

SHORT COMMUNICATION

Disseminated HSV-2 Infection in a Person Living with HIV (PLWH)

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A woman in her 20s with a past medical history of human immunodeficiency virus (HIV) and genital ulcer disease (GUD) presented with progressively pruritic, painful, and purulent cutaneous ulcers. Her HIV was inconsistently treated with antiretroviral therapy (ART) and her GUD was managed with intermittent courses of oral valacyclovir for presumed herpes simplex virus (HSV) infection. Two months prior to presentation, she was admitted for an eruption involving the pelvis and torso. Her eruption was positive for HSV-2 by viral PCR. Labs showed an HIV viral load (VL) of 86,184 copies/mL and a CD4 count of 9mm³, consistent with acquired immunodeficiency syndrome (AIDS). She was treated with 5 days of IV acyclovir and discharged with daily ART and 10 days of oral valacyclovir (1g BID).

Upon presentation to our hospital, she endorsed adherence to her medications with progressive skin erosions, new mild bilateral hearing loss, fevers, and weight loss. Physical examination revealed numerous large ulcerations with scalloped borders and a yellow-pink bases of the face, back, perianal region (Figure 1), and purulent ulcerations of the bilateral ears (Figure 2).

Laboratory results showed a suppressed HIV viral load of <20 copies/mL and a CD4 count of 13mm³.



Figure 1.



Figure 2.

A swab from her facial glabellar lesion was positive for HSV-2 by viral PCR. Viral culture confirmed HSV-2 infection, but susceptibilities could not be obtained. Culture of auricular fluid grew *Staphylococcus aureus*, group C beta-hemolytic *Streptococcus*, and mixed anaerobes. Magnetic resonance imaging of the head was negative for mastoiditis. A diagnosis of disseminated HSV-2 with bacterial otitis externa was made. We suspect immune reconstitution inflammatory syndrome (IRIS) was responsible for her exuberant HSV-2 presentation in the setting of appropriate treatment and a rapid decline in HIV VL. She received 9-days of IV acyclovir and transitioned to oral valacyclovir as her lesions improved. She was continued on ART and initiated on prolonged

antibiotics with resolution of her otitis externa. At five months post-discharge, her skin lesions were resolved and she continues on treatment dose valacyclovir.

HSV-2 is the leading cause of GUD, classically manifesting as painful clustered vesicles or erosions on an erythematous base.¹ Eruptions favor mucocutaneous surfaces, and recurrences are common due to the establishment of neuronal latency in the dorsal root ganglion.¹ Persons living with HIV (PLWH) have an increased risk for severe reactivation with atypical morphologies. Higher VL's and lower CD4 counts correlate with increased frequency, duration, and severity of flares.² In addition to the large ulcerations seen in our patient, fissures, hypertrophic masses, verrucous growths, and pseudotumors have been described in PLWH.¹

Initiation of ART in critically immunosuppressed PLWH can cause immune dysregulation and paradoxically amplify opportunistic infections, inflammatory conditions, and autoimmune diseases – a phenomenon called IRIS.³ Dermatological manifestations comprise approximately half of reported IRIS events.⁴ These include mycobacterial abscesses, sarcoid-associated erythema nodosum, and Kaposi sarcoma.⁵ Notably, up to 11% of patients started on ART can have HSV reactivation,³ and while oftentimes mild, these cutaneous presentations can be life-threatening.⁴ This case demonstrates a striking example of IRIS requiring extended, multidisciplinary management.

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