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CASE REPORT

Herpes Zoster of the Sacral Region without Motor Dysfunction in a 55-year-Old Female: A Case Report and Literature Review

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Abstract: Herpes zoster infection, commonly known as shingles, typically involves the thoracic and lumbar dermatomes. Sacral involvement is rare, occurring in only 4–8% of cases. This report presents a 55-year-old female with herpes zoster affecting the S1 dermatome, manifesting as burning pain and clustered vesicles on her right leg, without motor dysfunction or urinary symptoms. A clinical diagnosis was made based on a characteristic unilateral S1 dermatomal vesicular eruption. Despite the rarity of sacral herpes zoster without motor involvement, the patient responded well to antiviral treatment and neuropathic pain management, with no complications noted in the long-term follow-up. This case highlights the necessity for clinicians to consider sacral herpes zoster in patients with radicular pain to prevent misdiagnosis.

Keywords: herpes zoster, sacral dermatome, radiculopathy, shingles, neuropathic pain

Introduction

Varicella, commonly referred to as chickenpox, is a contagious disease caused by primary infection with varicella-zoster virus (VZV). Following primary infection, the virus remains latent in the sensory and autonomic ganglia. Complications are more frequent in geriatric and immunocompromised individuals, although primary varicella in older adults is rare.[1]

Herpes zoster (HZ) infection, also known as shingles, is caused by reactivation of the VZV. Reactivation typically occurs due to an age-related decline in immunity or immunosuppression, although HZ can also manifest in healthy individuals.[2–4] Clinically, HZ is characterized by a painful, unilateral rash distributed along a single dermatome in patients with prior varicella infection.[5] A common complication of HZ is post-herpetic neuralgia, defined as persistent pain lasting >3 months after the rash has healed.[3] HZ most frequently affects the thoracic and lumbar dermatomes; involvement of the sacral nerves is relatively rare, occurring in only 4–8% of cases.[4,6,7] In cases where the sacral nerves are affected, patients may experience sensory and motor deficits that affect the bladder and bowel, potentially resulting in urinary dysfunction, incontinence, or constipation.[6,8,9] This report presents a rare case of sacral HZ localized to the S1 dermatome, accompanied by significant burning pain but without motor dysfunction.

Case Presentation

A 55-year-old woman (weight: 100 kg, height: 160 cm, body mass index: 39.06 kg/m^2 (classified as class II morbid obesity)) presented to the Neurology Clinic with an aching pain that had begun 4 days prior to her visit. The pain primarily affected her right leg, extending from the lower back to the base of her foot. Described as continuous, progressive, and burning (particularly in her toes), the pain lasted approximately 2 weeks from its onset. A cluster of vesicles appeared on the lateral side of her right leg, extending to the gluteal region; these tender lesions had developed 1 day before her clinic visit and had

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persisted for 10 days. She also reported muscle weakness, itching, intermittent numbness, tingling, and loss of sensation in her right leg, which had initially been masked by the pain but became more apparent as the pain subsided. The patient had no known contact with sick individuals, no history of similar symptoms, and had neither taken new medications nor received immunosuppressive treatments. She had not been hospitalized for any serious illness and had no recent infections. Therefore, her immunological status was intact, with no evidence of immunosuppression. She denied any constitutional symptoms, and her only additional complaint was lower back pain. The pain had begun to interfere with her daily life, making it difficult to sit or walk on some occasions. As a result, she arrived at the clinic in a wheelchair.

Her medical history revealed attempts to reduce her weight through dieticians, restrictive diets, and exercise; however, this had been unsuccessful. Despite her morbid obesity, she did not present with any obesity-related complications and was not on any chronic medications. Notably, she had received the varicella-zoster vaccine during childhood. The patient was employed as a teacher but reported no significant recent stressors aside from typical daily pressures.

A physical examination showed clustered vesicles on an erythematous base extending unilaterally along the S1 dermatome from the lateral side of her right leg to the dorsolateral aspect of her right foot (Figures 1 and 2). These

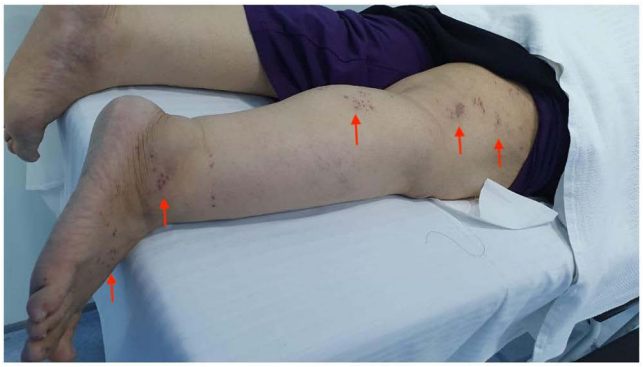

Figure 1 Grouped vesicular lesions over the right sacral and posterior thigh regions, consistent with Herpes Zoster. Arrows indicate areas of active vesiculation and crusting within the affected dermatome.

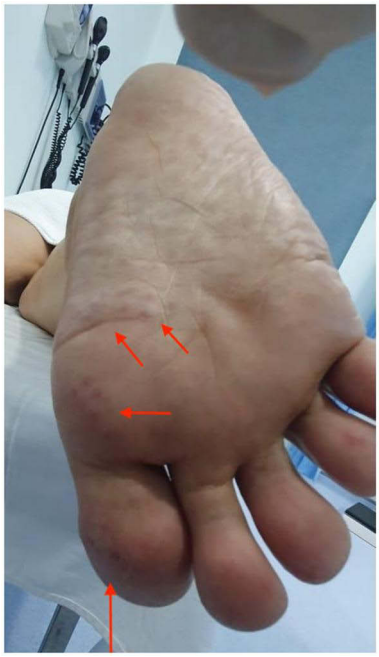

Figure 2 Plantar aspect of the foot showing scattered herpetiform vesicular lesions. Arrows highlight faintly erythematous lesions involving the S2–S3 dermatome, which are often overlooked during initial examination.

vesicles were tender to touch. A neurological examination revealed localized pain in the S1 dermatome, with no voiding dysfunction, hyperesthesia, or allodynia. The fine touch assessment showed a loss of sensation over the S1 dermatome, and proprioception was impaired in the distal digits of her right foot. In contrast, sensations and proprioception were intact in the contralateral side. The patient was afebrile, and her general examination was otherwise unremarkable.

Laboratory investigations showed mild anemia (hemoglobin: 11.4 g/dL), elevated low-density lipoprotein cholesterol (132 mg/dL), and vitamin D deficiency (20.6 ng/mL). Other parameters, including complete blood count (CBC), liver function tests (LFT), renal function tests (RFT), serological tests, and hemoglobin A1c, were within normal limits. Magnetic resonance imaging (MRI) of the lumbar spine, conducted for previous lower back pain, showed disc bulges at L3–L4 and L4–L5, partially encroaching on the corresponding exit foramina. Electromyography and nerve conduction studies were normal, ruling out any motor or sensory conduction abnormalities.

Based on her clinical presentation and the exclusion of other differential diagnoses, the patient was diagnosed with sacral (S1) HZ infection.

The treatment plan included oral acyclovir 400 mg every 8 h for 10 days, acyclovir cream applied topically to the affected area twice daily for 10 days, vitamin D3 50,000 IU weekly, calcium carbonate 600 mg once daily, and gabapentin 800 mg twice daily for 1 month. She was also counseled on the benefits of a weight-reduction diet and the potential benefits of physiotherapy for her lower back pain.

During her first follow-up visit, 7 days after starting treatment, the patient showed improvements, with reduced pain and resolution of the vesicles. She arrived using a cane instead of a wheelchair, as her pain intensity had decreased, although the loss of sensation in her right leg had become more prominent.

At her 1-month follow-up, conducted via a telephone call, the patient reported complete resolution of both pain and vesicles within 10 days of treatment initiation. Sensory symptoms, including tingling, numbness, and muscle weakness, had also subsided within 10 days. Notably, the sensation in her right foot took longer to recover than her thigh and shin. No complications, such as post-herpetic neuralgia, were observed during the follow-up period, and she continued to regain sensation gradually. The patient provided written informed consent for the publication of this case report and its associated figures.

Discussion

HZ infection, commonly referred to as shingles, is caused by the reactivation of the varicella-zoster virus, the same virus responsible for chickenpox (varicella). Following the initial infection, typically during childhood, the virus becomes latent in the dorsal root ganglia and can reactivate later in life, particularly in elderly or immunocompromised individual.[10,11] Although stressors, such as fever, trauma, or emotional disturbances, can increase the likelihood of reactivation, the primary mode of transmission for the varicella-zoster virus is through aerosolized droplets or direct contact with lesions. Once reactivated, the virus replicates and travels along sensory nerves, producing the clinical manifestations of HZ.

The typical presentation involves dermatomal radiculopathy, which is characterized by paresthesia, radiating pain, and the formation of painful vesicular lesions that are confined to a single dermatome.[2,10,12] While HZ can affect any sensory ganglion, it most commonly involves the thoracic dermatomes (56%), followed by the cranial, lumbar, and cervical dermatomes (11, 13, 11%, respectively), with sacral involvement being particularly rare, affecting only 4% of patients.[13] Sacral HZ is frequently associated with urinary dysfunction and bowel complications due to its involvement in the sensory and motor innervation of these systems.[14,15]

Table 1 presents a compilation of documented cases of rare sacral HZ. Due to its rarity, sacral HZ may be overlooked by clinicians, particularly when its symptoms are similar to those of other conditions. For example, a case report by Hung et al described a patient with sacral HZ who initially presented with symptoms of sciatic pain and weakness following a traffic collision. The initial diagnosis was a sacral fracture; however, this was later revised upon the appearance of vesicular lesions along the S1 dermatome. Only the subsequent appearance of vesicular lesions along the S1 dermatome served to confirm the diagnosis of HZ.[16] Furthermore, it has been demonstrated that trauma increases the risk of HZ at the site of injury by a factor of 12,[17] thereby further complicating the diagnosis in cases of recent physical trauma.[16,18]

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Table 1 Reported Case Studies in the Literature

Study	Number of Cases	Age (Year) and Gender	Manifestations	Diagnosis	Management	Outcome, Prognosis, and Complications
Hung et al[16]	1	70 male	Seven days following the onset of sciatic symptoms (pain, weakness, and numbness in the right leg) caused by a traffic collision, painful grouped erythematous plaques with vesicles were observed along the S2 dermatome on the right buttock and the posterior aspect of the right leg.	Sacral herpes zoster due to trauma.	Valacyclovir hydrochloride (500 mg/8 h for 5 days).	The lesions demonstrated healing, and the tingling pain exhibited a gradual resolution. The patient was discharged 42 days after admission.
Hur[6]	1	25 male	A dark red eschar of head skin was observed over the right side of the abdomen, accompanied by dysuria, bloody stools, anal pain, constipation, and voiding dysfunction with constitutional symptoms (fever, chills, and fatigue). On the eighth day, bilateral blisters and calluses were noted on both sides of the anus.	Provisional diagnosis: Scrub typhus. Final diagnosis: Herpes zoster.	Acyclovir (10 mg/kg/8 h for 7 days) and dexamethasone (10 mg/day for 6 days), which was subsequently modified to prednisolone (100 mg with tapering over 3 weeks).	The symptoms abated, and no complications were observed.
Chiriac et al[4]	1	35 male	The patient presented with acute, localized pain and grouped, painful erythematous vesicles on the lateral aspect of the calcaneus, the lateral plantar area, and the dorsal surface of digits III and IV, which correspond to the S1 dermatome.	Herpes zoster.	Acyclovir (3 g daily for 10 days) and non-steroidal anti-inflammatory drugs.	The patient exhibited favorable evolution and did not experience any short- or long-term complications.
Ekmekyapar et al[18]	1	77 male	The patient presented with pain and diffuse vesicular-pustular lesions over the right sacral (gluteal) region.	Herpes zoster.	Valacyclovir.	The patient was discharged without any scheduled follow-up appointments.
Dave et al[19]	1	76 male	The patient presented with maculopapular and vesiculopustular lesions on the left buttock and anal margin, which were associated with worsening anal pain, fresh rectal bleeding, and sudden onset of acute urinary retention.	Sacral herpes zoster.	A urethral catheter was placed, and 1500 mL of urine was drained. Intravenous acyclovir was administered (unspecified dose).	The patient's condition showed improvement, and was discharged 7 days later, with no complications reported.
Nishiyama et al[20]	1	77 female	The presence of multiple vesicles in the left sacral regions (S2 and S3) and the experience of pain was preceded by a state of voiding dysfunction, incomplete bladder emptying, and abdominal distention.	Elsberg syndrome due to herpes zoster in the sacral region.	A urinary catheter was inserted. Intravenous acyclovir (250 mg/8 h for 1 week) and acetaminophen (500 mg as needed) were administered.	The lesions and symptoms gradually reduced until the patient was discharged on the eighth day. A 6-month follow-up did not report any complications.

In contrast to the case described by Hung et al, the present case of sacral HZ occurred without any preceding trauma or stressor; therefore, this diagnosis was primarily based on the presentation of burning pain and vesicular lesions following the S1 dermatome. Despite the involvement of the sacral nerves, the patient did not develop neurogenic bladder or voiding dysfunction, with pain being the primary complaint.

This case report is limited by its clinical diagnosis without polymerase chain reaction confirmation of VZV, a short 1-month follow-up period, and lack of formal autonomic testing. However, the classic S1 dermatomal rash, normal MRI and nerve conduction studies to exclude other causes, and complete resolution of symptoms support the validity of our observations without affecting patient care.

Conclusion

Skin examinations are of great importance, yet frequently underappreciated, in diagnosing conditions such as sacral HZ. In this case, the early recognition of dermatological signs was crucial in reaching an accurate diagnosis. Physicians should consider sacral HZ as a potential differential diagnosis for patients presenting with acute radiculopathy, particularly when other causes have been excluded.

Ethical Statement

The study was approved by the Institutional Review Board of Imam Abdulrahman Bin Faisal University on on July 22, 2024 (IRB-PGS-2024-01-550), and we confirm that institutional approval was required for publication of the case details.

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Disclosure

The authors report no conflicts of interest in this work.

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