Anogenital Herpes Simplex Virus Infection Mimics Sacral and Gluteal Pressure Injuries

Igor Melnychuk, MD, CLT and Alexandra Gaither, OMS IV

Sacral and gluteal ulcerations are commonly encountered in wound care practice. Typically, these ulcerations are attributable to pressure injuries, skin tears, and erosive moisture-associated skin dermatitis. Encountering multiple gluteal ulcerations is uncommon and can be puzzling when they occur in areas away from bony prominences or when moisture is absent. However, there is a subtype of sacral and gluteal ulcerations unrelated to the above: Anogenital herpes simplex virus (HSV) infection can also present as recurrent sacral and gluteal ulcerations.

HSV INFECTION

Herpes simplex virus is very prevalent: More than 90% of adults have antibodies to HSV type 1, and HSV type 2 seroprevalence is 16%.[1] The cumulative lifetime incidence of HSV-2 is 25% in White women, 20% in White men, 80% in Black women, and 60% in Black men.[1] Even though it is typically thought that HSV-1 causes herpes labialis and HSV-2 causes herpes genitalis, HSV-1 causes a significant number of herpes genitalis infections as well. Both HSV-1 and HSV-2 are well adapted to infect most regions of the body, except for the restricted ability of HSV-2 to establish an orofacial infection.[1]

Among patients with primary HSV-2 infection, 9% develop extragenital lesions, most commonly on the buttocks.[1] Although 80% of seroprevalent individuals deny genital lesions, their genital lesions may not be recognized and diagnosed as herpetic.[2] Further, HSV-2 has high morbidity due to a 90% reactivation rate.[3] Herpetic infections are contagious because two-thirds of individuals with mucosal HSV-1 or HSV-2 shed the virus asymptomatically.[4]

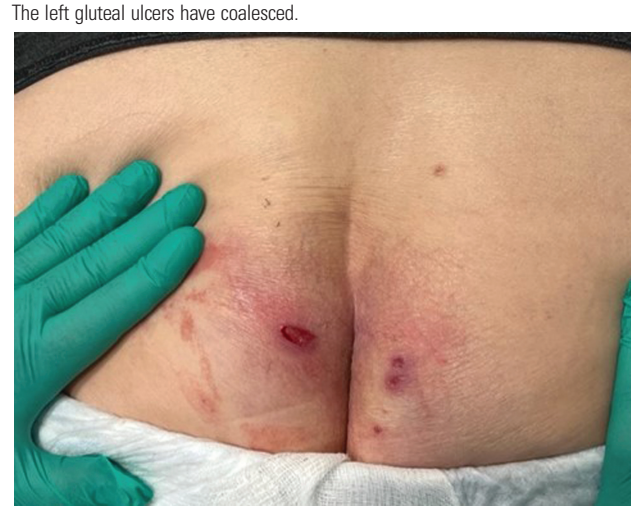
HSV-RELATED ULCERATION

Anogenital HSV is contained within sacral dorsal root ganglia, where it can remain dormant. It can cause sacral and gluteal ulcerations in both immunocompetent and immunocompromised adults, although HSV-related ulcerations are usually shallow and typically resolve faster in immunocompetent patients (Figure 1). Lesions in immunocompromised patients appear more often in atypical locations, such as the buttocks and lower back,[1,5–7] and can develop into chronic necrotic ulcerations that are larger and much more difficult to heal. These ulcerations can also worsen due to ongoing mechanical forces or recur. Without timely treatment, herpetic ulcerations can coalesce. Clinicians often fail to recognize the true culprit: HSV. Some examples of extragenital HSV manifestations include "herpes gladiatorum," occurring in various areas of skin-to-skin exposure from contact sports, and "herpetic whitlows," typically located on the tips of the fingers.

When anogenital HSV causes gluteal ulcerations, they typically form from multiple small vesicles. When vesicles rupture, round erosions form, which can coalesce due to pressure and shearing. Patients can also self-inoculate to distant sites. Many patients report a recurrence of gluteal ulcerations. During the reactivation phase, immunocompromised patients often develop more severe disease, lasting up to 2 weeks.[6] When nonhealing erosions and/or ulcers appear on the buttocks of an immunosuppressed patient, HSV infection should be

Figure 1. SHALLOW GLUTEAL ULCERATIONS IN AN IMMUNOCOMPETENT HOST

The left gluteal ulcers have coalesced.



The patient provided consent for this image to be published.

Igor Melnychuk, MD, CLT, is Clinical Assistant Professor, Edward Via College of Osteopathic Medicine Carolinas, Spartanburg, South Carolina; Adjunct Assistant Professor, Department of Surgery, University of North Carolina School of Medicine at Chapel Hill; and Chief, Wound Care Department, Charles George Department of Veterans Affairs Medical Center, Asheville, North Carolina, United States. Alexandra Gaither, OMS IV, is Student, Edward Via College of Osteopathic Medicine Carolinas. **Disclaimer:** The contents of this publication do not represent the views of the US Department of Veterans Affairs or the US Government. Submitted March 9, 2023; accepted in revised form April 27, 2023.

Figure 2. NECROTIC SACRAL AND GLUTEAL ULCERATIONS IN AN IMMUNOCOMPROMISED HOST

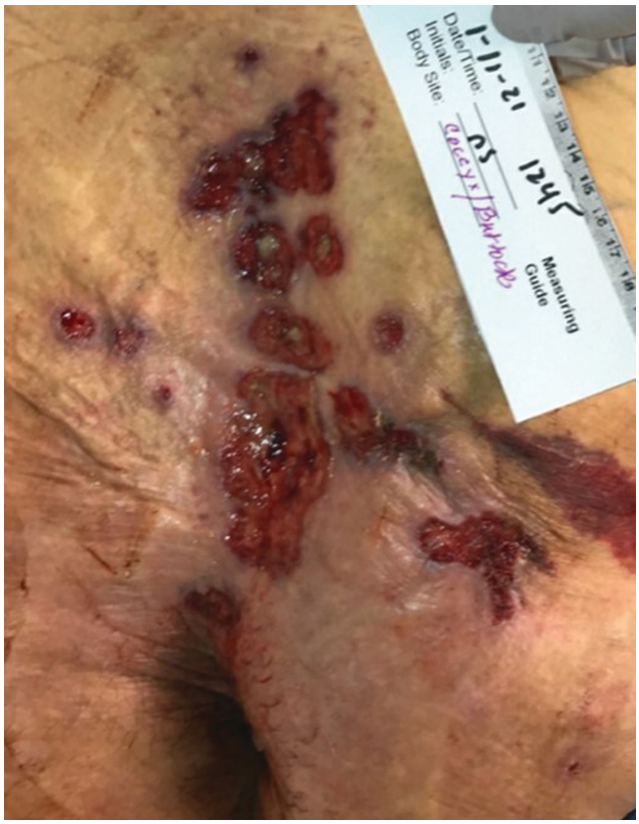


Image courtesy of John Greene, MD. The patient provided consent for this image to be published.

considered.[7] The hallmark of HSV ulcerations is serpiginous borders (Figures 2–4).[6]

Anogenital HSV may appear similar to erosive irritant contact dermatitis (ICD), which can also present with skin erosions or ulcerations, especially if pressure or shear is involved.[8] The presence of underlying erythema and edema of the affected area may point to ICD over HSV. Certainly, both conditions can coexist. Pressure injuries rarely appear as a cluster of ulcerations with serpiginous borders. Further, anogenital HSV lesions are not often located over bony prominences.

Violaceous edge discoloration of the long-standing HSV lesions seen in Figures 2–4 may add pyoderma gangrenosum (PG) to the differential diagnosis. Classical ulcerative PG typically starts as a painful papule or pustule and progresses to a painful ulcer with a violaceous undermined border and a purulent base.[9] Similarly, anogenital HSV may start as multiple blisters with clear fluid and progress to ulcerations. However, these may be painless or mildly painful, whereas pain associated with PG is typically severe and out of proportion to lesion. In addition, PG is typically associated with a preceding trauma and occurs in individuals with predisposing autoimmune conditions; it is rarely located in the gluteal or sacral areas where anogenital HSV is typical.

DIAGNOSIS AND TREATMENT

The diagnostic approach includes clinical recognition of the atypical manifestation. Multiple ulcerations may have started as blisters or recurred. They are often located in the sacral area, along the gluteal fold, or in the center of the buttocks. Serpiginous borders of the ulcer should always suggest herpetic origin. When the ulcerations are new, a swab checking for an HSV-1 and HSV-2 DNA is the recommended modality. If available, HSV DNA detection is the preferred diagnostic method because it is three to four times more sensitive than viral isolation, less affected by variation in specimen transport, and cost-effective as compared with culture.[10] However, the virus may no longer be recoverable in the ulcerations after a few weeks, and at that point checking HSV-1 and HSV-2 immunoglobulin G antibodies via Western blot

Figure 3. SACRAL ULCERATIONS IN AN IMMUNOCOMPROMISED HOST

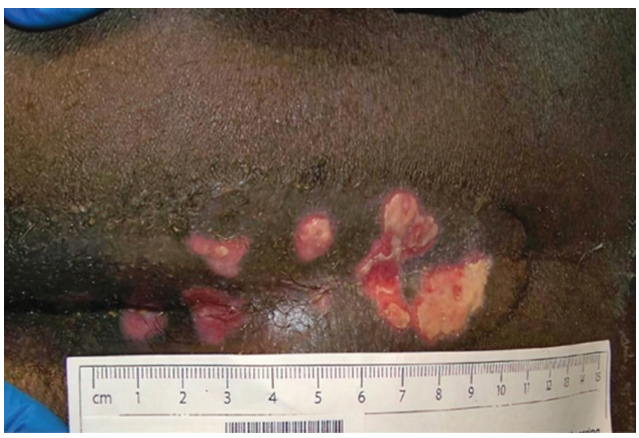


Image courtesy of John Greene, MD. The patient provided consent for this image to be published.

Figure 4. SERPIGINOUS BORDERS OF HERPETIC ULCERATIONS IN AN IMMUNOCOMPROMISED HOST

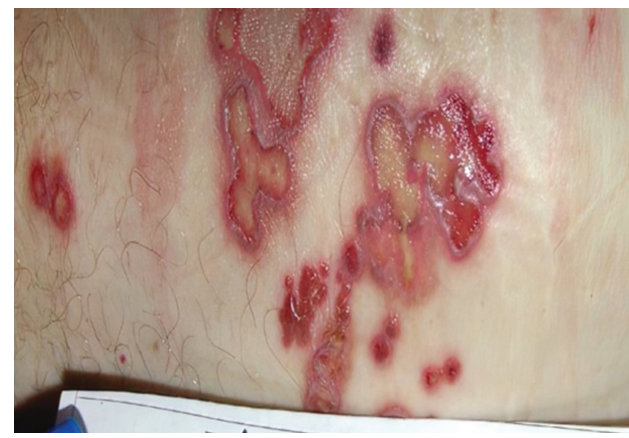


Image courtesy of John Greene, MD. The patient provided consent for this image to be published.

may confirm clinical suspicion. Herpetic cultures are not very sensitive and not used in general practice.

Treatment includes either oral aciclovir, valaciclovir, or famciclovir for 7 to 10 days. Unlike aciclovir, valaciclovir or famciclovir have better bioabsorption. Intravenous aciclovir at a dose of 5 mg/kg is used in critically ill patients or patients with severe immunocompromise.[1]

CONCLUSION

Wound care clinicians need to keep anogenital HSV infection in their differential diagnosis of sacral and gluteal ulcerations and be able to differentiate them from pressure injuries. Early recognition and treatment may improve outcomes. ●

REFERENCES

1. Schiffer JT, Corey L. Herpes simplex virus. In: Mandell, Douglas, and Bennett's Principles and Practice of Infectious Diseases. Bennett JE, Dolin R, Blaser MJ, Eds. Elsevier Health Sciences; 2014.
2. Löwhagen GB, Tunbäck P, Bergström T. Proportion of herpes simplex virus (HSV) type 1 and type 2 among genital and extragenital HSV isolates. Acta Derm Venereol 2002;82(2):118-20.
3. Phipps W, Saracino M, Margaret A, et al. Persistent genital herpes simplex virus 2- shedding years following the first clinical episode. J Infect Dis 2011;203:180-7.
4. Wald A, Huang ML, Carrell D, et al. Polymerase chain reaction for detection of herpes simplex virus (HSV) DNA on mucosal surfaces: comparison with HSV isolation in cell culture. J Infect Dis 2003; 188:1345-51.
5. Lautenschlager S, Eichmann A. The heterogeneous clinical spectrum of genital herpes. Dermatology 2001;202(3):211-9.
6. Yacoub AT, Kovacs SN, Jones L, Mai J, Greene JN. Atypical cutaneous manifestations of herpes infection in immunocompromised hosts. Infect Dis Clin Pract 2015;23(3):123-5.
7. Hanafusa T, Yamaguchi Y, Kitaba S, Katayama I. Intractable wounds from a herpes simplex infection in an immunosuppressed patient with rheumatoid arthritis. Arch Dermatol 2007;143(10): 1331-44.
8. Gray M, Bliss DZ, McNichol L. Moisture-associated skin damage. Expanding and updating practice based on the newest ICD-10-CM codes. J Wound Ostomy Continence Nurs 2022;49(2): 143-51.
9. Powell FC, Hackett BC, Wallach D. Pyoderma gangrenosum. In: Fitzpatrick's Dermatology in General Medicine. 8th ed. Goldsmith LA, Katz Sim Gilchrest BA, Eds. McGraw-Hill Companies, Inc; 2012:(1)371.
10. Wald A, Zeh J, Selke S, et al. Reactivation of genital herpes simplex virus type 2 infection in asymptomatic seropositive persons. N Engl J Med 2000;342:844-50.