A case cluster of possible tissue invasive gonorrhoea

I read with great interest the report by Brook et al of a 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 epidemiological circumstances made it clear that most or all of the patients in fact shared a common partner. 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 occurred new under the sun (to coin a phrase)!

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Pseudomonas aeruginosa infections and HIV

Ali, et al 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 pulmonary and septicemic illness 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 concurrently 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 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 this due to pseudomonas pneumonia. In the same study an apparent association with concurrent CMV infection was cautiously suggested, but the results of Ali et al do not support this.

More important however is their conclusion that the use of systemic pseudo-cys-prophylaxis is an independent risk factor for the development of Pseudomonas aeruginosa pneumonia is erroneous and is not supported by the data of the same study. 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.

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Pneumococcal vaccine and HIV infection

Hellberg and colleagues’ 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 colleagues2 in support of this claim. Sheppard and colleagues3 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 cancer4-5 by Franceschi and colleagues did no such thing. These authors studied women attending a genitourinary medicine clinic, who had smears taken.