Clinical mimicry by herpetic ulceration in a HIV positive teenager

Abhishek Bhardwaj, Bhagirath S. Rathore, Charu Sharma, Garima Singh
Departments of Dermatology and Venereology, and Obstetrics and Gynaecology, Subharti Medical College, Meerut, Uttar Pradesh, India

Address for correspondence:
Dr. Abhishek Bhardwaj, Department of Dermatology, Subharti Medical College, Subhartipuram, Bye-Pass Road, Meerut - 250 005, Uttar Pradesh, India. E-mail: dr.abhishekbhardwaj@gmail.com

Abstract

The human immunodeficiency virus (HIV) is known to cause altered disease presentations. We present here, the case of a 14-year-old boy who came to us with a chronic, painful, nonhealing ulcer of 4 months duration over the dorsum of right hand. Before our observation, he was variably diagnosed and treated as atypical mycobacterial infection, deep fungal infection, squamous cell carcinoma, and pyoderma gangrenosum. On administration of systemic corticosteroids his condition worsened, after which he was tested for, and found to be HIV positive. He was put onto valacyclovir, responded slowly, with healing after 2 months of antiviral therapy. The case report highlights unusual presentation in an under-considered age group and a slow response to otherwise effective therapy.

Key words: Human immunodeficiency virus, herpes, nonhealing ulcer, pyoderma gangrenosum

INTRODUCTION

The synergistic nature of human immunodeficiency virus (HIV) and herpes infections continue to rise in importance. Individuals affected by HIV have atypical presentations of herpes. Herpetic ulcerations increase chances of HIV transmission. In AIDS, more and more cases of bizarre ulcerations are being attributed to herpes simplex virus (HSV) infection. Asymptomatic perianal shedding of HSV, creates a ground for inoculation and causation of herpetic ulcers over hands, especially keeping in mind the peculiar hygiene habits.

CASE REPORT

A 14-year-old male presented to us with complaint of a nonhealing ulcer over dorsum of his right hand for 4 months. The ulcer began as a small pustule that ruptured and formed an ulcer within 7 days of appearance. There was no history contributing the onset to either an insect bite or trauma to the site. The ulcer was particularly painful. The severity of the pain was increasing with the increase in size of the ulcer and it had a tendency to worsen during night. There was no other symptom either dermatological or systemic.

On examination, an irregular ulcer measuring 8 cm × 6 cm was noticed over the dorsum of right hand. The ulcer was extending onto the index and middle fingers [Figure-1]. There were two discrete intact pustules over the skin covering the proximal interphalangeal joints beyond the edge of ulcer. The ulcer was covered partly by purulent exudate and unhealthy granulation tissue. The involved fingers showed a partly correctible flexion deformity.

The initial workup of the patient showed that his hemoglobin was 10 g%, total leukocyte count was 7,200, with differential leukocyte count (DLC) being polymorphs 58%, lymphocyte 40%, eosinophils and monocytes both 1%, erythrocyte sedimentation rate

How to cite this article:
Bhardwaj A, Rathore BS, Sharma C, Singh G. Clinical mimicry by herpetic ulceration in a HIV positive teenager. Indian J Sex Transm Dis 2015;36:74-6.
(ESR) was 48 mm, and random blood sugar was 94 g/dl. Mantoux test done showed little reaction and was deemed negative. Gram’s stain did not yield any result. KOH mount showed some budding yeast cells, but no pseudohyphae. Tzanck smear showed a few polymorphs but no multinucleate giant cells. Both fungal and bacterial cultures did not yield any result. The viral culture was not carried out in light of absence of multinucleate giant cells and financial constraints. The histopathology showed that there was a large ulcer with no overlying epidermis. Ulcerated area showed dense infiltrate of neutrophils and macrophages. Dermis beneath the ulcer showed dense infiltration of neutrophils and lymphohistiocytes. Blood vessels beneath the ulcer showed fibrinoid necrosis and extravasation of erythrocytes. Large necrotic areas were present in dermis. Histopathological interpretation was pyoderma gangrenosum.

On the basis of the biopsy report, the patient was diagnosed as a case of pyoderma gangrenosum and put onto systemic corticosteroids and other symptomatic drugs. Within 1 week, the symptoms of the patient began to worsen. The patient took another consultation where on further work up he was diagnosed to be HIV positive (HIV1). His CD4 count came out to be 154.

Considering the presentation of a nonhealing ulcer in a HIV positive patient as the clinical background and the past failures of his multiple treatments, the patient was diagnosed to be a case of herpetic ulceration. He was put onto valacyclovir in a dose of 500 mg BD. Due to a tardy response after 2 weeks [Figure 2], the dose of valacyclovir was escalated to 1 g BD. Other treatment modalities were symptomatic. Interestingly the patient got the best pain relief from amitriptyline 10 mg. Meanwhile the patient was put onto antiretroviral therapy (ART) after 1 week of starting valacyclovir. His ART comprised of zidovudine, stavudine, and nevirapine. After 2 weeks of valacyclovir therapy, the ulcer began to dry up and started to show signs of shrinking. The base of the ulcer got covered by a thin pinkish scar. The flexion deformity was also corrected [Figure 3]. Valacyclovir was stopped, while ART was continued.

**DISCUSSION**

Herpetic ulcers are usually noted over the perioral and genital region in the immunocompetent individuals. They can affect the hands and manifest as whitlow and cellulitis. They are seen commonly too in HIV positive individuals. These ulcers greatly enhance the possibility of transmission of HIV. In the setting of immunodeficiency, these ulcers become chronic (herpetic ulcers of more than 1 month duration are considered by the Centre for Disease Control to be an AIDS defining illness[1]) and
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Atypical. Atypical presentations of herpes such as hemorrhagic deep painful ulcers, vegetating plaque with ulceration, hyperkeratotic verrucous plaques, erythematous and papulovesicular rash, are found in HIV positive individuals. Besides persistent ulcers around mouth and genitalia, herpetic ulcers have been observed over perianal region and reported from dorsal as well as ventral aspect of hands and feet. Perianal region is significant because it is the most common site of asymptomatic viral shedding and can inoculate hands due to hygiene habits.

Our patient came with a chronic ulcer with vegetating margins. Before we initiated antiviral treatment, the patient had undergone multiple therapies at other centers. He was first diagnosed as atypical mycobacterial infection and treated with antitubercular therapy. On poor response, the diagnosis was revised and the patient was put on topical and systemic antifungals. There was no response to the revised treatment. Then the biopsy of the lesion was carried out. His biopsy report indicated his condition to be pyoderma gangrenosum. The patient was put onto systemic glucocorticosteroids. This aggravated his condition as steroids added to the already present immunodeficiency due to HIV. We identified the cause of his immunodeficiency and started his treatment with antivirals (valacyclovir), which ultimately gave him relief.

Besides atypical mycobacterial infection and deep fungal mycoses, herpetic ulceration can be easily misdiagnosed as pyoderma gangrenosum, such is the clinical mimicry of this condition. In fact, herpetic ulcers misdiagnosed as pyoderma gangrenosum have been reported on more than one occasion. Indeed, nonspecific findings on histopathology can add to the confusion in the setting of immunodeficiency.

Pyoderma gangrenosum is a diagnosis of exclusion to be considered when all causes of ulcers, including infections have been ruled out. Such a misdiagnosis can result in substantial complications in patients who have other causes of severe cutaneous ulceration. We highlight the clinical mimicry caused by herpetic ulceration leading to multiple misdiagnoses. There are reports where not only genital but also extragenital HSV infections have been misled by HIV infection.

Even on arriving at the correct diagnosis, the treatment requires escalated dosing of acyclovir (antivirals). One also needs to keep in mind acyclovir resistance in cases of poor response for the successful management of this condition, in the setting of immunocompromise as HIV.

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Source of Support: Nil. Conflict of Interest: None declared.