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Extra-Oral and Non-Genital Chronic Herpes Infection in an HIV-Infected Patient: A Case Report

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ABSTRACT

Objective: To report a case of uncommon site of chronic herpes infection in an HIV-infected male.

Case presentation: A 19-year-old Thai HIV-infected male who was diagnosed with chronic herpes simplex infection located on the scalp and face without lesion in oral and genital areas.

Conclusion: Chronic herpes simplex infection is predominantly located in oro-genital regions and presented as ulcerative lesions that frequently recur in immunocompromised patients. The prevalence is not known and HIV infection is a major risk factor. Misdiagnosis is common and might lead to delay of treatment. Oral acyclovir is the first line of treatment, although resistance to acyclovir has been found to be increasing.

Keywords: Chronic herpes infection; HIV; non-genital lesion (Siriraj Med J 2018;70: 449-451)

INTRODUCTION

Chronic herpes simplex infection (CHSV) is one of the atypical manifestations of herpes infection among immunocompromised host. CHSV manifests as wart-like and/or ulcerative mucocutaneous lesions lasting for at least 1 month.[1,2] The prevalence of CHSV is still unknown. In a cohort study, 0.2 percent of HIV-infected patients were reported to have this condition, and interestingly all lesions were located in genital area.[3] Misdiagnosis is common and might lead to delay of appropriate treatment.[4] To date, there have been a few cases of CHSV located outside oral and genital area.[4] We reported an HIV-infected patient diagnosed as CHSV whose lesions were located on the scalp and face without oral or genital lesions.

CASE REPORT

A 19-year-old heterosexual male was diagnosed HIV-positivite in 2014 which was controlled with triple anti-retroviral therapy (zidovudine, tenofovir and lopinavir/ritonavir). In July 2016, he presented to our skin clinic with two large well-demarcated ulcerated plaques that showed peripheral crusting on the scalp and right cheek. The lesions had developed over a period of one year (Fig 1). On this visit, his CD4 count and viral load respectively were 144 cells per microlitre and 1.8 million copies per ml.

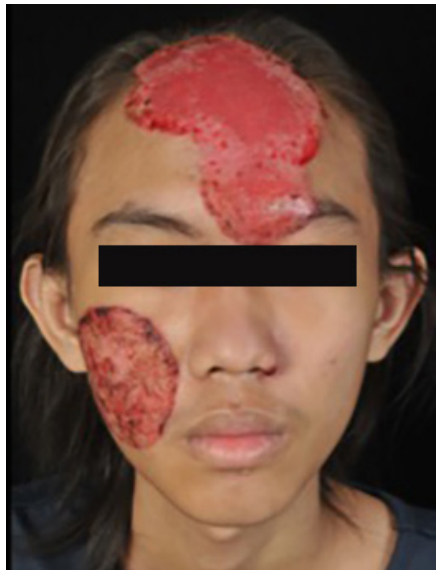

Fig 1. Two large well-demarcated ulcerated plaques with peripheral crusting on the scalp and right cheek.

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He had a history of complete course of pulmonary tuberculosis treatment in 2015 and also had herpes zoster infection on his right thigh 4 months ago that was successfully treated with oral acyclovir 800 mg five times a day for one week. Surprisingly, no history of herpes simplex had been reported before this visit.

On the date of visit, Tzanck smear at both lesions on his face showed negative finding for acantholytic cell or multinucleated giant cell. Direct fluorescent for HSV antigen detection assay from lesions on the scalp, forehead and cheek was positive, but negative for VZV. Blood for HSV and VZV IgG was positive, while blood for HSV and VZV IgM was negative. Tissue culture at scalp was positive for *Staphylococcus aureus* that was susceptible to methicillin and no growth for mycobacterium. Finally, this patient was diagnosed with CHSV with superficial bacterial skin infection. Treatment of oral acyclovir 400 mg per oral three times a day, dicloxacillin 2000 mg per day and topical antibiotics were prescribed. During 3 weeks of treatment, all lesions showed no improvement, but expansion of lesions was observed (Fig 2). Therefore, oral acyclovir 800 mg 5 times a day was given to the patient. After 2 months of treatment, the lesions gradually improved. All lesions were cleared with depressed scar and scarring alopecia was observed on scalp area. There was no new lesion developed after two months of follow up (Fig 3).

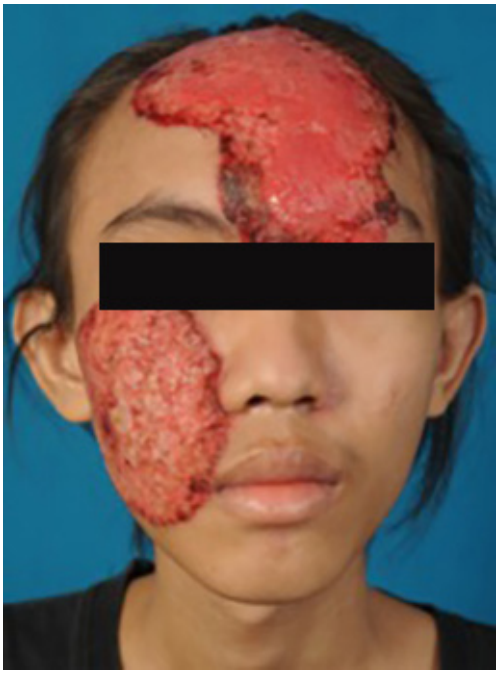

Fig 2. Chronic herpes simplex infection lesions after 3 weeks of oral acyclovir and oral/topical antibiotics treatments.

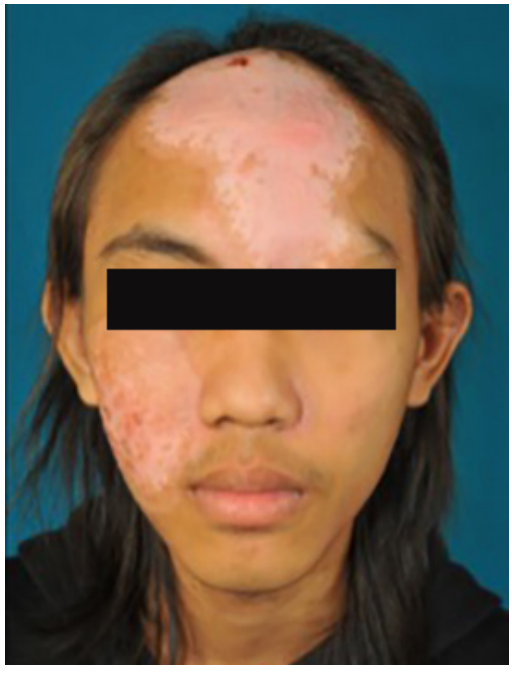

Fig 3. The lesions at follow-up visit after two months of high-dose oral acyclovir (zoster dosage regimen).

DISCUSSION

Chronic herpes simplex infections (CHSV) frequently recur with multiple small to extensive hyperkeratotic or ulcerative lesions in immunocompromised patients, including HIV infection.[5] However, no direct association between the CD4-positive count and the occurrence of CHSV had been observed.[5] The lesions are predominant at anogenital sites which persist for at least 1 month.[1] Wart-like CHSV is usually found on the skin. The ulcerative form, on the other hand, nearly always involves the mucosa.[4] Apart from oral and anogenital ulcerative lesions, they are typically asymptomatic.[5] These lesions are difficult to obtain the swab for cell culture or Tzanck smear; thus, DNA detection should be done to confirm the diagnosis.[6,7] The bacterial or Candida species colonization may present. Related internal dissemination or any fatal cases have not been reported.[5] The pathogenic mechanism remains unclear. A genetic predisposition seems likely.[3] An epidermal growth factor-dependent pathway and inadequate immune response have also been proposed.[8] Initially, CHSV should be treated with oral acyclovir at a dose of 200 mg five times a day for 1-2 weeks[5],although for disseminated or severe herpes infections, the treatment of choice is intravenous acyclovir 5-10 mg/kg every 8 hours. If acyclovir resistance is suspected, drug susceptibility test should be requested.[9] In case of treatment resistance, the dose should be increased to 800 mg at the same frequency and duration. If no improvement is seen, oral valacyclovir or famciclovir, topical foscarnet, and combination therapies (topical, oral, intravenous) should be

considered respectively. Cryosurgery, topical imiquimod, and oral thalidomide are the alternative steps. Resistance to thymidine kinase (TK)-dependent antiviral agents is one of the most common complications of CHSV. Hyperpigmentation, hypopigmentation and scarring are frequently observed after healing.[5] HIV-infected patients receiving suppressive acyclovir therapy were found to have lower HIV RNA levels,[10] but long-term prophylaxis can lead to the development of drug-resistance.[11]

As with our case, the patient had two large lesions of chronic herpes simplex infection lasting for a year. The lesions were typically asymptomatic and ulcerative, but located on an uncommon location. Tzanck smear was negative whereas HSV antigen detection by direct fluorescent antibody was positive. However, sensitivity of Tzanck smear and culture from ulcerative lesions are usually very low, so HSV DNA detection should be requested to confirm diagnosis. There was superimposed staphylococcal infection without any internal dissemination. The patient was prescribed with a standard dose of acyclovir, but extended for another week (totally 3 weeks) together with topical and oral antibiotics, but lesions progressed. Then, we doubled doses for 2 weeks according to treatment guideline. Without clinical improvement, the next step of treatment is oral famciclovir or valacyclovir,[5] but our patient could not afford. Therefore we continued the same acyclovir dose totally for 2 months. The lesions were smaller with no ulcer. There was a case report of a patient with chronic mucocutaneous herpes virus infection successfully treated with oral acyclovir up to 65 days.[12] After 3-month follow up, the lesions were obviously improved. HSV antigen detection was repeated and the result was negative. Hypopigmentation remained after healing. We advised the patient about the possibility of recurrence and the necessity of routine follow up under no suppressive acyclovir therapy.

Conflict of interest disclosure: The authors declare that there is no conflict of interest regarding the publication of this observation.

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