TO THE EDITOR

Changing trends in genital herpes due to *Herpes simplex* virus type 1 in Glasgow, 1985-88.

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 herpes 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.

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,⁶ 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,^{1,2,3,7} 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 counsell-

ing of patients with initial episodes of genital herpes.

ANNE SCOULAR*
BARBARA GS LEASK†
DAVID CARRINGTON†
Departments of
Genito-urinary Medicine* and
Virology,†
Glasgow Royal Infirmary,
86, Castle Street,
Glasgow, G4 0SF,
Scotland, UK

- 1 Corey L, Adams HG, Brown ZA, Holmes KK. Genital herpes simplex virus infections: clinical manifestations, course and complications. *Ann Intern Med* 1983;98:958-72.
- 2 Peutherer JF, Smith IW, Robertson DHH. Genital infection with herpes simplex virus type 1. *J Infection* 1982;2:33-5.
- 3 Lavery HA, Connolly JH, Russell JD. Incidence of genital herpes in Northern Ireland in 1973-83 and herpes simplex types 1 and 2 isolated in 1982-84. *Genitourin Med* 1986;62:24-7.
- 4 Barton IG, Kinghorn GR, Najem S, Al-Omar LS, Potter CW. Incidence of herpes simplex virus types 1 and 2 isolated in patients with herpes genitalis in Sheffield. *Br J Venereal Dis* 1982;58:44-7.
- 5 Reeves WC, Corey L, Adams HG, Vonter LA, Holmes KK. Risk of recurrence after first episodes of genital herpes. Relation to HSV type and antibody response. *N Engl J Med* 1981;305:315-9.
- 6 Wolontis S, Roizman B. Infection with herpes simplex viruses 1 and 2. *N Engl J Med* 1973;289:781-9.
- 7 Tardieu JC, Friedel J. Incidence of herpes simplex viruses types 1 and 2 in herpes genitalis in Strasbourg, France. (Letter). *Br J Venereal Dis* 1983;59:138.

Accepted for publication 30 January 1990.

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, 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

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

	July 1985– Sept 1986	Oct 1986– Sept 1987	Oct 1987– Dec 1988	χ^2	<i>p</i>
All patients	31%	29%	38%	2.03	>0.1
Females only	41%	47%	54%	4.63	<0.05
Males only	21%	21%	17%	1.1	>0.25
Females ≤ 25	45%	59%	65%	6.46	<0.025
Females > 25	33%	31%	34%	0.02	>0.5
Males ≤ 25	33%	26%	19%	1.85	>0.1
Males > 25	13%	6%	16%	0.16	>0.5

*Non-compliant patients.

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.³ The reported age of onset varies from one day to 13 years.² 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.⁴ In most of the reported cases, sexual assault was suspected despite a clear cut history,^{5,6} but there are

views that non-sexual transmission is possible.¹

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.

BHUSHAN KUMAR
RAJIV GUPTA
S C SHARMA

Department of Dermatology,
Postgraduate Institute of Medical Education
and Research, Sector—12, Chandigarh—
160012, India.

- Oriel JD. Anogenital papillomavirus infection in children. *Br Med J* 1988;296:1484-5.
- Oriel JD. Genital papillomavirus infections: Clinical manifestations. *Recent Advances in Sexually Transmitted Diseases*. 3rd ed. London: Churchill Livingstone, 1986:127-45.
- Stumpf PG. Increasing occurrence of condylomata acuminata in premenarchal children. *Obstet Gynecol* 1980; 56:262-4.
- Chuang Tsu-Yi. Condylomata acuminata (Genital Warts). An epidemiologic view. *J Am Acad Dermatol* 1987;16: 376-84.
- Schachner L, Hankin DE. Assessing child abuse in childhood condylomata acuminata. *J Am Acad Dermatol* 1985; 72:157-60.
- Shelton TB, Jerkins GR, Norman H. Condylomata Acuminata in the Pediatric patient. *J Urol* 1986;135: 548-9.

Accepted 23 January 1990

Penicillin concentrations in CSF during repository treatment for syphilis

Van der Valk *et al*¹ detected treponemical 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 treponemical levels in CSF. In these patients with treponemical levels (and in those with sub-treponemical 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 outpatients² was measured after the second to the ninth daily IM injection of procaine penicillin with probenecid by mouth. All our 50 patients had treponemical 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*¹ tested their patients after complete bed rest following the first injection and considered that they had followed "the same regimen" as we had² although the differences are material.

It is of interest that van der Valk *et al*¹ 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*.²

Previously we 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.³ All were shown to have achieved treponemical levels in CSF with this intramuscular regimen.

The work of Fishman⁴ 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

Figure Penile wart in a 6 year old child.