***Clostridium difficile* in the genital tract**

Sir,
Recent studies of anaerobic bacteria in both the male and female genital tracts have included techniques for the isolation of *Clostridium difficile*.¹⁻³ Hafiz *et al* isolated *Cl difficile* from 71% of vaginal specimens from patients attending a sexually transmitted disease (STD) clinic and 18% of women attending a family planning clinic, and from all of 42 men with non-specific urethritis (NSU).⁴

The results of more recent studies have been contradictory. *Cl difficile* was isolated from only two out of 79 patients with balanoposthitis, and not at all from 24 men with NSU, 19 men with both NSU and balanoposthitis, or from 28 asymptomatic controls.² Moreover, Moss failed to isolate *Cl difficile* from 20 men and 34 women attending an STD clinic.¹ A vaginal carriage rate of 11% in consecutive female patients attending an STD clinic and 18% in pregnant women was reported by O'Farrell *et al* using a selective broth medium.³ In this laboratory 206 vaginal swabs from 187 women, and urethral swabs from 20 men attending a special clinic were examined for *Cl difficile*. Swabs were broken off into cooked meat broth and incubated at 37°C for five days before subculture on to modified CCFA medium,^{5,6} but *Cl difficile* was not isolated from any specimen.

There exists an apparent dichotomy between the high carriage rates observed in both symptomatic and asymptomatic populations,^{3,4} and the negligible isolation rates encountered in this and other laboratories.^{1,2} This discrepancy might be explained by the use of isolation techniques of differing sensitivities, but the methods of Moss¹ and Masfari *et al*² were essentially similar to those employed by Hafiz *et al*,⁴ and all recent investigations including the present one used enrichment culture and a highly efficient selective medium. The existence of a geographical variation in urogenital carriage of *Cl difficile* remains a possibility and requires further study.

Yours faithfully,

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TO THE EDITOR, *British Journal of Venereal Diseases*

Case reports of urethritis

Sir,

We should like to comment on two articles in the February 1984 issue of the journal. We agree with Drs M N H Chowdhury and S S Pareek (pp 56-7) that recovery of group B streptococci from the patient with urethritis and from his wife, in the absence of other identifiable pathogenic micro-organisms, together with the successful response to penicillin, suggest a causal relation. There are, however, several aspects which are not clear. The details of the history of urethritis are insufficient to know whether it was consistent or intermittent in severity. Is it not feasible that the response to treatment was more apparent than real and coincided with a natural remission? A longer follow up period would have been helpful in making this assessment. Both patients were free from symptoms after three weeks (of being seen initially?), but were they free from signs of disease and organisms too? We are not told. Furthermore, an important aspect is the sensitivity of procedures which rule out the possibility of other micro-organisms being implicated. To know that chlamydiae were being isolated in the laboratory from other patients with urethritis or cervicitis at rates consistent with those recorded by workers elsewhere would put the negative result in this case into perspective. In addition, to know that culturing for gonococci was negative would have been helpful, as would assessment of the anaerobic flora. We see nothing wrong in presenting a case report, but when the intention is to

persuade the reader about the aetiology of disease there is a greater onus to set out the data explicitly so that there is an opportunity for making a balanced judgement.

The second article, by Drs D W Spelman and D Bradford (pp 58-9), on urethritis in a patient with agammaglobulinaemia was of particular interest to us because we have previously described genitourinary disease, including non-gonococcal urethritis, in patients with hypogammaglobulinaemia.¹ In the case described, it is difficult to resolve the question of whether the improvement in the patient's condition could be attributed to intraurethral instillation of immunoglobulin. Irrigation with saline might have afforded the same symptomatic improvement. Resolution of signs of disease based on quantitative analysis of leukocytes in discharge and urine samples would have been more convincing. Furthermore, it seems unlikely that immunoglobulin, which would remain only transiently in the urethra because of urination, could gain access to paraurethral glands and the prostate where putative pathogens are likely to shelter and then emerge. The administration of doxycycline together with intraurethral immunoglobulin after ureaplasmas were eventually found eliminates any possibility of knowing whether the immunoglobulin was responsible for subsequent ureaplasma negative cultures, unless the ureaplasmas were resistant to tetracycline. It is unfortunate that much of the microbiological investigation in this case came late so that it is impossible to know what micro-organism might have been responsible for the start of disease or for its longevity. Our experience, however, shows that ureaplasmas are able to multiply and attain large numbers ($\geq 10^6$) in the urethra of hypogammaglobulinaemic patients, and to cause urethritis. It will be difficult to resolve the question of whether local irrigation with pooled immunoglobulin is effective in treating this condition because this would entail withholding antibiotics from affected patients. We would not recommend such a trial as ureaplasmas may travel to joints and cause septic arthritis.³ Some rational basis for local treatment, however, would be the showing that pooled immunoglobulin contains antibodies to the strain of organism implicated.

Yours faithfully,

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