MATTERS ARISING

The diagnosis of prostatitis

Dr Thin's review of prostatitis¹ throws much light on an ill-understood condition. Of particular practical importance is his clarification of the way in which reflux of urine into the prostate occurs, and the part that this may play in the infective process.

However, he states that acute bacterial prostatitis is uncommon. Many years' experience of providing a laboratory diagnostic service to general practitioners in a large health district has convinced me that this condition is far commoner than may be apparent to urologists and other doctors who work in hospital. Specimens of urine with request forms describing the clinical syndrome characteristic of this condition, as described by Dr Thin, are received in the laboratory every week from up to 30 men of all ages. They invariably show gross pyuria and yield common bacterial urinary pathogens, often, but not always, in high counts. High bacterial counts are, of course, the consequence of multiplication of bacteria in the bladder, which acts as an incubator. Men with acute prostatitis do not necessarily also have bladder infection, and in these patients bacterial counts may be low. We have shown that men without symptoms referable to the urinary tract or prostate do not excrete Gram-negative organisms in the urine,² suggesting that the presence of such organisms, in whatever count, should be taken as indicative of infection.

Apart from those with the fever and malaise characteristic of acute prostatitis, urine specimens are also received from a large number of men with dysuria or frequency, many of which show heavy pyuria.³ Some may have chronic prostatitis caused by the common bacterial urinary pathogens; others may have infection with one of the less common pathogens listed by Dr Thin. To this list should be added *Gardnerella vaginalis*,⁴ *Haemophilus influenzae*,⁵ almost certainly *Chlamydia trachomatis*⁶ and possibly *Corynebacterium spp.*⁷ Many of these pathogens are only detected if appropriate culture techniques are used.⁸ Our laboratory procedure is to request a further specimen from all men in whom pyuria is unexplained by overnight culture on Cled agar. This specimen is then cultured on Cled agar and chocolate blood agar for incubation in an atmosphere containing 5% CO₂ for 48 hours, and on nalidixic acid blood agar for incubation anaerobically for 48 hours. If pyuria is still present and all cultures are negative the infection is presumed to be due to *C. trachomatis*, mycoplasma or ureaplasmas. As yet, we do not apply techniques for detection of *C. trachomatis* in urine, but such techniques have been applied successfully elsewhere.^{9,10}

On the assumption that the very unpleasant condition of chronic prostatitis occurs as a consequence of inadequately diagnosed or treated acute prostatitis, our laboratory reports on urine specimens from men carry a suggestion that the patient should be treated,

in accordance with the sensitivity of the isolate, for 14 days with an agent that achieves therapeutic concentration in the prostate. Such agents include cotrimoxazole, doxycycline and ciprofloxacin; erythromycin may also be used for infections with Gram positive organisms. Treatment with agents such as nitrofurantoin, the penicillins or cephalosporins will merely sterilise the urine temporarily but leave the prostatic focus of infection untouched.

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- 1 Thin RN. The diagnosis of prostatitis: a review. *Genito Urin Med* 1991;67:279-83.
- 2 Sugarman I, Pead LJ, Payne SR, Maskell RM. Bacteriological state of the urine in symptom-free adult males. *Br J Urol* 1990;66:148-51.
- 3 Clarke M, Pead L, Maskell R. Urinary infection in adult men: a laboratory perspective. *Br J Urol* 1985;57:222-6.
- 4 Abercrombie GF, Allen J, Maskell R. *Corynebacterium vaginale* urinary tract infection in a young man. *Lancet* 1978;i:766.
- 5 Gabre-Kidan T, Lipsky BA, Plorde JJ. *Haemophilus influenzae* as a cause of urinary tract infections in men. *Arch Intern Med* 1984;144:1623-7.
- 6 Bruce AW, Reid G. Prostatitis associated with *Chlamydia trachomatis* in 6 patients. *J Urol* 1989;142:1006-7.
- 7 Maskell R, Pead L. *Corynebacteria* as urinary pathogens. *J Infect Dis* 1990;162:782-3.
- 8 Maskell R. *Urinary Tract Infection in Clinical and Laboratory Practice*. London. Edward Arnold, 1988. Appendix 2. 246-54.
- 9 Caul EO, Paul ID, Milne JD, Crowley T. Non-invasive sampling method for detecting *Chlamydia trachomatis*. *Lancet* 1988;ii:1246-7.
- 10 Matthews RS, Bonigal SD, Wise R. Sterile pyuria and *Chlamydia trachomatis* *Lancet* 1990;336:385.

Declining trends in some STDs in Belgium

Dr Walckiers and her colleagues report interesting trends in some sexually transmitted diseases in Belgium based on data derived from sentinel networks of general practitioners and laboratories,¹ and acknowledge some of the shortcomings of their approach. From the laboratory data it is very difficult to infer the frequency with which infections occur in the population. This is because neither the proportion of infected individuals who present to health facilities, nor the frequency with which those health facilities carry out testing in such individuals are known. For sentinel networks of general practitioners, diagnosis is largely syndromic and information about the occurrence of individual pathogens cannot be derived.

Might I suggest that where both clinical and laboratory sentinel surveillance systems are operating in parallel, the former might, where practical, collect information about practitioners' testing practices both for screening, and diagnosis. Such information would enable the number of infected patients seen by clinicians to be estimated from the number of positive laboratory tests reported. Although such information would tell us nothing about the proportion of all infected individuals who present, it seems reasonable to assume that this proportion is less likely to be subject to rapid variation over time than

testing practices; at least for diseases such as chlamydial and gonococcal urethritis in men. If in addition clinical information could be provided to laboratories concerning whether a test was carried out for screening or diagnostic purposes, then the frequency of occurrence of infections in asymptomatic individuals could also be estimated.

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- 1 Walckiers D, Piot P, Stroobant A, Van Der Veken J, Declercq E. Declining trends in some sexually transmitted diseases in Belgium between 1983 and 1989. *Genitourin Med* 1991;67:374-7.

Choosing equipment for treating genital warts

We read with interest the excellent paper by Anne Scoular which included an update of surgical techniques available for the treatment of recalcitrant warts.¹

We wish to comment that curettage as a treatment modality was not mentioned in this paper. Curettage is very cheap and efficiently removes isolated hyperkeratotic warts which resist chemotherapy.² The equipment consists of a Volkman's spoon, a curette which can be reused after sterilisation. Ethyl chloride spray is used as a local anaesthetic prior to the procedure and bleeding can be easily controlled using silver nitrate sticks or Monsel's solution. It is our experience that patient acceptability equates with that of other destructive methods of wart treatment and healing occurs without scarring.

Care, however is required in order to avoid the potential hazards of ethyl chloride in the work place as it is a potent anaesthetic.³ Occupational safety limits are set at 1000 parts per million for long term use and 1250 parts per million for short term use.^{4,5} Furthermore, as it is a highly flammable substance, the necessary precautions must be taken, such as ensuring that it is not used near a naked flame, in high temperatures or when sparks are likely (near electrical equipment). It should be stored in a cool, dark place at or under 20°C and adequate ventilation ensured when it is used.⁶

Significant toxicity may occur in medical attendants but only after prolonged exposures as there are two case reports in the literature of psychological and neurological symptoms which occurred after daily exposure to ethyl chloride over several months. Both cases resolved spontaneously on withdrawal of exposure.^{7,8}

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- 1 Scoular A. Choosing equipment for treating genital warts in genito-urinary medicine clinics. *Genitourin Med* 1991;67:413-5.
- 2 Kinghorn GR. Genital papillomavirus infection: treatment. In: Oriel HD, Harris JEW, eds. *Recent Advances in Sexually Transmitted Diseases*. Vol 3, Edinburgh. Churchill Livingstone. 1986:147-55.
- 3 McEvoy GK, Ed. *American Hospital Formulary Services—Drug Information*. Bethesda. American Society of Hospital Pharmacists Inc, 1991.

- 4 Reynolds JEF (Ed) Martindale. *The Extra Pharmacopoeia*, 29th ed. London: Pharmaceutical Press. 1989.
- 5 National Institute of Occupational Safety and Hazards. Recommendations for occupational safety and health standards. 1988. Morbidity and mortality weekly report (suppl) 1988;37:8.
- 6 Ethyl chloride BP. *Production Information*. Berkshire UK. Bengue and Company Ltd 1982.
- 7 Nordin C, Rosengust M, Hollstedt C. Sniffing of ethyl chloride—an uncommon form of abuse with serious mental and neurological symptoms. *Int J Addict* 1988;23:623–7.
- 8 Hes JP, Cohn DF, Streffer M. Ethyl chloride sniffing and cerebellar dysfunction. *Israel Ann Psychiatry* 1979;17:122–5.

Increased incidence of cervical cytological abnormalities in women with genital warts

I was interested to read the study by Rowen *et al*¹ showing a higher rate of smear abnormalities in women with or contacts of genital warts. Their observations agree with my own (Griffiths M, MD thesis, University of London), where I found abnormal smears in 28% of women with warts and only 9% of controls. Both studies effectively repeat the findings of Franceschi and colleagues² who found an excess of abnormal smears (largely of “superficial dyskaryosis”) in women with warts compared with other STD clinic attenders, though a review of their paper demonstrates that high grade abnormalities were more common in controls.

However, we have shown no difference in the risk of cervical epithelial disease between the two groups,³ when judged by colposcopy and histology. We hypothesised that the reason for this apparent discrepancy might, at least in part, be due to more cautious examination and reporting of smears coming from women known to have warts, resulting in a relative over-reporting of (particularly minor) abnormalities by cytologists. This hypothesis was supported by the findings of a pilot study in which smears from women with warts were sent to cytology with clinical details of either “warts” or “routine” according to prior randomisation. The study showed an excess of “abnormal” smears among “warts” patients but this difference just failed to reach statistical significance owing to sample size.

I believe that cytologists are more likely to report abnormal smears if the clinical information given refers to a history of warts, and therefore would be interested to know whether the cytologist in this particular study was blind to clinical information concerning the patients’ history of warts.

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- 1 Rowen D, Carne CA, Sonnex C, Cooper P. Increased incidence of cervical cytological abnormalities in women with genital warts or contact with warts: a need for increased vigilance? *Genitourin Med* 1991;67:460–3.
- 2 Franceschi S, Doll R, la Vecchia C. *et al*. Genital warts and cervical neoplasia: an epidemiological study. *Br J Cancer* 1983;48:621–8.
- 3 Griffiths M, Sanderson D, Penna LK. Cervical epithelial abnormalities among women with vulval warts—no more common than among controls. *Int J Gynaecol Cancer* (In Press).

Rowen *et al* have looked at an important issue regarding the relationship between genital warts and cervical cancer.

There are a number of small points in respect of the data they present which require clarification: the indications for taking a cervical smear are actually not given and it is not clear whether the 185 patients represent the total number smeared over the 5 month period of study. It is really quite important to know who was invited to participate and who declined.

The proportion of abnormal smears was much lower in the non-wart group (7 of 55) than in the wart group (52 of 117). However, the wart group is twice the size of the non-wart group, which may not be representative of women patients as a whole.

Although it is clearly stated that 59 patients had a cervical biopsy, it is less clear how many were colposcoped. Surely some patients with abnormal smears showed no abnormality on colposcopy and therefore did not have a biopsy. If these patients are included in table 3, it is not clear from the legend, but 65 (117–52) patients seem to have gone missing.

While the authors’ conclusions appear valid from the data they present, the relevance of mildly abnormal smears is called into question. Their biopsy results show that cervical intra-epithelial neoplasia (CIN) was present in 30% (13 of 43) of patients with warts, 11% (1 of 9) of patients in contact with warts, but in 43% (3 of 7) of patients without warts or wart contact. From this it could well be concluded that genital warts are not related to CIN.

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Rowen *et al* reply:

The letters from Drs Griffiths, Evans and Kell concerning our recent paper are read with interest and raise some points which merit discussion. In our study the screeners were aware of clinical details. It is of course possible to over report smears. However, we do not feel that significant numbers are over reported as several safeguards are in place to prevent this. Firstly, a relatively junior screener cannot send out an abnormal report without the smear being reviewed by a senior screener. Secondly, a smear deemed to show mild dyskaryosis must be reviewed by a pathologist. Thirdly, follow-up smears from women with borderline abnormalities on previous smears are screened by a senior MLSO. If any abnormality is found on that smear, it and the previous smear are then reviewed by the cytopathologist. Furthermore, if there were significant over reporting one might suspect that the “current smear normal, previous smear abnormal” group in our study would be larger than we found.

Dr Griffiths’ results from his pilot study in which screeners were blinded to the real clinical details are of interest. There may or may not be an excess of smears reported as abnormal in the “warts” group. However his conclusion that the failure to demonstrate a statistically significant difference in rates of smears reported as abnormal in the two groups was simply due to sample size cannot be justified at this stage. If a full scale study, with sufficient numbers in each group subsequently demonstrates a significant difference in rates, then one may draw the conclusion that the pilot study failed to demonstrate significant differences because of sample sizes.

With regard to the points raised by Drs Evans and Kell. Patients attending our clinic are offered cervical cytology if (a) they have not had a smear within the last 3 years or (b) they or their sexual partners have genital warts and they have not had a smear within one year. The 185 women in the study were drawn from 191 women having smears during the study period. No patients declined to answer the life-style questions, but six patients, all from the warts/warts contact group were not offered colposcopy appointments as they were about to leave the area and thus were not included in the study.

All patients in the study with abnormal smears, except two, who defaulted from follow up, were colposcoped, as were all, except 3 from the warts/warts contact group who had normal smears.

Table 3 in the paper should have been headed “Abnormal cytology results compared with colposcopy results” and “Biopsy proven CIN”. Thus the NO CIN column represents those whose biopsies were negative and those who had a normal colposcopy and were therefore not biopsied. We apologise for the confusion this may have caused.

In the small number of women with abnormal smears but no history of warts or wart contact, we would agree that there was a high rate of CIN. They did however differ from other groups by virtue of having significantly more sexual partners and it is possible some may have been infected with HPV without developing warts. What is not known is the natural history of sub-clinical HPV infection and whether such lesions ultimately develop into frank warts or aceto-white lesions and if not, whether these sub-clinical lesions are also associated with abnormal cytology in the absence of warts.

We also agree that we did not find a significant incidence of CIN in the warts/warts contact groups, a point alluded to in the discussion. We did find differences in rates of cytological abnormalities between the warts/warts contact group and the non wart/wart contact group and forward the notion that these abnormalities may be the result of an acute reaction to HPV infection which had settled by the time colposcopy was performed.

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Increased evidence of cervical cytological abnormalities in women with genital warts

We read with great interest Dr Rowen *et al*’s¹ paper examining the need for increased cytological vigilance in women with genital warts or contact with genital warts, and agree that this group should also be offered colposcopic examination of the cervix irrespective of their cervical cytology result. Our results and experience are in agreement with the above conclusion. We present figures from our department on women with genital warts and negative cytology. In the period May 1987 to June 1988, 248 women with genital warts and 12 with wart contacts, attending the genitourinary medicine out-patient clinic, Royal Liverpool University Hospital