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Overlapping Neurological Insults: Case Report of Herpes Zoster in Multibacillary Leprosy with Reversal Reaction

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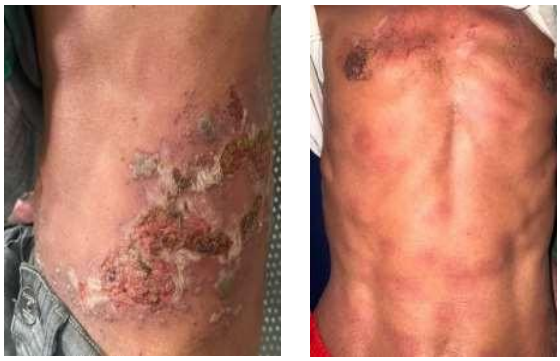
Abstract

Herpes zoster (HZ) is a reactivation of the varicella zoster virus (VZV). Low immunity, whether due to advanced age, immunosuppressants such as corticosteroids, or chronic infectious conditions such as Morbus Hansen (MH), is the primary predisposing factor for VZV reactivation. A 48-year-old man, previously diagnosed with Morbus Hansen, presented with a painful, burning, erythematous rash that began 5 days before presentation, followed by the development of grouped, fluid-filled blisters on the left waist, some of which ruptured and formed crusts. In the TZANK test, multinucleated giant cells were identified. The patient was given acyclovir, gabapentin, and mupirocin ointment. Leprosy therapy was continued, but corticosteroids were temporarily discontinued until the HZ lesions improved. After 1 week, improvements were observed in the lesions. Adequate therapy is crucial to prevent severe complications in immunocompromised patients. Clinicians should be aware of the potential for this dual disorder, particularly in immunocompromised patients, to promptly identify and manage this condition, thereby minimizing nerve damage, improving patient outcomes, and preventing disability. This case also underscores the need for careful neurological assessment and tailored strategies and therapies in patients with complex infectious neuropathies.

1. Introduction

Herpes zoster (HZ), or shingles, is a reactivation of the varicella zoster virus (VZV) [1]. This virus resides in the spinal and cranial sensory ganglia after a primary infection in childhood. Initially, a painful, erythematous, maculopapular rash develops, and the lesions subsequently progress to clustered vesicobullous lesions that eventually crust over. The hallmark of HZ is its unilateral appearance and is limited to a single dermatome. This distinguishes it from other dermatological rashes. One of the main problems with herpes zoster infection is its propensity to occur when the immune system is compromised. Low immunity, whether due to advanced age, chronic illness, immunosuppressant use, or chronic infectious conditions such as Hansen's disease (MH), is a major predisposing factor for VZV reactivation [2].

In North America, Europe, and Asia, the incidence of shingles ranges from 3 to 5 per 100,000 people. Globally, the average annual incidence ranges from 3.4 to 4.82 per 1,000 people, with rates as high as 11 per 1,000 in people aged 80 and older [3]. In the United States and Europe, the incidence of shingles is approximately 2.5 per 1,000 people in the 20- to 50-year-old age group, 5 per 1,000 in



(A) (B)

Figure 1. Clinical photographs of the patient taken on April 24, 2025. A) In the anteroposterior left abdominal region of dermatome T9 – T11, vesicles and bullae are seen in groups with an erythematous base, some containing cloudy fluid, accompanied by erosion, excoriation and blackish crusts, multiple in number, zosteriform arrangement, and unilateral distribution. B) In the anterior thoracic and abdominal regions, erythematous patches are seen, the boundaries are not clear, the edges are regular, the size is nummular-plaque, scattered discretely, multiple in number, generalized distribution.

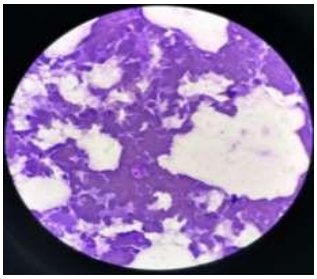


Figure 2. A Tzanck examination revealed multinucleated giant cells (black arrows).

the 51-79 age group, and 10 per 1,000 in those aged 80 and older. In Indonesia, from 2011 to 2013, HZ incidence was 2,232 cases, with a peak in the 45-64 age group (37.95%), and women had a higher incidence than men [4].

A person's immune status significantly influences the clinical course of herpes zoster and should be carefully considered in management and efforts to prevent serious complications [2]. In patients with multibacillary MH with type 2 leprosy reactions who receive Multi Drug Resistant (MDR) therapy and corticosteroid therapy, the risk of complications is higher, including the possibility of Post Herpetic Neuralgia (PHN) and more severe disseminated herpes zoster [5, 6]. The purpose of this case report is to emphasize the importance of close clinical monitoring and adequate therapy in immunocompetent patients with herpes zoster, including patients with MH, and to review the regulation of corticosteroid administration for the management of leprosy reactions in these patients.

2. Cases

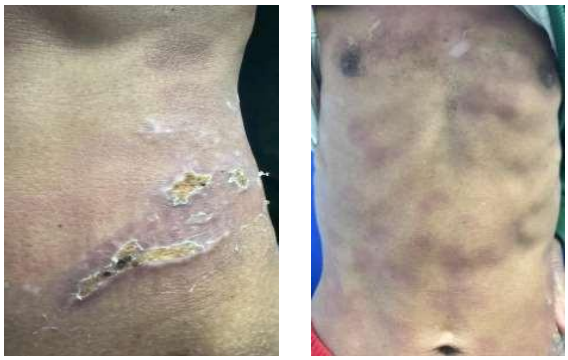
A 48-year-old male patient presented to the dermatology and venereology clinic at Zainoel Abidin General Hospital with a complaint of a painful, erythematous rash that had appeared 5 days earlier and subsequently progressed into clusters of fluid-filled blisters on the left waist (Figure 1). One day before the presentation, several of the blisters had ruptured, leaving erosions with an erythematous base. The patient experienced burning, dermatomal pain and fever approximately 2 days before the onset of the rash. He had a history of chickenpox in childhood, and no family members had experienced a similar condition.

The patient had been diagnosed with Multibacillary Hansen's Morbus in July 2024. The patient has been undergoing leprosy treatment since July 2024, with a history of Multi-Drug Therapy use in the 10th month. Several months after receiving MDT therapy, the patient experienced a reversal-type reaction that required the patient to take corticosteroids. Dermatological status examination showed that in the anteroposterior left abdominal region of dermatomes T9–T11, vesicles and bullae were grouped. In several areas, erosion and crusting were present, with multiple lesions arranged in a zosteriform pattern and a unilateral distribution along the dermatome. Tzanck's test was also performed, and the result was positive, with multinucleated giant cells identified (Figure 2).

The treatment consisted of acyclovir 800 mg tablets five times daily for 7 days, gabapentin 300 mg tablets twice daily, ibuprofen 400 mg tablets once daily, 0.9% NaCl compresses morning and evening for 10 minutes on the wound, followed by mupirocin ointment. Leprosy therapy with the multibacillary MDT regimen was continued. After one week of therapy, improvement in the lesions was observed. Corticosteroid administration was temporarily discontinued until the HZ lesions improved (Figure 3). The redness had decreased, the wounds had dried, some scabs had fallen off, and the pain had subsided (Figure 4).

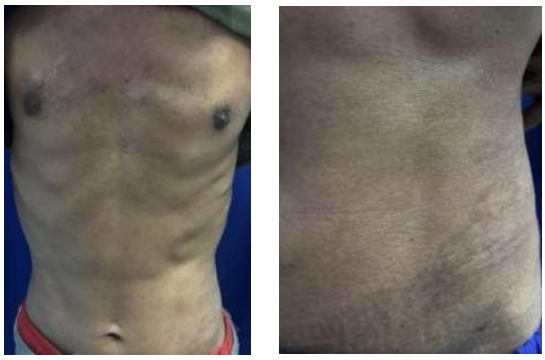
3. Discussions

Based on the history and physical examination performed on a 48-year-old male patient at the Dermatology and Venereology Clinic of Zainoel Abidin Regional General Hospital, a reddish rash with fluid-filled blisters suddenly appeared on his left waist five days ago. The patient complained of severe pain, making it difficult to perform activities. The rash initially appeared as erythematous macules and papules, which progressed to vesicles and bullae over three to four days, and then to scabs. The patient had previously been diagnosed with



(A) (B)

Figure 3. Clinical photographs of the patient taken on May 2, 2025, show improvement in the lesion. A) The wound has dried with erosion and crusting. B) The red patches have begun to disappear, with no new patches appearing.



(A) (B)

Figure 4. Clinical photographs of the patient taken on August 8, 2025. A) Improvement of the herpes zoster lesion. Only a blackish scar remains. B) Improvement of the MH lesion. The reddish patch has faded.

Morbus Hansen's type Borderline Lepromatous in July 2024. Several months later, after receiving MDT therapy, the patient experienced a reversal reaction, requiring corticosteroid therapy.

The clinical symptoms of Herpes Zoster are characterized by three stages: pre-eruptive, acute exudative, and chronic. In addition to pain, non-cutaneous symptoms such as headache, general malaise, and photophobia can also occur in the pre-eruptive phase. During the acute eruptive phase, several painful vesicles develop. The vesicles often rupture, ulcerate, and eventually dry out. This is the most contagious stage. Pain is often severe and unresponsive to non-steroidal pain medications. The acute eruptive phase can last 2–4 weeks [7]. Pain may persist longer. Chronic herpes zoster infection is characterized by severe pain that persists for>4 weeks. Patients experience dysesthesia, paresthesia, and sometimes a shock-like sensation. The pain is disabling and can last for several months. In these patients, the lesions are immunocompetent, so the lesions usually crust over within seven to ten days [8, 9].

The patient had a history of chickenpox, which could lead to varicella-zoster virus reactivation. After contracting chickenpox, the virus establishes a lifelong latent infection in neurons of the ganglia. When the immune response and specific antibody titers to the varicella-zoster virus decline, the latent varicella-zoster virus particles reactivate, causing a localized skin rash within a dermatome. This reactivation is triggered by risk factors, including decreased cellular immunity to VZV, which can be caused by immunosuppressant drugs such as corticosteroids. When VZV-specific T-cell-mediated immunity falls below a critical threshold, the virus reactivates and is no longer contained, leading to viral multiplication and spread within the ganglion and neuronal necrosis and severe inflammation, a process accompanied by severe neuropathic pain [10].

Hansen's disease is a chronic bacterial infection caused by Mycobacterium leprae, characterized by severe skin lesions and nerve damage [11, 12]. Multibacillary leprosy (MB) consists of three spectrum types, namely lepromatous (LL), borderline lepromatous (BL), and midborderline (BB), which are distinguished based on clinical, bacteriological, and immunological features [13]. The bacilli spread widely to the skin, nerves, and other organs, but not as much as in the pure lepromatous type [14].

In the lepromatous form, there is a diminished cellular immune response, leading to uncontrolled bacterial infections and local and systemic immunosuppression [10]. This condition involves immune control of latent viruses, such as varicella-zoster, that reside in nerve ganglia. This diminished cellular immunity allows the dormant varicella-zoster virus to reactivate, leading to herpes zoster. Furthermore, leprosy reactions, which involve increased pro-inflammatory cytokines such as TNF-α and neuroinflammation, can also trigger nerve damage and reduce the body's ability to control the virus [12].

In these patients, herpes zoster treatment is administered while ongoing treatment for Hansen's disease is continued. The herpes zoster medications administered are acyclovir, gabapentin, and ibuprofen. Acyclovir is an antiviral agent that inhibits viral DNA polymerase, thereby causing premature termination of viral DNA synthesis. Gabapentin is given to reduce neuropathic pain in patients caused by damage and death of sensory neurons due to herpes zoster virus infection. Ibuprofen, a nonsteroidal anti-inflammatory drug (NSAID), also helps relieve pain [15].

The effectiveness of the combination of gabapentin and ibuprofen in managing shingles pain or preventing PHN

has not been proven in clinical studies. However, given their distinct mechanisms of action, gabapentin acts on the central nervous system to reduce neuropathic pain, whereas ibuprofen reduces inflammation; combining them may provide additional benefits in managing acute pain. However, further research is needed to confirm the safety and effectiveness of this combination [10]. This patient's ongoing treatment for Hansen's disease is being continued. The patient previously received a multibacillary Hansen's disease treatment regimen, consisting of rifampin, clofazimine, and dapsone, which will be taken for 12 months [10, 16].

This patient also experienced a reversal reaction, or type 1 reaction, several months after starting Hansen's disease treatment. This reaction can occur during therapy or after its completion. This reaction typically occurs in patients with Borderline Leprosy (BB) and Black Leprosy (BL), characterized by old or new lesions that become more inflamed, erythematous, and edematous. Nerve pain, tenderness, paresthesias, decreased nerve function, fever, discomfort, joint pain, and ulcerated skin lesions may also be present. Patients with reversal reactions are often treated with corticosteroid therapy [10, 17]. The use of corticosteroids to treat leprosy reactions can also suppress the immune system, increasing the risk of viral reactivation. Thus, decreased cellular immunity and changes in inflammatory cytokines are the main factors that activate varicella-zoster virus and trigger herpes zoster in patients with leprosy [13].

4. Conclusions

This case illustrates the rare but significant overlapping neurological complications of a leprosy reaction and a concurrent herpes zoster infection. This overlap not only presents a diagnostic challenge for clinicians but also necessitates a comprehensive treatment approach to address the patient's pain. Accurate diagnosis and adequate therapy can minimize nerve damage, improve patient outcomes, and prevent disability. This case also underscores the need for careful neurological assessment and treatment in patients with complex infectious neuropathies.

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