Correspondence

TO THE EDITOR, Genitourinary Medicine

Intralesional interferon alpha-2b in treating refractory male and anal warts

Sir,

Previous studies have shown that intralesional injection of interferon alpha-2b (Intron A, Schering Corp.) $1 \times 10^6$ IU three times a week for three weeks is effective in reducing the size and numbers of genital warts. These controlled studies have, however, focused on the treatment of one to three selected externally located genital warts.

Refractory warts are often located in the urethra and anal canal. We used the above regimen to treat 10 male patients who had had warts for more than one year. Five had anal warts (four to five warts a patient), and five had genital warts (one to two warts a patient). The patients had previously received conventional treatment, including podophyllin, electrocautery, cryotherapy, and laser surgery. None had antibodies against human immunodeficiency virus. To reduce the systemic adverse reactions to interferon, all patients were treated with 1-3 g acetylsalicylic acid on each day of injection.

After an observation period of 12 weeks, cure was obtained in five patients (three with genital warts and two with anal warts). A 50% or more reduction in the size of the warts was seen in two. Minimal influenza like symptoms were seen initially in five. A reversible decrease in the numbers of leucocytes was seen in all patients, but none became leucopenic. No other biochemical abnormalities were detected.

We conclude that intralesional injection of interferon alpha-2b may be a suitable way of treating refractory warts located in the urethra and the anal canal.

Yours faithfully,
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References


Risk of AIDS after rape

Sir,

We are grateful to Dr Kay for his comments on our paper “Incidence of sexually transmitted diseases in rape victims during 1984” (Genitourin Med 1987;63:62), in which the risk of acquiring human immunodeficiency virus (HIV) was not discussed. Women who have been raped are left anxious and psychologically helpless. They have to go through a grieving process for their particular loss. By adding the fear of infection with HIV to their anxiety, one is encouraging non-resolution of their psychological trauma. In 1984, we felt that it would have been unethical to raise the fear of the acquired immune deficiency syndrome (AIDS) in these women. At that time, there was almost no evidence of AIDS in female partners of infected men in the UK, Europe, or the USA.

When the risk of acquiring AIDS after rape is small, but present, as it is now, researchers will have to consider ethical ways of screening without causing unnecessary anxiety to patients. Concern about acquiring AIDS or HIV infection may be the only reason for a woman’s attendance at a department of genitourinary medicine following rape. In patients who are very distressed at their initial attendance, and for whom it does not seem to be appropriate to discuss HIV, sera may be taken at three monthly intervals and stored for subsequent testing. This protocol would fit in with recommended patient review to exclude the development of other sexually transmitted diseases, such as syphilis and hepatitis B.

HIV is transmitted via blood and semen. Sexual dysfunction occurs during rape. The alleged rapist may ejaculate before introduction or fail to ejaculate, though the victim may be unaware that this has occurred. Risk of HIV transmission will be lessened if sexual dysfunction has occurred. Furthermore, Jones et al found that only three of 36 regular sexual partners of seropositive patients with haemophilia A were themselves seropositive. There was a contributory factor of a blood transfusion in one woman. The length of the relationship, contraceptive usage, and type of coitus practised were not discussed.

It seems reasonable to conclude at the moment, however, that the risk of acquiring HIV after rape remains low, but should be considered.

Yours faithfully,
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References


Is Entamoeba histolytica in homosexual men a pathogen?

Sir,


He stated in his penultimate paragraph that we found “improvement in proctitis after treatment” (for E histolytica). In fact the reverse obtained. Patients who still harboured this protozoan showed a greater improvement in rectal inflammation histologically than the men from whom E histolytica was eradicated. We went on to point out the differences between our test and control groups, compared with McMillan’s test and control groups. We endeavoured to make the two groups as similar as possible with the exception of the test variable (presence of E histolytica), whereas McMillan, we feel incorrectly, was content to have appreciably different histories of diarrhoea in his test and control groups.

Though we sympathise with Dr McMillan and his colleagues and accept that it is difficult to have well matched test and control groups, we feel that their control group was biased in many respects.

Finally, we feel at a loss to know what non-specific proctitis, whatever that means, has to do with whether E histolytica is a pathogen or not. Quite simply E histolytica that has produced disease in the colon causes invasion of the mucosa. This in turn produces acute proctocolitis and serum antibody to E histolytica. Trophozoites of E histolytica are invariably found in mucus or faeces from such patients. None of these changes were found in any of our patients, perhaps with the exception of a few with histologically con-