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A Complex Case of Sacral Herpes Zoster Complicated by a Vulvar Abscess Unmasking Uncontrolled Type 2 Diabetes Mellitus

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ABSTRACT

Introduction: Genital herpes zoster (HZ), a reactivation of the latent Varicella-Zoster Virus (VZV), is an uncommon clinical entity, as the sacral dermatomes are infrequently affected. The severity and clinical course of HZ can be profoundly influenced by underlying conditions that impair cell-mediated immunity, such as diabetes mellitus (DM).

Case Illustration: We present the case of a 60-year-old female who presented to the emergency department with a two-day history of an acute, severely painful, unilateral vesicular eruption in the sacral dermatome, consistent with HZ. Her condition was complicated by the rapid development of a large, fluctuant vulvar abscess. Crucially, initial laboratory investigations revealed severe hyperglycemia with a random blood sugar of 306 mg/dL, leading to a new diagnosis of Type 2 Diabetes Mellitus (T2DM).

Discussion: The patient's management necessitated a coordinated,

multidisciplinary approach. This involved high-dose oral antiviral therapy to control VZV replication, urgent surgical incision and drainage of the vulvar abscess for source control, broad-spectrum systemic antibiotics to treat secondary bacterial infection and sepsis, and aggressive glycemic control with a multi-dose insulin regimen. This report delves into the intricate pathophysiological triad where the impaired immunity of undiagnosed diabetes precipitated VZV reactivation, the acute viral infection triggered severe metabolic decompensation, and the resultant state of acute hyperglycemia and immune paralysis created a permissive environment for a life-threatening bacterial superinfection and abscess formation.

Conclusion: This case underscores the critical importance of including HZ in the differential diagnosis of acute, unilateral ulcerative genital lesions. Furthermore, it highlights how a severe or complicated HZ presentation can be the initial clinical manifestation of previously undiagnosed and uncontrolled T2DM, mandating prompt investigation for underlying metabolic disorders. The successful outcome demonstrates the necessity of an integrated multidisciplinary strategy in managing such complex clinical emergencies.

Keywords: Herpes Zoster; Shingles; Genital Ulcer; Vulvar Abscess; Type 2 Diabetes Mellitus; Immunocompromised Host.

INTRODUCTION

Herpes zoster (HZ), commonly known as shingles, is a viral disease caused by the reactivation of the Varicella-Zoster Virus (VZV), the same neurotropic alphaherpesvirus responsible for the primary infection of varicella (chickenpox).[1] Following the resolution of varicella, VZV establishes a lifelong latency within the sensory neurons of the cranial nerve and dorsal root ganglia.[3] The reactivation of this dormant virus is primarily restrained by a robust VZV-specific cell-mediated immunity (CMI), particularly involving T-lymphocytes.[6] A decline in this specific CMI, a phenomenon associated with immunosenescence, immunosuppressive therapies, or underlying disease, can permit the virus to replicate and travel anterogradely along the sensory nerve to the corresponding dermatome.[8] This process culminates in the classic clinical presentation of HZ: a painful, unilateral vesicular rash that is characteristically confined to a single dermatome.[1]

While HZ is a common condition, its topographical distribution is not uniform. The thoracic and lumbar dermatomes are most frequently affected.[10] Involvement of the sacral dermatomes is comparatively rare, reported in only 4–8% of all HZ cases.[11] When the S2 or S3 dermatomes are involved, the eruption manifests in the genital and perianal regions, a presentation termed genital HZ.[1] This atypical location can pose a significant diagnostic challenge, as its appearance may mimic more prevalent causes of genital ulcer disease, such as genital herpes simplex virus (HSV) infection.[1]

The clinical severity and risk of complications from HZ are significantly amplified in individuals with compromised immune systems. Diabetes mellitus (DM) is now firmly established as an independent risk factor that not only increases the incidence of HZ but also predisposes patients to a more severe disease course and a higher likelihood of complications.[8] The pathophysiology underlying this association is rooted in the deleterious effects of chronic hyperglycemia on the immune system. Hyperglycemia impairs both innate and adaptive immune responses, leading to attenuated T-cell function, which is the critical defense mechanism for

maintaining VZV latency.[6]

In an immunocompromised host, the complications of HZ can extend beyond the debilitating postherpetic neuralgia (PHN). The breach in the cutaneous barrier caused by the vesicular lesions creates a portal of entry for bacteria.[25] Secondary bacterial superinfection, commonly caused by *Staphylococcus aureus* or *Streptococcus pyogenes*, is a major concern.[3] In the context of the profound immune dysfunction seen in uncontrolled diabetes, this superinfection can progress from simple impetiginization to severe, deep-seated infections such as cellulitis, necrotizing fasciitis, or, as demonstrated in this case, abscess formation.[27]

Involvement of the sacral dermatomes is a relatively uncommon manifestation of herpes zoster, accounting for a small fraction of total cases. When the virus reactivates in the S2 or S3 dorsal root ganglia, the characteristic vesicular eruption appears in the genital and perianal regions, a condition known as genital HZ. This specific anatomical localization presents a significant diagnostic challenge. The initial presentation of painful vesicles and subsequent ulcerations in the genital area can closely mimic more common sexually transmitted infections, particularly genital herpes simplex virus (HSV) infection, leading to potential misdiagnosis and delayed initiation of appropriate antiviral therapy. Furthermore, sacral HZ can be associated with significant complications beyond the cutaneous eruption, including neurogenic bladder, urinary retention, and severe neuralgia, making accurate and timely diagnosis imperative for preventing long-term morbidity. [1]

Diabetes mellitus is not merely a condition that increases the incidence of HZ; it fundamentally alters the clinical course of the disease, predisposing patients to more severe presentations and a markedly higher risk of debilitating complications. The chronic hyperglycemic state characteristic of uncontrolled diabetes fosters a state of immune dysregulation, most notably impairing the cell-mediated immunity that is critical for containing the Varicella-Zoster Virus. This compromised immune surveillance allows for more aggressive viral replication, leading to a greater

burden of disease, more extensive and severe rashes, and a protracted healing process. Consequently, diabetic patients with HZ are more susceptible to complications such as secondary bacterial superinfections, deep tissue necrosis, and the development of postherpetic neuralgia (PHN), which is often more severe and refractory to treatment in this patient population. [1]

This report aims to present and analyze a complex case of sacral HZ complicated by a large vulvar abscess, which served as the sentinel event unmasking previously undiagnosed and uncontrolled Type 2 Diabetes Mellitus (T2DM). This case is particularly instructive as it vividly illustrates the intricate, bidirectional pathophysiological interplay between these conditions and underscores the absolute necessity of a rapid, coordinated, and multidisciplinary management approach to achieve a favorable outcome.

CASE ILLUSTRATION

Patient Information and Clinical History

A 60-year-old married female homemaker (Ny. B), residing in Dsn. Kramat, Sedarum, Nguling, Pasuruan, presented to the Emergency Department (IGD) of UOBK Rumah Sakit Umum Daerah Grati on August 14, 2025. The patient’s chief complaint was a two-day history of progressively worsening pain and swelling in her genital area. She reported that the condition began with pain and was followed by the eruption of blisters ("lenting di kemaluan"). The pain and swelling had become increasingly severe, and she also experienced systemic symptoms, including generalized malaise ("badan greges"), nausea, and a subjective sensation of feverishness ("ngongsrong"). The patient's past medical history was significant for a primary varicella (chickenpox) infection during childhood. She explicitly denied any known history of diabetes mellitus, hypertension, or cardiac disease. Her family history was non-contributory for similar conditions.

Physical Examination on Admission

On initial assessment, the patient appeared weak but was fully conscious, alert, and oriented (Compos Mentis, Glasgow Coma Scale score of 15). Her vital signs were as follows: blood pressure of 140/80 mmHg, a tachycardic heart rate of 100 beats per minute, a respiratory rate of 20 breaths per minute, and a body temperature of 36.4°C. A random blood sugar (GDS) measurement taken on arrival was markedly elevated at 306 mg/dL.

Systemic examination of the head, eyes, ears, nose, and throat was unremarkable, with no conjunctival pallor or scleral icterus. The neck was supple with no palpable lymphadenopathy or thyroid enlargement. Cardiovascular examination revealed a regular rhythm with normal heart sounds (S1, S2) and no audible murmurs or gallops. The respiratory examination was clear to auscultation bilaterally, with vesicular breath sounds and no rhonchi or wheezing. The abdomen was soft, non-tender, with normal bowel sounds, and no organomegaly.

The dermatological examination was most notable.

- **Location:** The lesions were localized to the genital region, involving the vulva, vagina, and perianal area.
- **Distribution:** The eruption demonstrated a striking unilateral pattern, strictly confined to the left side and not crossing the midline. This distribution was consistent with the involvement of the sacral (S2-S3) dermatomes.
- **Efflorescence:** As depicted in Figure 1, the examination revealed a large, edematous, and profoundly erythematous to violaceous plaque involving the entire left labia majora. Superimposed on this plaque were multiple grouped vesicles and pustules, some of which had ruptured to form erosions with early serosanguinous crusting. The entire lesion was exquisitely tender to palpation, indicative of acute neuralgia

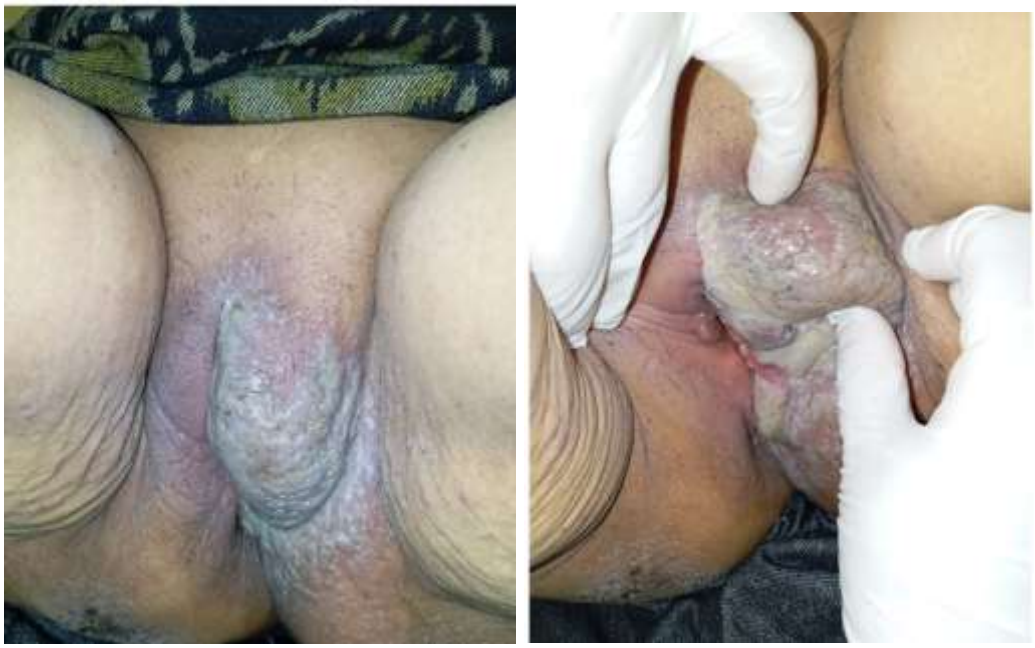

Figure 1. Clinical Presentation of the Patient Upon Admission

Investigations

A comprehensive set of investigations was performed to evaluate the patient's infectious, metabolic, and systemic status.

Laboratory Findings: Initial blood work revealed a significant inflammatory response and severe metabolic derangement. Key findings are summarized in Table 1. The results were consistent with a bacterial infection (leukocytosis with neutrophilia), an acute phase reaction (thrombocytosis), and uncontrolled hyperglycemia. Mild azotemia was also noted.

Table 1: Summary of Laboratory Investigations on Admission and Follow-up

Parameter	Result on Admission (14/08/2025)	Follow-up GDA (Day 2)	Follow-up GDA (Day 3)	Normal Reference Range
Random Blood Sugar (GDS/GDA)	**306 mg/dL**	205 mg/dL	171 mg/dL	< 200 mg/dL
White Blood Cell (WBC) Count	**13,080 /mm³**	-	-	5,000-10,000 /mm³
Segmented Neutrophils	**75.8 %**	-	-	40-75 %
Lymphocytes	13.8 %	-	-	15-45 %
Platelet Count	**441,000 /mm³**	-	-	150,000-400,000 /mm³
Blood Urea Nitrogen (BUN)	**23.85 mg/dL**	-	-	7.94-20.1 mg/dL
Serum Creatinine	0.98 mg/dL	-	-	0.6-1.1 mg/dL

Parameter	Result on Admission (14/08/2025)	Follow-up GDA (Day 2)	Follow-up GDA (Day 3)	Normal Reference Range
				(Female)
Hemoglobin	13.6 g/dL	-	-	12.0-16.0 g/dL
Hematocrit	41.4 %	-	-	40-42 %

Anatomical Pathology: On August 15, 2025, a fine-needle aspiration biopsy (FNAB) of the fluctuant vulvar mass was performed to characterize the lesion and rule out malignancy.

- **Macroscopic Findings:** The procedure involved a puncture into an edematous vulvar mass measuring approximately 5 x 4 cm. This yielded yellowish, purulent fluid, confirming the clinical suspicion of an abscess
- **Microscopic Findings:** Cytological examination of the aspirate revealed a dense inflammatory infiltrate. The cellular population was composed predominantly of lymphocytes, histiocytes, and plasma cells, with a minimal presence of neutrophils. This was set against a background of amorphous material, cyst macrophages, and extensive necrotic debris. Importantly, no signs of malignancy were identified
- **Pathological Conclusion:** Benign cystic lesion with non-specific chronic inflammation

Ancillary Investigations:

- **Electrocardiogram (ECG):** The ECG demonstrated a sinus tachycardia with a heart rate of approximately 100 beats per minute. The cardiac axis, intervals, and segments were within

normal limits, with no evidence of acute ischemia, infarction, or significant arrhythmia

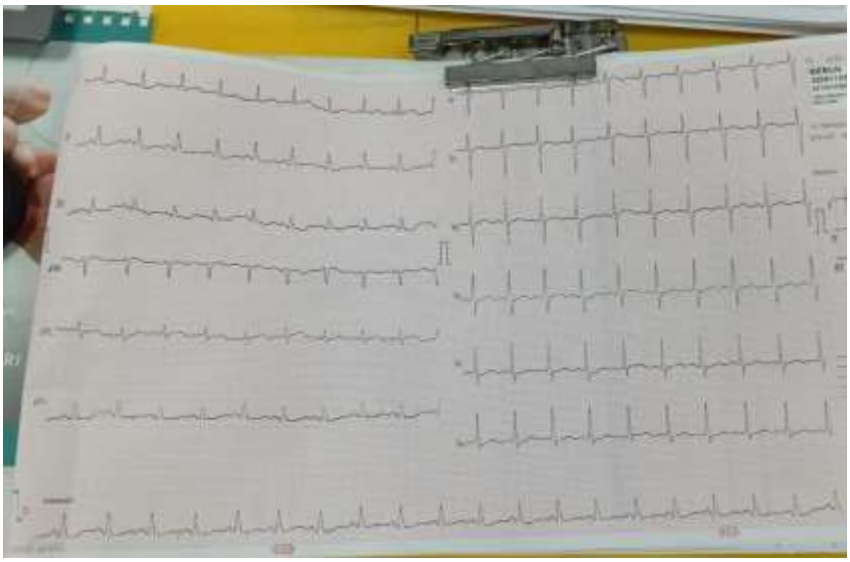

Figure 2. Electrocardiogram (ECG)

- **Chest X-Ray:** A chest radiograph (AP view) showed clear lung fields without evidence of consolidation, effusion, or pneumothorax. The cardiomediastinal silhouette was of normal size and configuration. The presence of sternal wires indicated a prior sternotomy, although this was not reported in the patient's anamnesis

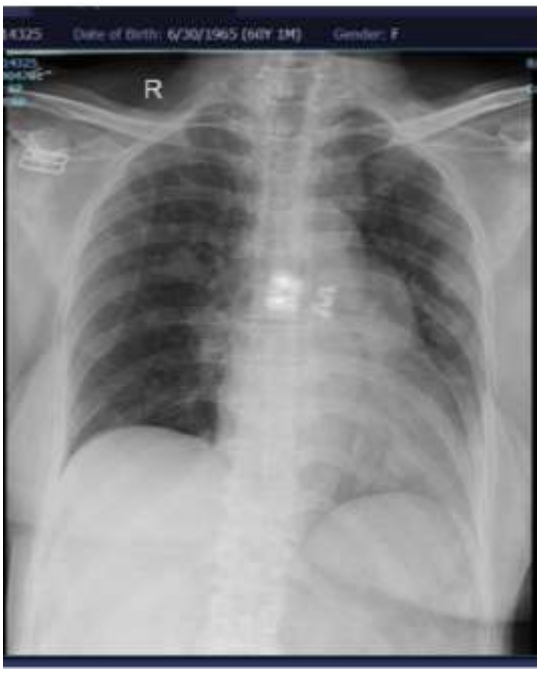

Figure 3. Chest Radiograph (AP view)

Diagnosis and Management

Based on the constellation of clinical and laboratory findings, a final diagnosis was established: **Sacral Herpes Zoster with a secondary Vulvovaginal-Perianal Abscess and newly diagnosed Type 2 Diabetes Mellitus**.

The patient's complex condition necessitated a multidisciplinary management plan involving the Departments of General Surgery, Internal Medicine, and Dermatology & Venereology (DVE).

Surgical Intervention: On August 15, 2025, the patient underwent an urgent incision and drainage of the vulvar abscess under local anesthesia. A significant amount of purulent material was evacuated, and a drain was placed to ensure continued drainage and prevent re-accumulation.

Figure 4. Post Surgical Intervention

Pharmacological Therapy: A comprehensive medication regimen was initiated:

- **Systemic Support:** Intravenous (IV) Normal Saline at 20 drops/minute for hydration.
- **Antibacterial Therapy:** IV Ceftriaxone 1 gram twice daily to provide broad-spectrum coverage for the secondary bacterial infection.
- **Antiviral Therapy:** Oral Acyclovir was started at a dose of 800 mg five times daily for a planned 7-day course to target the underlying VZV infection.
- **Analgesia:** IV Metamizole (Antrain) 1 gram three times daily for pain management.
- **Gastroprotection:** IV Ranitidine 50 mg twice daily, later switched to IV Pantoprazole 40 mg once daily.
- **Glycemic Control:** An aggressive insulin regimen was initiated to manage the severe hyperglycemia. This included subcutaneous Sansulin R (a short-acting regular insulin) 10 IU three times daily before meals, and Sansulin L (a long-acting insulin) 10 IU once daily.
- **Topical Therapy:** A meticulous local wound care protocol was implemented, consisting of

compresses with sterile saline solution three times daily, application of Fusidic acid (Fuladic) cream twice daily for topical antibacterial action, and Acyclovir ointment once daily to the herpetic lesions.

Clinical Course and Follow-up

The patient was admitted to the inpatient ward for close monitoring and continued treatment. Follow-up notes from the initial post-operative period included a diagnosis of sepsis, reflecting the severity of her systemic inflammatory response on presentation. Post-operatively, she experienced significant pain that limited her ability to sit or walk, necessitating continued IV analgesia and care with diapers.

Over the subsequent days, her clinical condition showed marked improvement. The multidisciplinary interventions were effective in controlling the infection, managing pain, and stabilizing her metabolic state. By the second post-operative day, her pain had significantly subsided, and her glycemic control had improved dramatically, with a morning random blood glucose level of 171 mg/dL. Her vital signs remained stable.

Given her stable condition and positive response to treatment, the patient was deemed ready for discharge. Her discharge plan included a transition to oral medications: Cefixime 100 mg twice daily to complete the course of antibiotics and Ibuprofen 400 mg twice daily for pain control. She was provided with her insulin regimen to continue at home, along with comprehensive education on diabetes management and self-administration of insulin. Instructions were given for ongoing topical wound care, including compresses and application of creams. Follow-up images show the progressive healing of the surgical site.

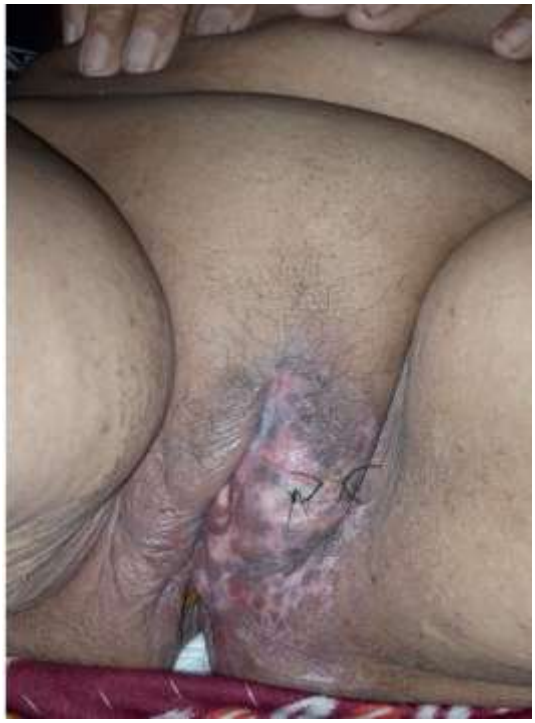

Figure 5. H+7 Post Surgical Intervention

DISCUSSION

This case presents a compelling clinical scenario where three distinct pathological processes—a viral infection (HZ), a bacterial complication (abscess), and a metabolic disorder (T2DM)—converged to create a life-threatening emergency. The discussion will explore the intricate pathophysiological links between these conditions, the diagnostic challenges posed by genital HZ, the critical importance of a synergistic, multidisciplinary management strategy, and the broader clinical implications of this presentation.

The Pathophysiological Triad: A Vicious Cycle of Diabetes, Zoster, and Abscess

The patient's presentation was not merely a coincidental occurrence of three separate illnesses but rather a vivid illustration of a complex, bidirectional pathophysiological feedback loop.

This dynamic cascade began with a state of underlying metabolic dysfunction that precipitated viral reactivation; the subsequent viral infection then triggered acute metabolic decompensation, and this combined state of immune paralysis and severe hyperglycemia created the ideal environment for a severe bacterial complication.

The process likely began with the patient's undiagnosed and untreated T2DM, which established a baseline state of chronic, low-grade inflammation and, critically, impaired CMI.[17] Chronic hyperglycemia is known to directly induce T-lymphocyte dysfunction, reducing the effectiveness of VZV-specific immune surveillance that is essential for maintaining viral latency in the sensory ganglia.[6] Studies have shown that patients with DM have significantly lower VZV-specific CMI compared to healthy individuals, which is believed to be the primary mechanism for the increased risk of HZ.[14] This pre-existing vulnerability set the stage for VZV reactivation. Once the patient's CMI waned below a critical threshold, VZV reactivated within the sacral ganglia. The virus then replicated and migrated via anterograde axonal transport to the cutaneous terminals of the sensory nerve, resulting in the characteristic dermatomal zoster eruption.[1]

The onset of the acute HZ infection, with its associated severe neuropathic pain and systemic inflammation, acted as a potent physiological stressor. Such stress is known to trigger a surge in counter-regulatory hormones like cortisol and catecholamines, which promote hepatic gluconeogenesis and glycogenolysis while simultaneously inducing peripheral insulin resistance.[21] This phenomenon of "stress hyperglycemia" was superimposed on her underlying, pre-existing insulin resistance from T2DM, leading to the severe hyperglycemia (GDS 306 mg/dL) observed on admission. This effectively "unmasked" her previously silent diabetes.[36] Furthermore, pro-inflammatory cytokines released during the acute viral infection, such as Tumor Necrosis Factor-alpha (TNF-α) and Interleukin-6 (IL-6), are known to directly contribute to insulin resistance, further fueling this vicious metabolic cycle.[38] The HZ infection, therefore, not only resulted from the diabetic state but also actively contributed to its acute and severe deterioration.

This state of acute, severe hyperglycemia delivered a second, devastating blow to an already compromised immune system. Hyperglycemia is known to acutely impair the function of neutrophils, the first line of defense against bacterial pathogens. Specifically, it disrupts chemotaxis (the ability to move toward an infection), adherence to endothelial cells, and phagocytosis (the ability to engulf and kill bacteria).[22] The ruptured vesicles of the HZ rash provided a clear portal of entry for cutaneous flora, most commonly *Staphylococcus aureus*.[3] In this environment of immune paralysis, the dysfunctional neutrophils were unable to contain the bacterial invasion. This allowed for unchecked bacterial proliferation, leading to extensive tissue necrosis (as confirmed by the pathology report) and the subsequent formation of the deep-seated vulvar abscess.[3]

Diagnostic Considerations in Genital Herpes Zoster

The initial presentation of painful genital blisters and ulcers necessitates a broad differential diagnosis, with genital herpes simplex (HSV) being the most common etiology.[14] However, in the context of genital ulcers, the *pattern* of the lesions is often a more powerful diagnostic clue than the *morphology* of an individual lesion. This case powerfully emphasizes the primacy of a thorough physical examination in differentiating HZ from its more common mimics.

Genital HSV typically presents with grouped vesicles that can be bilateral or cross the midline, is known for its frequent recurrences, and is often preceded by a milder prodrome of localized tingling or itching.[14] In stark contrast, genital HZ is defined by its strict adherence to a unilateral dermatomal boundary, a direct reflection of its origin from a single sensory ganglion.[50] The prodromal phase of HZ is characterized by a severe, burning, or stabbing neuralgia, and recurrence is rare.[1] Other potential causes of genital ulcers, such as primary syphilis (typically a single, painless, indurated chancre) or chancroid (multiple painful, purulent ulcers with ragged edges and associated suppurative lymphadenopathy), have distinct clinical features that were not consistent with this patient's presentation.[45]

Therefore, despite the morphological similarity of the vesicles to those of HSV, the

unwavering unilateral and dermatomal distribution of the rash in this patient was the pathognomonic sign. This key finding allowed for a confident clinical diagnosis of HZ, which correctly guided the immediate initiation of high-dose antiviral therapy, a crucial step in management. [75]

When a patient presents with an acute, painful, ulcerative, and edematous lesion of the vulva, the differential diagnosis must extend beyond viral etiologies. In this case, the unilateral dermatomal distribution was the key feature pointing towards HZ. However, other conditions causing vulvar inflammation and abscess formation warrant careful consideration, particularly Bartholin's abscess and hidradenitis suppurativa, as their initial presentations can overlap and create diagnostic confusion. [75]

A Bartholin's gland abscess is a primary consideration in any patient with a tender, fluctuant mass in the posterior aspect of the vulva. These glands, located on either side of the vaginal opening, can become obstructed, leading to cyst formation. If the trapped fluid becomes infected, a painful abscess develops, typically as a unilateral, erythematous, and exquisitely tender swelling. The pain from a Bartholin's abscess can be severe, often causing difficulty with walking or sitting. While it presents as a focal abscess, it lacks the characteristic vesicular prodrome and the distinct dermatomal pattern of grouped lesions seen in HZ. The diagnosis is primarily clinical, based on the location and appearance of a tender mass at the 4 or 8 o'clock position of the labia minora. [75]

Another important differential diagnosis is hidradenitis suppurativa (HS), a chronic, inflammatory skin condition that affects apocrine gland-bearing areas, including the axillae, groin, and perineum. HS is characterized by recurrent, painful, deep-seated nodules and abscesses. Over time, these can lead to the formation of sinus tracts and significant scarring. While an acute flare of HS in the vulvar region can present as a painful, swollen, and purulent lesion resembling the complication in this case, its chronicity and typical presentation with multiple lesions, comedones (blackheads), and a history of recurrence help differentiate it from a primary HZ eruption. HS does

not follow a dermatomal pattern and is not preceded by the classic neuropathic prodrome of HZ. [75]

Therefore, while the bacterial complication of a vulvar abscess was a central feature of this patient's illness, its origin was clearly linked to the primary viral eruption. The strict unilaterality, the presence of vesicles and pustules grouped in a dermatomal pattern, and the acute onset of severe neuralgia were the pivotal clinical signs that distinguished this case from other causes of vulvar abscesses. This underscores the importance of a meticulous physical examination to identify the underlying cause of a secondary bacterial infection, ensuring that both the primary trigger (VZV) and the resulting complication (abscess) are appropriately managed. [75]

This case also brings into sharp focus the critical importance of proactive and preventive medicine in high-risk populations. The entire cascade of life-threatening events experienced by this patient was initiated by the reactivation of VZV in the setting of undiagnosed diabetes. This highlights a crucial opportunity for prevention through vaccination. The recombinant zoster vaccine (RZV) has demonstrated high efficacy in preventing HZ and its complications, including PHN. Given that individuals with diabetes are at a significantly higher risk for developing HZ and suffering a more severe disease course, they are a key target demographic for vaccination. Clinical guidelines should continue to emphasize and promote HZ vaccination for all eligible adults, with a particular focus on those with underlying conditions like diabetes mellitus that impair their immune function. [76]

Furthermore, this case serves as a powerful reminder of the broader implications of glycemic control in preventing infectious complications. The patient's uncontrolled hyperglycemia created a permissive environment for both viral reactivation and subsequent bacterial superinfection by directly impairing immune cell function. Patient education is a cornerstone of diabetes management and should extend beyond metabolic control to include a thorough understanding of infection risk. Educating patients with diabetes about the importance of maintaining optimal glycemic control as a means of bolstering their immune system, coupled with counseling on the

benefits of vaccinations like the HZ vaccine, can empower them to take an active role in preventing severe, and potentially fatal, complications such as the one presented in this report.[76]

A Multidisciplinary Management Imperative

The successful outcome in this critically ill patient was not merely the result of interdepartmental cooperation but of the *synergistic* effect of three distinct yet interdependent therapeutic pillars. The failure of any single pillar would have likely led to the failure of the others and a poor clinical outcome. This case serves as a model for integrated care in complex infectious and metabolic emergencies. The patient's condition was effectively a three-pronged crisis: a viral assault, a bacterial siege, and a metabolic collapse, each requiring a specialized and timely intervention.[15]

The first pillar, managed by the Dermatology service, was **antiviral therapy**. The prompt administration of high-dose oral acyclovir (800 mg, five times daily) was critical to suppress VZV replication. This intervention is essential not only to accelerate the healing of the cutaneous rash but, more importantly, to mitigate ongoing nerve damage and reduce the substantial risk of developing debilitating PHN.[53] This risk is known to be higher and the neuralgia more severe in patients with diabetes, making early and aggressive antiviral treatment even more crucial.[8]

The second pillar was **surgical source control**, provided by the General Surgery team. The large vulvar abscess represented a contained but overwhelming septic focus that could not be sterilized by antibiotics alone. The principle of "source control" is paramount in the management of abscesses.[57] The urgent incision and drainage was a life-saving intervention that removed the nidus of infection, prevented further local tissue destruction, and was essential for halting the progression to systemic sepsis and potentially fatal necrotizing fasciitis.[18]

The third and foundational pillar was **systemic and metabolic stabilization**, overseen by the Internal Medicine service. This pillar was the bedrock upon which the other two rested. The

aggressive management of severe hyperglycemia with a multi-dose insulin regimen was not just a treatment for diabetes; it was a critical immunomodulatory therapy. By rapidly lowering blood glucose levels, the team helped to restore the function of neutrophils and T-cells, thereby enabling the patient's own immune system to more effectively combat both the viral and bacterial infections.[17] Concurrently, the administration of IV fluids corrected dehydration, while broad-spectrum IV antibiotics contained the bacteremia and prevented multi-organ failure.

The synergy between these pillars is clear: antiviral therapy would be less effective if the immune system remained paralyzed by uncontrolled hyperglycemia. Systemic antibiotics would ultimately fail without the surgical drainage of the abscess. Surgical wound healing would be severely impaired by ongoing viral-mediated tissue destruction and the catabolic state of poor glycemic control. The patient's steady recovery is a direct testament to the successful and timely integration of these three essential pillars of care.[61]

Clinical Implications and Public Health Significance

This case carries important implications beyond the management of a single patient. It highlights the role of HZ as a potential "sentinel event" that can unmask significant underlying systemic disease, particularly T2DM.[23] The incidence of undiagnosed diabetes is high among patients presenting with HZ, suggesting that the viral reactivation is often the first clinically apparent consequence of a long-standing, subclinical immune dysfunction caused by chronic hyperglycemia.[21] Therefore, a new diagnosis of HZ, especially when severe, complicated, or occurring in an atypical location, should prompt clinicians to maintain a high index of suspicion for occult T2DM and initiate appropriate metabolic screening.[23]

Furthermore, this case underscores the importance of preventative medicine. Given that individuals with DM are at a significantly increased risk for developing HZ and its complications, they represent a priority population for VZV vaccination. The availability of effective recombinant zoster vaccines offers a powerful tool to prevent the initial viral reactivation, thereby averting the

entire cascade of complications witnessed in this patient. Public health strategies and clinical guidelines should continue to emphasize the importance of HZ vaccination in at-risk groups, including the growing population of individuals with diabetes mellitus.

CONCLUSION

This case report details a rare presentation of sacral herpes zoster in a 60-year-old woman, which was severely complicated by a vulvovaginal abscess and served as the sentinel event that unmasked previously undiagnosed and uncontrolled Type 2 Diabetes Mellitus.

The key clinical lesson from this case is the vivid illustration of a dangerous pathophysiological triad. The impaired cell-mediated immunity from chronic, undiagnosed diabetes predisposes the patient to HZ reactivation. The acute inflammatory and stress response from the HZ infection then precipitates severe hyperglycemia, unmasking the metabolic disorder. Finally, this combined state of viral-induced skin barrier disruption and hyperglycemia-induced immune paralysis facilitates life-threatening secondary bacterial complications like abscess formation.

This case reinforces that a severe, atypical, or complicated presentation of HZ, particularly in an older adult, should be considered a medical red flag. It should prompt an immediate and thorough evaluation for underlying immunosuppressive conditions, with undiagnosed diabetes mellitus being among the most common. Finally, the successful management of this critically ill patient highlights the indispensable value of a rapid, integrated, and multidisciplinary therapeutic strategy that simultaneously and aggressively addresses the viral, bacterial, and metabolic components of the illness.

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