Recurrent lumbosacral herpes simplex virus infection

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We present the case of a 54-year-old white woman with episodic lumbosacral lesions that she had been treating as psoriasis. Evaluation revealed classic herpes simplex virus (HSV) infection. The discussion reviews the significance and potential complications of recurrent lumbosacral HSV infection.

Herpes simplex virus (HSV) is a DNA virus responsible for recurrent skin infections presenting with clusters of fluid-filled vesicles on an erythematous base. The lesion forms a characteristic scalloped border as the vesicles progress to crusting, erosions, and/or ulcerations. The lesions usually resolve fully in 2 to 4 weeks, frequently leaving a residual area of discoloration at the site of involvement. The virus infects a susceptible person through contact with mucous membranes or open, abraded skin. Most infections are recurrent, with subsequent episodes reappearing at or near the same anatomical location due to the viral invasion, latency, and reactivation within sensory dorsal root ganglions. The outbreaks can be triggered by trauma, ultraviolet light, temperature extremes, emotional stress, or immunosuppression (1).

CASE PRESENTATION

A 54-year-old white woman presented to our dermatology clinic for her biannual skin evaluation. She had a history of rosacea and psoriasis, but no personal or family history of skin cancer. She had previously been on doxycycline for her rosacea with an evident flare since being taken off the month prior to her evaluation due to high liver enzymes. She was given a prescription for topical ivermectin 1% to apply to her face once a day. Her psoriasis was adequately controlled with clobetasone spray 1 to 2 times per day applied regularly to her arms, scalp, and legs. The patient stated that she also used the spray on her lower back when she had a "psoriasis" flare with less than adequate response in that region. On direct questioning, she stated that the episodes on her back were associated with an initial burning sensation in addition to being more painful and tender than the rest of her psoriasis plaques.

Examination of the face revealed moderate erythema and telangiectasia with accompanying papules on her cheeks and

nose, typical of rosacea. The patient also had scattered psoriasis scaly plaques on her arms and occipital scalp. Evaluation of the patient's lumbosacral region revealed two well-circumscribed clusters of small vesicles to the left of midline (*Figure 1*). The cluster directly at the base of her spine measured 15 × 10 mm and showed crusts and scabbed, deroofed vesicles. The cluster to the left measured 15 × 15 mm and showed multiple thin-walled fluid-filled vesicles on an erythematous base. These findings were indicative of HSV infection with a characteristic history of prelesion "burning" symptoms and recurrences in the same anatomical area.

DISCUSSION

HSV is categorized as type 1 and 2 viruses which typically cause infections on the oral and genital mucosa, respectively (2). HSV-2 causes 70% to 90% of genital herpes infections (3). Nonoral and nongenital sites are not infrequently involved, possibly due to self-inoculation, primary acquisition, or viremic spread (4). Although recurrences predominantly occur at the same location, studies have shown that 21% of patients with primary genital herpes develop nongenital involvement (5). The sites most often affected are the lumbosacral area and legs, as the pudendal nerve which innervates the external genitalia originates from the sacral nerve ganglia of S2-4 (*Figure 2*) (5-7).

With evidence of genital herpes recurrence at nongenital sites, patients with primary infection should be counseled to look for the development of lesions in the sacral ganglia distribution, as in our patient. On the other hand, patients with nongenital herpes should also be evaluated for genital herpes. Patients should be warned of viral shedding from the genital area, as data have shown concomitant shedding with reactivation in the buttock area even in the absence of active genital lesions (8). To minimize transmission of infection, patients are

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Figure 1. Location and presentation of two lumbosacral clusters of vesicles in different stages of development and healing.

counseled to avoid sexual contact during recurrences (8). The viral shedding also raises concern for neonatal herpes infection during delivery. Women most commonly shed from the vulva, cervix, and perianal areas, and genital shedding at delivery causes a 300-fold higher risk of transmitting the virus (9, 10).

Palliative or bedridden patients are at additional risk for HSV. Although macerated dermatitis, *Candida* infections, and pressure sores are most commonly seen in the posterior lower body area, a lesion that does not heal despite appropriate treatment should be assessed for HSV-2. Early detection and management can decrease complications and pain (11). Recurrences of HSV-2 lesions on the buttocks occur less frequently than genital recurrences but tend to last longer, thus making intermittent rather than suppressive therapy possible (5).

Our patient noted that the episodes on her lower back occurred approximately once or twice a year. While the pain usually subsided within a few weeks, the lesions themselves took up to 2 to 3 months to completely heal. The recurrences had been occurring for many years with no patient recall of ever having genital lesions. She had a hysterectomy several years previously. The patient was prescribed valacyclovir, and her obstetrician was notified of her condition. Prophylactic treatment was not recommended, and the patient was advised to take valacyclovir at the very first sign of a flare, particularly early stinging or burning in the lumbosacral region.

Figure 2. Distribution of sacral nerve ganglia dermatomes within the perineal region.

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