Fixed drug eruption due to foscarnet

G M Connolly, B G Gazzard, D A Hawkins

Abstract
A case of fixed drug eruption (FDE) secondary to foscarnet is reported. This drug has recently become available on a compassionate use basis for treatment of cytomegalovirus (CMV) infection which may cause significant disease in immunosuppressed patients. Foscarnet provides a useful alternative to the only licensed anti-CMV drug currently available, namely ganciclovir (DHPG), as it has a different toxicity profile. In particular, it does not appear to cause bone marrow suppression which is of importance in AIDS patients as many of them are taking concurrent zidovudine.

Case history
A 36 year old man with AIDS (oesophageal candidiasis diagnosed in December 1987) developed CMV retinitis in January 1989. He was commenced on intravenous foscarnet (Astra Pharmaceuticals) given as a loading dose of 20 mg/kg, followed by a continuous infusion of 200 mg/kg/24 hours. Five days later he developed four painful ulcers on his glans penis, which were oval and erythematous with dusky centres (see fig). Swabs were taken for Herpes simplex virus (HSV) antigen detection by ELISA, HSV culture and sensitivities. In addition a specimen from the ulcer was examined under dark ground illumination for Treponema pallidium and routine bacteriological cultures were performed. All of these tests were subsequently reported negative. There was no change in his long standing non-tender inguinal lymphadenopathy. The patient was then given acyclovir 400 mg five times a day for possible HSV infection, although we thought this would have been prevented by his foscarnet therapy.  

The ulceration did not improve. However, after two weeks the foscarnet was discontinued as his retinitis was considered stable and within 10 days the ulcers on his penis completely healed. One month later his visual symptoms recurred and after an ophthalmological opinion confirmed reactivation of his CMV retinitis he was recommenced on foscarnet. Five days later the ulcers reappeared on his glans penis. Foscarnet was discontinued and 10 days later the ulcers had completely healed. He was given ganciclovir (DHPG) for his CMV retinitis without recurrence of the penile ulceration. At the time of the recurrence of the ulceration the above microbiological tests were repeated and again reported negative. Syphilis serology also remained negative.

Discussion
Fixed eruption has been reported after many drugs, most commonly after phenolphthalein, barbiturates, sulphonamides, dapsone, quinine and its derivatives, tetracycline and oxyphenbutazone. Some reports suggest that there are specific histological changes diagnostic of fixed drug eruption. We did not feel it was ethical to biopsy this man's ulcer especially as we did not need to continue foscarnet. The appearance and clinical course of the
lesions made it unlikely that other causes of genital ulceration were responsible. In particular infectious causes such as donovanosis, amoebiasis or chancroid were unlikely to have healed spontaneously in this way on two separate occasions and also the patient had not had any sexual partners for longer than one year. In addition, the lesions were typical of fixed drug eruptions (multiple, oval, erythematous with dusky centres) and the glans penis is a favoured site. The time course to healing after cessation of foscarnet was a typical 10 days on each occasion.

The most effective way to make a diagnosis of fixed drug eruption is of course to re-challenge the patient with the drug in question. Our patient was inadvertently re-challenged and we feel this confirms our diagnosis. To our knowledge this is the first report of a foscarnet induced FDE.

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