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Treatment of Facial Verrucae with Topical Imiquimod Cream in a Patient with Human Immunodeficiency Virus

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Imiquimod is a recently developed imidazoquinolin heterocyclic amine that is an immune response modifier. Treatment with topical 5% imiquimod cream has shown promising results in the treatment of genital warts in immunocompetent individuals. We report here the first case of successful treatment with topical 5% imiquimod cream of facial verrucae in an individual with human immunodeficiency virus. ***Key words: verrucae; HIV; antiviral.***

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Imiquimod, a non-nucleoside heterocyclic amine, is a member of a new class of immune-response modifying agents (1). Topical use of imiquimod as an immune system modifier in the treatment of genital and perianal warts in immunocompetent patients has been described recently (2, 3). However, the effectiveness of this agent in the treatment of warts located on other areas of the skin is unknown. Furthermore, it is also not clear what effect, if any, imiquimod would have in patients whose immune system is impaired. We describe here the first reported response of facial warts to treatment with topical 5% imiquimod cream in a patient with human immunodeficiency virus (HIV).

CASE REPORT

A 37-year-old Hispanic male with HIV (documented by ELISA and Western blot 3 years previously) presented with a 3-month history of multiple papular lesions on his face which had not responded to topical antibiotics prescribed by his primary physician. On physical examination, there were several verrucous papules on his face, most located on the cheeks and chin area, consistent with verruca vulgaris (Fig. 1). The largest and most hyperkeratotic lesions were treated with liquid nitrogen cryotherapy, but given the number of lesions and the risk of post-inflammatory hypopigmentary changes on the face, the patient was started on topical imiquimod 5% cream.

He initially started application of the cream twice a week to the entire face, and as that was tolerated without side-effects, he increased the frequency to 3 times a week. When he returned for follow-up after 2 weeks, he noted that many of the lesions had resolved, and many more had decreased in size (Fig. 2). He noted no side-effects from the medication, so he was instructed to apply the imiquimod daily to the face.

When he returned 2 weeks later, he noted increased erythema and slight ulceration of some of his perioral verrucae, and treatment was discontinued. No recurrence of his verrucae was noted on his follow-up 2 weeks after stopping treatment. Although this patient was on "triple therapy" antivirals (DDI, D4T, saquinivir) as well as trimethoprim-sulfamethoxazole, he had been on these medications for months prior to starting imiquimod and no medication changes were made during treatment. A CD4 count after treatment was 167 cells/mm^3, which was lower than his previous CD4 count of 210 cells/mm^3 a few months prior to treatment.

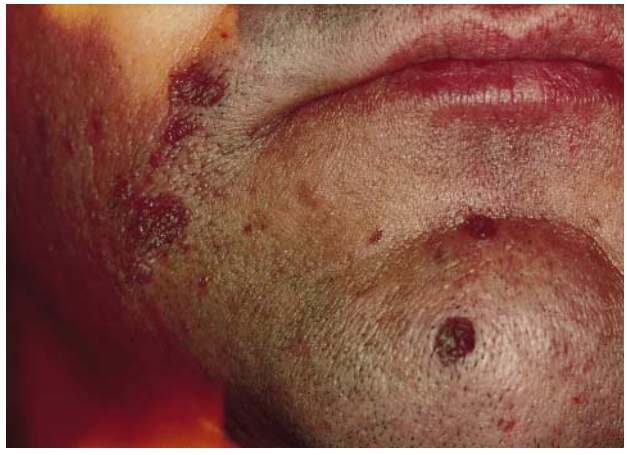

Fig. 1. Facial verrucae at presentation.

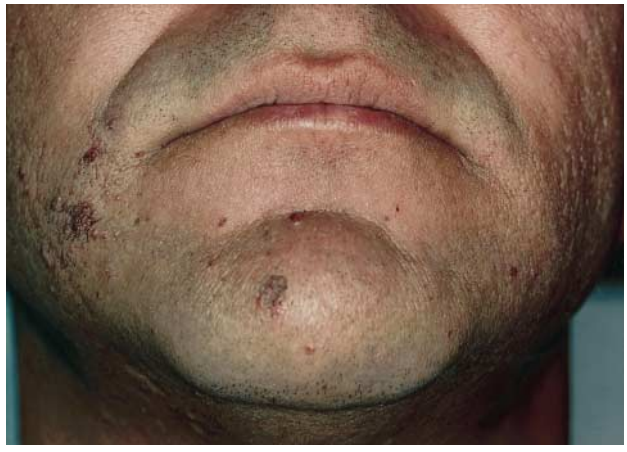

Fig. 2. Frontal view of lesions following 2 weeks of treatment with imiquimod.

DISCUSSION

Imiquimod is an imidazoquinolinamine, which has been used with success in treating both genital herpetic infections and certain tumours in mouse models, as well as treating external anogenital warts in human studies (1–3). Its mechanism, however, has not been completely defined. It has no direct antiviral properties, but has been shown to increase levels of interferon-alpha and other cytokines including tumour necrosis factor, thus upregulating natural immune defences (1). These properties make imiquimod an interesting agent in

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the treatment of verrucae in immunosuppressed patients, such as those with HIV, as it is known that this subset of patients tend to have verrucae which are more extensive and more resistant to current therapies most likely due to deficiencies in their immune system.

The treatment of warts in general involves physically destructive methods, and this often becomes a problem in the treatment of facial warts as patients are concerned about the cosmetic effects of the therapy used. This is even more concerning in darkly pigmented individuals, as post inflammatory hyper- and hypo-pigmentation can be a problem. A non-cytotoxic agent like imiquimod may therefore have a role in the treatment of facial warts, but its efficacy is unknown as studies in humans to date have only involved treatment of anogenital warts (2–4). There is no reason to assume, however, that treatment should be limited only to the genital area, as the mechanism of HPV infection does not vary by body site (although HPV type may) and neither should the immune response. The use of imiquimod has also been expanded to other viral infections of the skin. Syed et al. (5) reported recently on the treatment of molluscum contagiosum in males with an analogue of imiquimod 1% in cream. After 4 weeks of treatment, imiquimod cream cured 82% of the patients and 86.3% of the molluscum lesions. The only side-effects were mild, local reactions.

In our patient, both twice a week and 3 times a week dosing was well tolerated without any significant side-effects, and therapy resulted in clinical regression of his facial warts. We limited our treatment to the smaller verrucae on the face; in the more hyperkeratotic verrucae, it is less likely that the imiquimod cream would be able to penetrate the stratum corneum. We thus relied on cryotherapy to treat the larger verrucae on the face. The drop in CD4 count after treatment makes it less likely that our patient's immune response was due to agents other than imiquimod.

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