Pneumococcal vaccine and HIV infection

Helberg and colleagues1 state "An association between cervical dyskaryosis, as well as the role of HPV in cervical cancer in situ and in invasive cancer, has been demonstrated." They quote Franceschi and colleagues1 in support of this claim. Sheppard and colleagues2 report the psychological distress of patients diagnosed with genital warts for whom "...there is the fear of the link between genital warts and cervical cancer".

The paper which is frequently quoted as establishing a link between genital warts and cervical cancer was a case-control study performed by Franceschi and colleagues3 did no such thing. These authors studied women attending a gynaecological medicine clinic, who had smears taken.

Among the women attending with genital warts there was a significant excess of smear showing "superficial dyskaryosis". None of these women had evidence of high-grade CIN and certainly none of them had cervical cancer. All of the more severe cytological abnormalities occurred in women with trichomomas and gonorrhoea.

Having performed a second preliminary study, two of the authors returned to Italy where they conducted a more rigorous study4, which demonstrated no evidence of an association between genital warts and cervical carcinoma, in situ or invasive cervical cancer. Ever since discovering the second negative paper it has always amazed me how widely quoted is the first paper by these authors, whilst the second is almost universally ignored. Is it because the first paper was in a British journal and the second one in an American journal? Did the first paper have a "snappier title" or was it because the first paper confirmed people's prejudices and the second didn't? The original group of an association was further refuted by our own work.5

Could it be that the myth of genital warts needs the same treatment as the other myth about cervical cancer—that "it has been around for 150 years now so it must be true in virgins"—finally debunked in 1992?6

MALCOLM GRIFFITHS
Department of Obstetrics and Gynaecology Luton and Dunstable Hospital NHS Trust Luton Road, Luton, LU4 0DE

Carcinoma of the penis: A cluster of cases in young men

The authors of the recent article Carcinoma of the penis in a HIV positive patient1 emphasise that this malignancy is rare in the immunocompetent population, especially among young men. Indeed, in 1989 (the most recent year for which figures are available2) there were only 45 notified cases in men under the age of 50 years in England and Wales.

It may therefore be of interest to report that recently, in the space of seven months, no fewer than four apparently immunocompetent men presented to this department with ulcerating lesions, with varying degrees of suspicion of malignancy. The men's ages ranged from 34 to 48 years. Although none had a HIV test, they were all heterosexual with no high risk factors for HIV infection. Two of the four had clinical appearances suggestive of lichen sclerosus, a third had a history of genital warts and all were uncircumcised.

A case cluster of possible tissue invasive gonorrhoea

I read with great interest the report by Brook et al of a case cluster of five cases of invasive gonococcal infection.1 The authors appear to be unaware of a similar report published over twenty years ago.2 We described a cluster in which a male patient with gonorrhoea infected seven of eight female contacts. Two other female partners could not be located. Among the seven infected women, two had disseminated gonococcal infection, four had pelvic inflammatory disease, and one had a Bartholin gland abscess. Three weeks after successful treatment of his urethritis, the male index case returned with disseminated gonococcal infection, having resumed intercourse with some of the same partners prior to their diagnosis and treatment.

In 1973 we lacked the ability to definitively prove that all of our patients were infected with the same strain of Neisseria gonorrhoeae. However, the epidemiologic circumstances made it clear that most or all of the patients in fact shared a common strain. We also cited several other reports from 1940 to 1972 that documented complications of gonococcal disease in couples or in mother-infant pairs.3 Collectively, these reports provided the first hint of variations in pathogenicity among gonococci. There is nothing new under the sun (to coin a phrase)!

H. HUNTER HANDSFIELD
Hartford Medical Center 325 Ninth Avenue, Room 359/799 St. Louis, Washington 91804/2499 U.S.A.

Pseudomonas aeruginosa infections and HIV

Ali, et al1 provide an interesting overview of their experience over a five year period with pseudomonas infections in HIV seropositive patients. Their report of an increase in the frequency of both pneumonic and septicaemic disease due to this organism concurs with other recent studies. Two points arise however, which merit further discussion. A report from this centre is incorrectly referenced1 as illustrating that pneumonias due to Staphylococcus aureus and non-locally acquired gram-negative organisms occur with increased frequency in patients with indwelling central venous catheters (CVCs). In fact, what the quoted study demonstrated was an increased frequency of Pseudomonas aeruginosa as an isolate in the blood cultures of HIV seropositive patients with septicaemia (found in 19 of 52), especially those with indwelling CVCs; in only two of these patients was infection due to Pseudomonas aeruginosa established.

It is important to re-emphasise the conclusion that the use of systemic pyreno-cys-tis prophylaxis is an independent risk factor for the development of Pseudomomas aeruginosa pneumonia is erroneous and is not supported by the data presented. As the authors note, the affected patient group were all in the advanced stages of HIV disease with low CD4 counts. Not surprisingly therefore, the vast majority were also on Pneumocystis carinii prophylaxis. However, without showing an increased risk for this group over a similarly severely immunosuppressed matched group not taking PCP prophylaxis (which for obvious reasons would be difficult to gather), this conclusion cannot be drawn. The low CD4 count, on the other hand, may be the relevant variable.

DAVID MOORE
MARK NELSON
Kohler Centre, St Stephen's Clinic, Cheltenham and Gloucestershire Trust 369 Fulham Road, London SW10 9TH, UK


2 Mertens TE, Hayes RJ, Smith PG. Epidemiological methods to study the interaction between HIV infection and other sexually transmitted diseases. AIDS 1990; 4:57-65.

2 Mertens TE, Hayes RJ, Smith PG. Epidemiological methods to study the interaction between HIV infection and other sexually transmitted diseases. AIDS 1990; 4:57-65.

A case cluster of possible tissue invasive gonorrhoea

I read with great interest the report by Brook et al of a case cluster of five cases of invasive gonococcal infection.1 The authors appear to be unaware of a similar report published over twenty years ago.2 We described a cluster in which a male patient with gonorrhoea infected seven of eight female contacts. Two other female partners could not be located. Among the seven infected women, two had disseminated gonococcal infection, four had pelvic inflammatory disease, and one had a Bartholin gland abscess. Three weeks after successful treatment of his urethritis, the male index case returned with disseminated gonococcal infection, having resumed intercourse with some of the same partners prior to their diagnosis and treatment.

In 1973 we lacked the ability to definitively prove that all of our patients were infected with the same strain of Neisseria gonorrhoeae. However, the epidemiologic circumstances made it clear that most or all of the patients in fact shared a common strain. We also cited several other reports from 1940 to 1972 that documented complications of gonococcal disease in couples or in mother-infant pairs.3 Collectively, these reports provided the first hint of variations in pathogenicity among gonococci. There is nothing new under the sun (to coin a phrase)!

H. HUNTER HANDSFIELD
Hartford Medical Center 325 Ninth Avenue, Room 359/799 St. Louis, Washington 91804/2499 U.S.A.

Pseudomonas aeruginosa infections and HIV

Ali, et al1 provide an interesting overview of their experience over a five year period with pseudomonas infections in HIV seropositive patients. Their report of an increase in the frequency of both pneumonic and septicaemic disease due to this organism concurs with other recent studies. Two points arise however, which merit further discussion. A report from this centre is incorrectly referenced1 as illustrating that pneumonias due to Staphylococcus aureus and non-locally acquired gram-negative organisms occur with increased frequency in patients with indwelling central venous catheters (CVCs). In fact, what the quoted study demonstrated was an increased frequency of Pseudomonas aeruginosa as an isolate in the blood cultures of HIV seropositive patients with septicaemia (found in 19 of 52), especially those with indwelling CVCs; in only two of these patients was infection due to Pseudomonas aeruginosa established.

It is important to re-emphasise the conclusion that the use of systemic pyreno-cystis prophylaxis is an independent risk factor for the development of Pseudomomas aeruginosa pneumonia is erroneous and is not supported by the data presented. As the authors note, the affected patient group were all in the advanced stages of HIV disease with low CD4 counts. Not surprisingly therefore, the vast majority were also on Pneumocystis carinii prophylaxis. However, without showing an increased risk for this group over a similarly severely immunosuppressed matched group not taking PCP prophylaxis (which for obvious reasons would be difficult to gather), this conclusion cannot be drawn. The low CD4 count, on the other hand, may be the relevant variable.

DAVID MOORE
MARK NELSON
Kohler Centre, St Stephen's Clinic, Cheltenham and Gloucestershire Trust 369 Fulham Road, London SW10 9TH, UK

A case cluster of possible tissue invasive gonorrhoea.

H H Handsfield

doi: 10.1136/sti.71.5.336

Updated information and services can be found at: [http://sti.bmj.com/content/71/5/336.1.citation](http://sti.bmj.com/content/71/5/336.1.citation)

**Email alerting service**

Receive free email alerts when new articles cite this article. Sign up in the box at the top right corner of the online article.

Notes

To request permissions go to: [http://group.bmj.com/group/rights-licensing/permissions](http://group.bmj.com/group/rights-licensing/permissions)

To order reprints go to: [http://journals.bmj.com/cgi/reprintform](http://journals.bmj.com/cgi/reprintform)

To subscribe to BMJ go to: [http://group.bmj.com/subscribe/](http://group.bmj.com/subscribe/)