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CASE REPORT

Clinical Management of Herpes Zoster Complicated by MRSA Infection

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Abstract: A 59-year-old male presented with a 10-day history of erythematous vesicular lesions on the left chest and shoulder, accompanied by pain in the back. Clinical examination revealed grouped vesicles with some ulceration and exudate, leading to a diagnosis of herpes zoster. After a clear diagnosis, antiviral treatment with valacyclovir and pain relief with pregabalin was administered. Apply povidone-iodine and normal saline (in a 1:10 ratio) externally, once a day, for 30 minutes each time. One week later, the lesions developed ulcers and crusts, with increased exudate; cultures yielded a significant growth of Methicillin-resistant Staphylococcus aureus(MRSA). Based on the drug sensitivity results, cefuroxime combined with levofloxacin was administered for anti-infection treatment. Following two weeks of treatment, the patient showed significant clinical improvement. The patient's rashes have significantly subsided, the ulcers have healed and scabbed over, and the exudation has noticeably decreased. Herpes zoster combined with MRSA infection makes the treatment more complicated. This case illustrates the complexities of managing herpes zoster with MRSA and highlights the importance of comprehensive diagnostic and treatment strategies.

Keywords: herpes zoster, MRSA

Introduction

Herpes zoster, commonly known as shingles, is a viral infection caused by the reactivation of the varicella-zoster virus (VZV), which remains dormant in the sensory ganglia following primary infection with varicella (chickenpox). The incidence of herpes zoster increases with age, particularly among individuals over 50 years, and is further exacerbated by immunocompromised states, such as those resulting from chronic diseases or immunosuppressive therapies.[1] The correlation between advanced age and susceptibility to VZV reactivation is evident in US epidemiology, where adults aged 50 years and above constitute a substantial proportion-about 50%-of the approximately one million yearly herpes zoster cases, underscoring an age-related decline in immunity.[2] Clinically, herpes zoster is characterized by a painful dermatomal rash, which typically presents as vesicular lesions that may rupture and crust over, often accompanied by significant neuropathic pain known as postherpetic neuralgia. The relationship between zoster infection and destruction of neurons and satellite cells has been well established, with neurologic damagebeginningeven before the characteristic zoster rash appears.[3,4] In otherwise healthy elderly individuals, postherpetic neuralgia-the predominant sequela of herpes zoster-results in debilitating pain and a marked decline in quality of life.[5–7] Involvement of the trigeminal nerve's first division by herpes zoster, known as herpes zoster ophthalmicus (HZO), poses a risk of long-term visual sequelae stemming from inflammation or neural damage.[8] Complications can include secondary bacterial infections, such as those caused by Staphylococcus aureus.Herpes zoster, commonly known as shingles, is a viral infection that arises from the reactivation of the latent varicella-zoster virus and is defined by an outbreak of fluid-filled vesicles on the skin. The fragility of these vesicles and their tendency to rupture can breach the skin barrier, creating a portal of entry for bacteria and predisposing the individual to secondary bacterial infections.[9] Once the vesicles rupture, the underlying skin is no

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longer protected. This allows bacteria such as Staphylococcus aureus to colonize the area, multiply, and cause local or systemic infections.[10]

Furthermore, herpes zoster infection itself can lead to temporary immune deficiency, further weakening the body's ability to defend against bacteria.[11] This decline in immune function may be related to the direct effect of the virus on immune cells, making patients more prone to secondary complications such as bacterial infections of the skin.

The diagnosis of herpes zoster is primarily clinical, based on the characteristic rash and associated symptoms. However, laboratory investigations, including complete blood counts, inflammatory markers, and cultures, are essential to rule out other conditions and to assess for potential complications, such as bacterial superinfection. The management of herpes zoster typically involves antiviral therapy, analgesics, and, in some cases, corticosteroids to reduce inflammation and pain.[12] Early intervention is crucial to mitigate the risk of complications and improve patient outcomes.

A study reported a case of herpes zoster infection complicated by MRSA infection, leading to endocardial myocardial abscess and ultimately resulting in the patient's death. Immunosuppression increases the risk of such infections, making early empirical anti-MRSA treatment crucial.[13] When herpes zoster is accompanied by bacterial infection, sensitive antibiotics should be selected based on the results of drug sensitivity tests for treatment.

Case Presentation

A 59-year-old male presented with a 10-day history of erythematous vesicles on the left side of the chest and shoulder neck region. On examination, erythema was noted on the left chest and shoulder neck area, with clustered vesicles on the surface, some of which were ruptured and oozing (Figure 1). The patient also reported accompanying pain in the shoulder and back. The patient was diagnosed with herpes zoster. All laboratory tests (Comprehensive tumor markers, Syphilis, HIV tests and so on) returned within normal limits without significant abnormalities. The patient's blood sugar and glycated hemoglobin levels were both within normal ranges. The following treatment regimen was initiated:

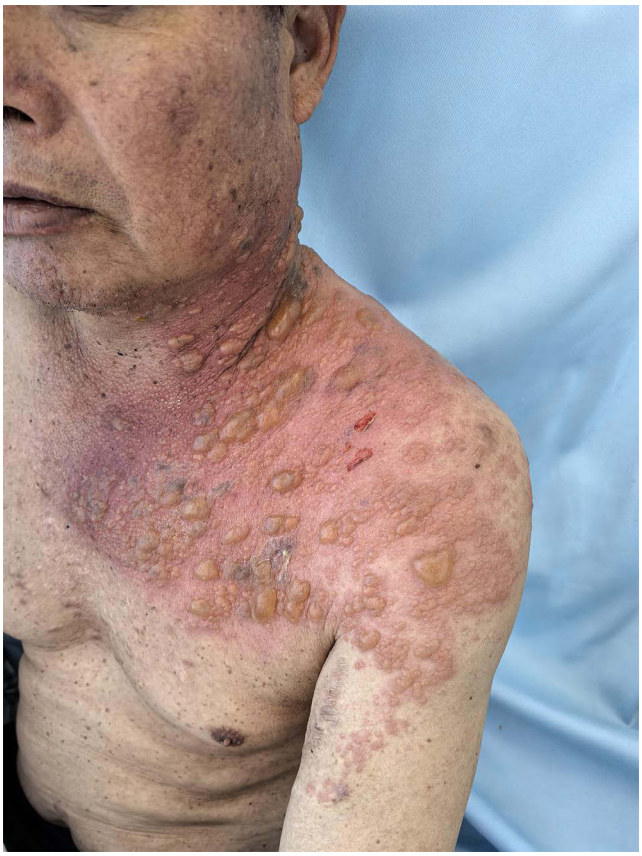

Figure 1 Clinical presentation of skin lesions (before the treatment).

bromhexine and galantamine injection, oral acyclovir hydrochloride, oral pregabalin and diclofenac sodium delayed-release capsules.Apply povidone-iodine and normal saline (in a 1:10 ratio) externally, once a day, for 30 minutes each time. One week later, the lesions progressed to erosion, ulceration, and crusting, with increased secretion. Upon pressing the crusts, yellow exudate was observed (Figure 2). Wound secretion culture found a large growth of MRSA and no fungal growth (Figure 3). The results of the drug sensitivity test showed MRSA are sensitive to cefotaxime and levofloxacin. The patient was treated with cefotaxime-dextrose and levofloxacin-sodium chloride for infection control. After two weeks of treatment, the patient showed significant improvement.The patient's rashes have significantly subsided, the ulcers have healed and scabbed over, and the exudation has noticeably decreased. Meanwhile, the patient's neuralgia has improved compared to before. After one month of follow-up, the patient did not experience post-zoster neuralgia.

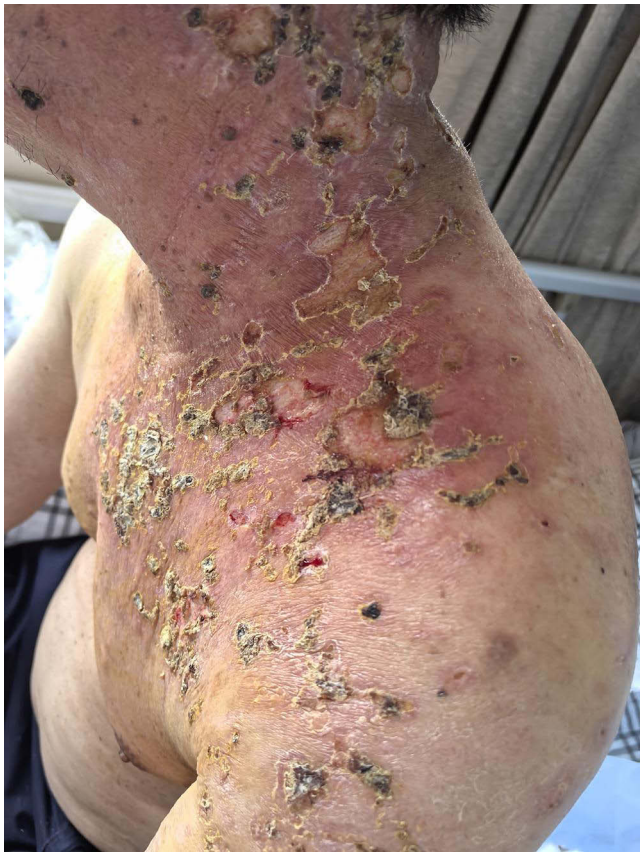

Figure 2 Clinical presentation of skin lesions (suffering from infection).

Figure 3 Auxiliary examination (culture result).

Discussion

The case presented is significant due to the patient's age, the atypical presentation of herpes zoster, and the resultant secondary infection with MRSA. Herpes zoster, or shingles, is caused by the reactivation of the varicella-zoster virus, typically presenting as a painful vesicular rash in a dermatomal distribution. In our patient, the localized rash on the left thoracic region and shoulder, accompanied by pain, aligns with classical presentations of herpes zoster. However, the rapid progression to ulceration and secondary bacterial infection highlights the potential for complications in this condition. Herpes zoster combined with MRSA infection makes the treatment more complicated. Older patients with herpes zoster, especially those with weakened immune systems due to aging, are more prone to severe courses and complications. Among them, secondary bacterial infections are rare but possible complications of herpes zoster, especially in the blister areas, which may lead to aggravated skin infections and prolonged recovery time. Therefore, it is crucial to be vigilant of signs of bacterial infections (such as redness, pus formation, or fever) and to carry out early intervention, including using symptomatic treatments (such as reducing fever and avoiding bacterial infections) and, when necessary, using antiviral drugs to alleviate symptoms and prevent the progression of complications. Early intervention helps shorten the course of the disease.

Current literature reports that herpes zoster can lead to various complications, including postherpetic neuralgia and secondary infections, particularly in immunocompromised individuals or those with pre-existing conditions that may predispose them to infections.[10] Early identification and early treatment can reduce the risk of infection worsening in patients and shorten their illness duration. In this case, the patient's extensive ulceration and subsequent culture results revealing MRSA underscore the need for vigilant monitoring and appropriate management strategies in patients with herpes zoster, particularly in older adults or those with compromised immune systems.[14]

The treatment plan employed a multifaceted approach combining antiviral therapy, analgesics, and topical antiseptics, which is consistent with current recommendations for herpes zoster management. The inclusion of antibiotics to address the secondary MRSA infection was imperative, given the potential morbidity associated with such infections. This case reinforces the need for healthcare providers to remain vigilant for secondary infections in patients presenting with herpes zoster, particularly when lesions exhibit signs of worsening or non-healing.

It is important to acknowledge the limitations of this study. As a single-case report, our findings are inherently limited in their generalizability. The outcomes and observations described herein are specific to the unique clinical presentation and individual characteristics of this patient.

This case illustrates the complexities that can arise in the management of herpes zoster and highlights the necessity for a comprehensive approach that considers not only the immediate symptoms but also the broader implications of potential complications and associated conditions. The insights gained here can guide future clinical practice, ensuring that similar cases are managed with a heightened awareness of the risks involved and the need for follow-up.

The unique aspects of our case-specifically, the rapid progression to severe ulceration in an otherwise immunocompetent, middle-aged adult-highlight a potentially severe and underrecognized complication of herpes zoster. This atypical presentation underscores the critical need for heightened clinical vigilance. We strongly advocate for the routine obtainment of wound cultures and susceptibility testing in all herpes zoster cases exhibiting signs of secondary bacterial infection, such as purulent drainage, worsening cellulitis, or failure to improve with standard antiviral therapy. This practice is essential to guide targeted antimicrobial therapy. Furthermore, empiric antibiotic selection, when indicated, should be informed by local antibiograms and a high index of suspicion for drug-resistant organisms, such as MRSA, to prevent treatment failure and disease progression.

Ultimately, this case serves as a compelling call to action. The rising threat of antimicrobial resistance necessitates that our management of common viral infections like herpes zoster evolves. Future prospective, multi-center studies are urgently needed to establish evidence-based clinical guidelines for the diagnosis and management of drug-resistant bacterial superinfections in herpes zoster. Such research will be pivotal in optimizing patient outcomes in an era of increasing antimicrobial resistance.

Ethics Approval

The publication of case report does not require ethical approval. We confirm that no institutional approval was required for publishing the case details.

Consent for Publication

Informed consent was obtained for the publication of the case. This article adheres to the applicable CAse REport (CARE) guidelines.

Informed Consent for Publication

The patient had signed informed consent and provided consent for the publication of the case details and any accompanying images.

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Disclosure

The authors report no conflicts of interest in this work.

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