American Journal of Case Reports

e-ISSN 1941-5923
© Am J Case Rep, 2026; 27: e954654
DOI: 10.12659/AJCR.954654

Received: 2026.06.29
Accepted: 2026.10.30
Available online: 2026.11.20
Published: 2026.XX.XX

A 64-Year-Old Diabetic Man Presenting With a Painful Vesicular Rash and Left Shoulder Weakness Due to Segmental Zoster Paresis: A Case Report

Authors' Contribution:
Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

ABCDEF 1 Wan Mohamad Izham Wan Ismail
ACDE 1 Khasnur Abd Malek
CDE 2 Shalini Bhaskar
DE 3 Natasha Mohd Nasran

1 Department of Primary Care Medicine, Universiti Teknologi MARA, Cawangan Selangor, Sungai Buloh Campus, Sungai Buloh, Selangor, Malaysia
2 Department of Medicine, Faculty of Medicine, Universiti Teknologi MARA, Cawangan Selangor, Sungai Buloh Campus, Sungai Buloh, Selangor, Malaysia
3 Department of Radiology, Hospital Al-Sultan Abdullah, Universiti Teknologi MARA, Cawangan Selangor, Puncak Alam, Selangor, Malaysia

Corresponding Author: Khasnur Abd Malek, Department of Primary Care Medicine, Faculty of Medicine, Universiti Teknologi MARA (UiTM), 47000, Sungai Buloh, Selangor, Malaysia
e-mail: drkhasnur@uitm.edu.my, drkhasnurabdmalek@gmail.com, melkay03@gmail.com
Financial support: None declared
Conflict of interest: None declared

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Patient: Male, 64-year-old
Final Diagnosis: Segmental zoster paresis secondary to herpes zoster
Symptoms: Clusters of fluid-filled vesicles • left arm weakness • stinging pain
Clinical Procedure: Magnetic resonance imaging of the brachial plexus • nerve conduction study
Specialty: Family Medicine • Neurology

Objective: Unusual clinical course

Background: Segmental zoster paresis (SZP) is a rare neurological complication resulting from the reactivation of the varicella-zoster virus, leading to lower motor neuron weakness within the affected dermatome. Although uncommon, it can mimic more ominous pathologies, such as brachial plexopathy or malignancy. We report this case to highlight the clinical presentation and diagnostic approach necessary for accurate clinical diagnosis and follow-up evaluation.

Case Report: A 64-year-old man presented with a painful vesicular rash localized to the C5-C6 dermatomes, accompanied by acute-onset left shoulder weakness. Physical examination revealed motor deficits predominantly within the C5 distribution, initially raising strong clinical suspicion for focal brachial plexopathy. The patient was managed with a multimodal regimen comprising prednisolone, gabapentin, vitamin B complex supplementation, and physiotherapy. Antiviral therapy was not given, as he presented 10 days after rash onset. While early intervention relied strictly on clinical assessment, diagnostic confirmation was achieved later. Follow-up magnetic resonance imaging, nerve conduction studies, and electromyography performed 4 months after onset successfully localized the lesion and supported the diagnosis of SZP. Furthermore, these delayed evaluations effectively excluded competing structural or compressive causes.

Conclusions: This case demonstrates that while the initial identification of SZP relies on high clinical suspicion to differentiate it from acute plexopathy, delayed electrodiagnostic and imaging studies were useful during follow-up in this patient. Rather than necessitating urgent testing during the acute phase, subsequent electrodiagnostic evaluation supported lesion localization and diagnostic confirmation.

Keywords: Brachial Plexus • Brachial Plexus Neuropathies • Herpes Zoster
Full-text PDF: https://www.amjcaserep.com/abstract/index/idArt/954654

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Introduction

Segmental zoster paresis (SZP) is a rare neurological complication of herpes zoster (HZ). Epidemiologically, SZP is an uncommon manifestation, affecting approximately 0.5% to 5% of patients with HZ [1,2]. The condition predominantly affects older adults, immunocompromised individuals, and individuals with diabetes mellitus. In these populations, diabetes is a well-documented predisposing factor for heightened neuroinflammation. This inflammatory process extends beyond the dorsal root ganglion to involve the ventral roots or anterior horn cells, ultimately resulting in motor deficits [3-5]. Additionally, in patients with diabetes, distinguishing SZP from structural or metabolic mimics like diabetic radiculoplexopathy presents a distinct clinical challenge due to overlapping presentations of acute, asymmetric limb weakness [6].

Clinically, focal motor weakness of SZP typically manifests 1 to 4 weeks following the onset of the cutaneous eruption, although concurrent or preceding motor deficits have also been described [7]. The weakness is characteristically asymmetric and segmental. It most frequently involves the upper extremities (C5-C7 myotomes), closely mimicking a structural brachial plexopathy [8]. Clinical presentation classically reveals lower motor neuron signs restricted to the involved segments. These signs include focal muscle weakness, hypotonia, and diminished or absent deep tendon reflexes [3].

Therapeutic strategies for acute HZ reactivation and subsequent SZP usually involve a timely, multimodal approach. In the acute cutaneous phase, first-line antiviral therapy is administered for 7 days, typically using oral acyclovir, valacyclovir or famciclovir. To achieve optimal antiviral effectiveness, treatment should be initiated within 72 hours of the onset of the vesicular rash to arrest viral replication and mitigate severe nerve damage [9]. Once motor weakness manifests, management shifts toward mitigating neural inflammation and controlling debilitating neuropathic pain. This phase frequently employs systemic corticosteroids such as prednisolone alongside gabapentinoids [10,11]. Supportive interventions can also contribute to long-term functional recovery. These include neurotropic vitamin B complex supplementation to facilitate nerve regeneration and early, targeted physiotherapy to prevent disuse muscle atrophy while maximizing myotomal rehabilitation [10].

We report the case of a 64-year-old man with type 2 diabetes mellitus who presented with typical HZ complicated by acute-onset unilateral left shoulder weakness. As a confirmatory case contribution, this report reinforces the clinical recognition of SZP when a dermatomal rash and focal weakness coexist. Furthermore, it illustrates the diagnostic differentiation of SZP from structural plexopathy in a diabetic patient and highlights the interpretive value of delayed magnetic resonance imaging (MRI) and electrodiagnostic findings in a real-world presentation.

Case Report

A 64-year-old man presented with sudden-onset left upper limb weakness that had been present for 10 days. The patient reported that he was unable to abduct or elevate his left arm. The weakness coincided with the appearance of clusters of fluid-filled vesicles over the left upper arm, accompanied by sharp, stinging pain rated as 8/10 on the visual analog scale. He denied any associated numbness, tingling, preceding trauma, or recent vaccinations. There was no fever or constitutional symptoms, and motor function remained intact in the right upper limb and both lower limbs. The patient works as a security officer. He attends regular follow-up for non-communicable diseases, with no previous limb weakness; however, no formal neurological examination had been documented before symptom onset.

His medical history was significant for type 2 diabetes mellitus and ischemic heart disease, status post percutaneous coronary intervention 10 years prior. He also had a 20 pack-year smoking history but had quit 11 years earlier. His regular medications included bisoprolol, sacubitril/valsartan, rosuvastatin, metformin, empagliflozin, and insulin detemir. None of these medications is immunosuppressive, and apart from diabetes mellitus, no other cause of immunosuppression was identified.

On examination, the patient was alert, afebrile, and showed stable vital signs with a normal gait. Cutaneous assessment revealed multiple clusters of erythematous lesions in varying stages of evolution, ranging from exudative ruptured vesicles to crusted scabs (**Figure 1A**). The rash originated over the posterior aspect of the left shoulder and extended anteriorly across the upper arm to the antecubital fossa, corresponding to the C5-C6 dermatomal distribution (**Figure 1B**).

Neurological examination of the left upper limb demonstrated normal muscle tone but significant focal weakness. Muscle strength was severely reduced during shoulder abduction and adduction, both graded at Medical Research Council (MRC) grade 1/5. In contrast, elbow flexion, elbow extension, wrist flexion, wrist extension, and handgrip were all preserved at grade 4/5. The left biceps reflex was diminished, whereas the triceps and brachioradialis reflexes were entirely preserved. Sensory examination and cranial nerve assessment were unremarkable.

Initial laboratory investigations, as shown in **Table 1**, including a full blood count, erythrocyte sedimentation rate, serum electrolytes, renal function, liver function, and thyroid

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Figure 1. Clinical photographs of the left upper limb showing the herpes zoster rash and subsequent muscle wasting. (**A**) Multiple clusters of erythematous lesions over the posterior aspect of the left shoulder at presentation. (**B**) Lesions extending anteriorly across the upper arm to the antecubital fossa, corresponding to the fifth and sixth cervical (C5-C6) dermatomal distribution. (**C**) Post-inflammatory hypopigmentation and hyperpigmentation along the C5-C6 dermatomes 3 months after treatment. (**D**) Wasting of the left supraspinatus, infraspinatus, and deltoid muscles at 3 months.

function tests, were all within normal limits. Glycemic control was suboptimal, indicated by a glycated hemoglobin A1c (HbA1c) level of 7.0%. The C-reactive protein was mildly elevated at 8.3 mg/L. Tumor markers, including alpha fetoprotein, cancer antigen 125, carcinoembryonic antigen, and prostate-specific antigen, were within normal ranges, thereby reducing the likelihood of paraneoplastic etiologies. Screening for other infections 2 weeks after onset was negative for hepatitis B surface antigen (HBsAg), hepatitis C antibody (anti-HCV), human immunodeficiency virus (HIV) 1/2 antigen/antibody, and *Treponema pallidum* antibody (**Table 1**). Varicella-zoster virus (VZV) immunoglobulin G (IgG) and immunoglobulin M (IgM) serology were not performed, as the diagnosis of herpes zoster was made clinically based on the painful, unilateral dermatomal vesicular eruption. Serial laboratory measurements after treatment initiation were not available. A nerve conduction study was not performed during the acute phase of the illness.

The patient was started on oral prednisolone 30 mg daily for 2 weeks, followed by a gradual taper over 6 weeks. He was also prescribed gabapentin 300 mg twice daily for neuropathic pain and vitamin B complex supplementation. An intensive physiotherapy program was instituted immediately, focusing on muscle strengthening and functional rehabilitation. Antiviral therapy was not administered, as he presented 10 days after rash onset, beyond the 72-hour window in which antiviral treatment is most effective.

Magnetic resonance imaging (MRI) of the brachial plexus was performed 2 months after symptom onset. The scans showed preserved and symmetric signal intensity throughout the roots, trunks, divisions, cords, and branches of the brachial plexus. However, indirect evidence of nerve injury was present, characterized by distinct muscle atrophy and fatty infiltration in the left shoulder girdle muscles compared with the right side. These structural changes involved the deltoid, trapezius, serratus, subscapularis, infraspinatus, and supraspinatus muscles. Elements of muscle edema were also observed, evidenced by a hyperintense signal on T2-weighted (T2W) and short tau inversion recovery (STIR) sequences and mild post-contrast enhancement. Overall, these neuroimaging features (**Figure 2**) were consistent with the late subacute to chronic phase of a muscle denervation injury.

By the 3-month follow-up, the cutaneous lesions had transitioned to post-inflammatory hypopigmentation and hyperpigmentation (**Figure 1C**). However, prominent muscle wasting was noted over the left scapular region, specifically involving the supraspinatus and infraspinatus muscles with partial deltoid involvement (**Figure 1D**). Motor power remained stagnant, with shoulder abduction and adduction at grade 1/5, while distal segments (elbow flexion, elbow extension, wrist flexion, wrist extension, and handgrip) remained stable at grade 4/5.

A nerve conduction study was performed 4 months after symptom onset. The left axillary compound muscle action potential (CMAP) recorded from the deltoid muscle showed a reduction of more than 50% compared to the unaffected right side (**Figure 3**). Subsequent needle electromyography evaluated the left deltoid, supraspinatus, rhomboid major, and brachioradialis muscles. The evaluation revealed polyphasic changes and increased amplitudes of the motor unit action potentials (MUAPs) without spontaneous activity. These combined electrodiagnostic findings support a diagnosis of segmental zoster paresis with focal motor involvement localized to the C5-C6 nerve roots.

Discussion

Segmental zoster paresis (SZP) is a rare but potentially disabling neurological complication of herpes zoster (HZ). When

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Table 1. Laboratory investigations at presentation and 2 weeks after onset.

Analyte	Results	References
At Onset		
Hemoglobin, g/dL	14.3	13-17
White blood cell, × 10^9/L	7.90	4-10
Neutrophil,%	71.6	40-80
Lymphocyte,%	17.8	20-40
Monocyte,%	8.4	2-10
Eosinophil,%	2.0	1-6
Basophil,%	0.0	0-2
Platelet, × 10^9/L	234	150-410
Erythrocyte sedimentation rate (ESR), mm/hr	4	0-15
Urea, mmol/L	3.50	2.76-8.07
Sodium, mmol/L	140	136-145
Potassium, mmol/L	3.9	3.5-5.1
Creatinine, umol/L	88	62-106
eGFR, ml/min/1.73 m^2	81	
Corrected cCalcium, mmol/L	2.32	2.2-2.55
Phosphate, mmol/L	1.24	0.8-1.45
Chloride, mmol/L	101	98-107
Magnesium, mmol/L	0.84	0.66-0.99
Albumin, g/L	45.4	35-52
Free T4, pmol/L	16.7	12-22
TSH, mIU/L	0.302	0.27-4.2
C-reactive protein, mg/L	**8.3**	< 5
HbA1c,%	**7.0%**	≤ 5.6
2 weeks after onset		
Hepatitis C antibody (anti-HCV)	Non-reactive	
Hepatitis B surface antigen (HBsAg)	Non-reactive	
HIV Ag/Ab combo assay (anti-HIV)	HIV 1/2 Negative	
Treponema pallidum antibody test (Syphilis screening)	Negative	
Alpha-1-Fetoprotein, ng/mL	2.0	≤ 9
Cancer AG 125 (CA 125), U/mL	10.9	≤ 35
Carcinoembryonic AG (CEA), ng/mL	2.1	≤ 3
Prostate specific antigen (total), ng/mL	0.840	≤ 4

Abbreviations: ESR, erythrocyte sedimentation rate; eGFR, estimated glomerular filtration rate; Free T4, free thyroxine; TSH, thyroid-stimulating hormone; HbA1c, glycated hemoglobin A1c; Anti-HCV, hepatitis C virus antibody; HBsAg, hepatitis B surface antigen; HIV Ag/Ab, human immunodeficiency virus antigen/antibody; CA 125, cancer antigen 125; CEA, carcinoembryonic antigen.

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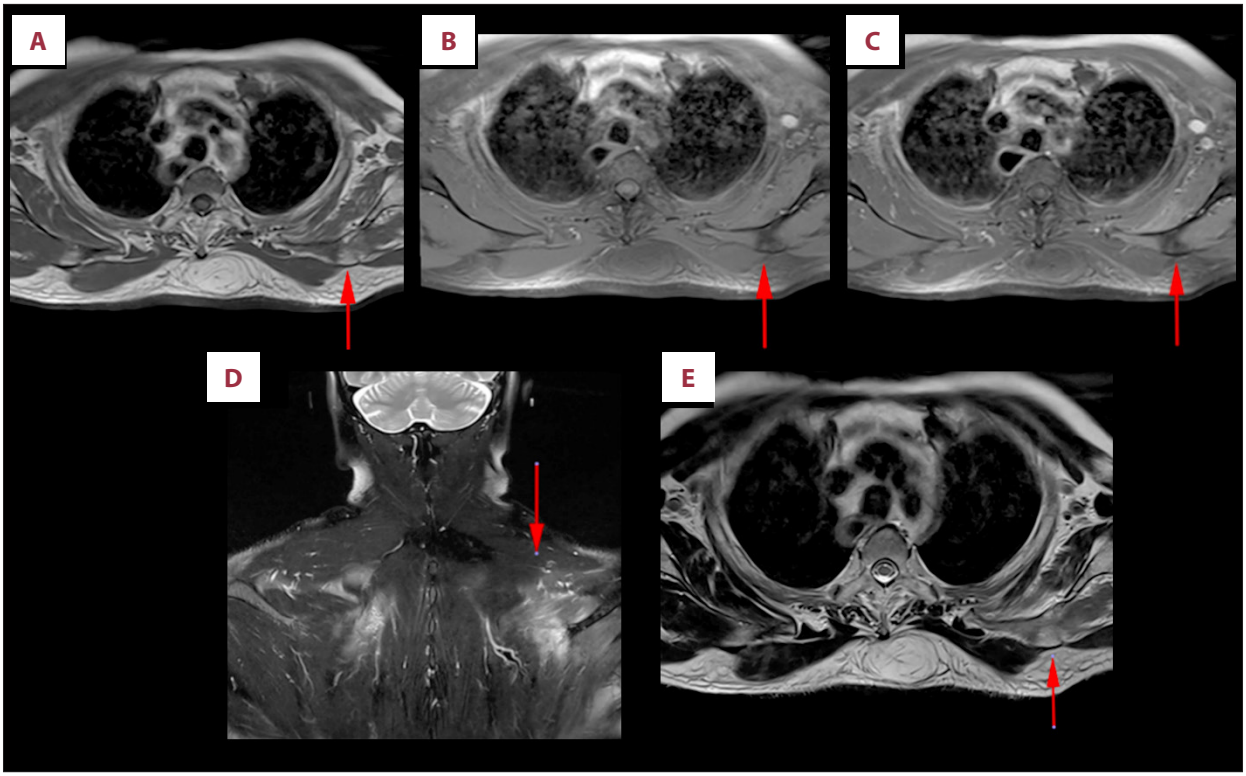


Figure 2. Magnetic resonance imaging (MRI) of the brachial plexus and shoulder girdle two months after symptom onset, showing denervation changes in the left shoulder girdle muscles. (A) Axial T1-weighted image showing loss of muscle bulk in the left shoulder girdle muscles, with areas of high signal intensity suggesting fatty replacement. (B) Pre-contrast and (C) post-contrast axial fat-suppressed T1-weighted images showing mild enhancement of the left shoulder girdle muscles, suggesting oedema, with hypointense areas suggesting fatty infiltration. (D) Coronal short tau inversion recovery (STIR) and (E) axial T2-weighted images showing high signal intensity within the left shoulder girdle muscles, suggesting oedema.

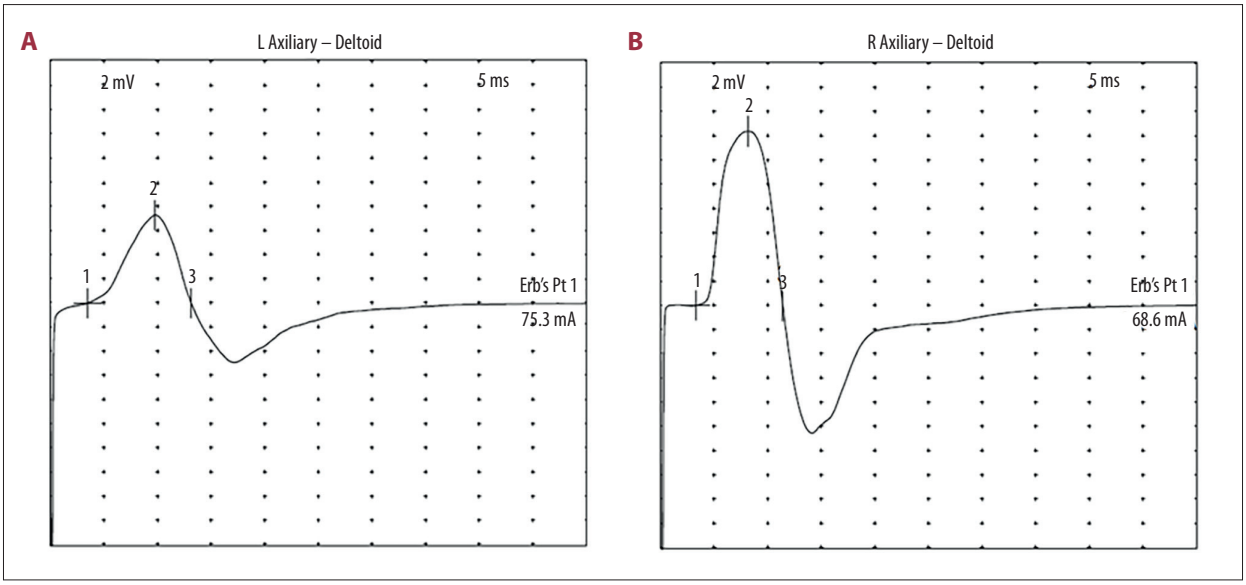


Figure 3. Motor nerve conduction study of the bilateral axillary nerves four months after symptom onset. (A) Stimulation of the left axillary nerve, recording from the deltoid muscle, shows a reduction in compound muscle action potential (CMAP) amplitude of more than 50% compared with (B) the unaffected right axillary nerve.

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muscle weakness occurs, the upper limb is the most commonly affected region. Reported rates of motor impairment range from 0.5% to 5% in HZ cases, resulting in clinically significant limb paresis in approximately 3% to 5% of those instances [1,6]. Reactivation of latent varicella zoster virus prompts viral migration from the dorsal root ganglion to the ventral horn cells and anterior motor roots. This migration triggers localized neuroinflammation, axonal degeneration, and intraneural edema within the brachial plexus [1,3]. This process produces a characteristic "patchy" pattern of neuronal injury, accounting for the variable and often incomplete motor deficits observed within a single myotomal distribution [3].

Motor weakness in SZP typically develops days to weeks after the onset of the cutaneous rash, but it may occur concurrently or, less commonly, precede it [1,11,12]. Delayed muscle weakness is more frequently observed in immunocompromised individuals [4], underscoring the need for prolonged clinical vigilance in high-risk populations. In our case, the close temporal association between the vesicular rash and abrupt motor deficit strongly suggested pathology beyond uncomplicated cutaneous shingles. This relationship correctly directed attention toward a diagnosis of SZP. Importantly, this case highlights that a dermatomal rash accompanied by acute focal shoulder weakness can closely resemble a structural brachial plexopathy. Maintaining high clinical suspicion is especially vital in patients with diabetes, given the potential for confounding metabolic neuropathies.

Differentiating SZP from other causes of acute limb weakness is critical for clinicians. Although the diagnosis is primarily clinical [1,12], laboratory investigations play an important role in excluding systemic conditions. They also identify metabolic risk factors, such as diabetes mellitus, that predispose patients to more severe viral complications.

In this clinical context, magnetic resonance imaging (MRI) is useful for excluding competing structural etiologies. These include cerebrovascular events, cervical myelopathy, malignancy, or autoimmune plexopathies [4]. In acute SZP, MRI may demonstrate T2-weighted hyperintensity and nerve enlargement, features reported in up to 70% of cases [11]. Electrodiagnostic studies were supportive of lesion localization and diagnostic confirmation, even when performed well after the acute phase [2]. In this case, nerve conduction studies and electromyography demonstrated a chronic localized axillary neuropathy with features of axonal loss and reinnervation. This close concordance between clinical findings and electrophysiological evidence supports the diagnosis of SZP. It also provides important prognostic information, as severe axonal damage, particularly in older patients, is associated with a slower and often incomplete recovery [2].

Although MRI and electrodiagnostic studies have recognized diagnostic roles in SZP, real-world clinical timelines may differ from idealized protocols, as in this case. When diagnostic workups are delayed by weeks or months, the primary clinical utility of these modalities shifts. Rather than capturing acute, transient neuroinflammatory changes, delayed MRI and electrodiagnostic evaluations can still serve as supportive tools to rule out competing structural causes, such as mechanical nerve compression, malignancy, or structural plexopathy [2]. For instance, an MRI performed several weeks after disease onset may reveal only secondary denervation changes like muscle atrophy. In our patient, imaging obtained later in the clinical course was mainly useful to exclude competing structural causes rather than to directly confirm the initial infection.

Timely treatment is generally recommended to limit neuronal injury and optimize clinical outcomes. Antiviral therapy with acyclovir, valacyclovir, or famciclovir is most effective when initiated within 72 hours of rash onset, successfully reducing viral replication, pain severity, and cutaneous healing time. Although evidence for benefit beyond this 72-hour window is limited [13,14], antiviral therapy may still be considered in select immunocompromised patients [13]. In our patient, antiviral therapy was not given because he presented 10 days after rash onset, and management focused on corticosteroids, neuropathic pain control, and rehabilitation. Systemic corticosteroids play an adjunctive role by reducing nerve edema and inflammation. Some evidence suggests that a short course of moderate-dose corticosteroid may offer a favorable balance between safety and efficacy. This regimen accelerates pain relief while suppressing key inflammatory mediators, such as interleukin-6 and interleukin-10 [15]. Neuropathic agents, including gabapentin or pregabalin, are commonly used for symptomatic pain control [12]. Given the increased risk of HZ in people with diabetes [5], vaccination against herpes zoster is an important preventive consideration in this population.

Rehabilitation is an important component of SZP management. Functional recovery is often a slow process taking 1 to 2 years [12]. Structured physiotherapy may help to prevent secondary complications, such as muscle wasting, joint instability, and contractures, while neural regeneration occurs [10]. Adjunctive modalities, including electrotherapy and transcutaneous electrical nerve stimulation (TENS), may help preserve muscle viability in chronic cases. Functional adaptation strategies further enable patients to maintain independence during prolonged recovery [1]. Despite comprehensive therapy, our patient had minimal improvement. This may reflect the guarded prognosis associated with axonal forms of SZP, aligning with literature reports of poorer functional outcomes in older individuals with axonal brachial plexopathy [3].

While this report provides valuable clinical insights, its inherent boundary conditions must be acknowledged. This case

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supports the clinical recognition of SZP in older adults with diabetes presenting with upper-limb involvement. However, because it is limited to a single case observation, it cannot establish syndrome-level frequencies, comparative diagnostic performance between modalities, broader treatment efficacy, or long-term prognosis beyond this specific presentation.

Conclusions

Segmental zoster paresis is a rare neurological complication of herpes zoster that warrants careful diagnostic consideration, particularly when a dermatomal rash and focal weakness coexist. In this case, delayed diagnostic investigations were useful mainly to exclude alternative structural pathologies and to support localized axonal injury when acute baseline data were missing. In our patient, who did not receive antiviral therapy, management relied on corticosteroids, neuropathic pain control, and physiotherapy. Structured and sustained rehabilitation remains an important component of long-term management to preserve muscle function, prevent secondary complications, and optimize functional recovery.

Acknowledgements

We would like to acknowledge our neurology fellows (Dr Hanis Rabeah Kamaruzaman, Dr Mimi Nashra Mohd Noh), physiotherapist (Fatin Nabilah Ahmad Azri), and medical assistant (Mohamed Rosmainy Izan Mohamed Badri) and their affiliations from Hospital Al-Sultan Abdullah, Universiti Teknologi MARA, Selangor, Malaysia for their valuable input into this case report.

We gratefully acknowledge Faculty of Medicine, Universiti Teknologi MARA (UiTM) for their support and for funding the article processing charges for this publication.

Department and Institution Where Work Was Done

Hospital Al-Sultan Abdullah and Primary Care Specialist Centre, Universiti Teknologi MARA (PPPUiTM), Cawangan Selangor, Selangor, Malaysia.

Consent

The patient has given his informed consent for publication and use of his imaging in the case report.

Declaration of AI Use

Generative artificial intelligence (AI) tools (Gemini, Google; and Claude, Anthropic) were used during the preparation and revision of this manuscript only to check grammar and to improve the language, readability, and overall flow of the text. They were not used to generate clinical data, findings, or interpretation. The authors reviewed, edited, and validated all suggestions provided by these tools and take full responsibility for the integrity and final content of the work.

Declaration of Figures' Authenticity

All figures submitted have been created by the authors who confirm that the images are original with no duplication and have not been previously published in whole or in part.

References:

1. Chen GB, Tuan SH, Liou IH, et al. Segmental zoster paresis of unilateral upper extremity: A case report and literature review. Medicine. 2020;99(28):e20466
2. Liu Y, Wu BY, Ma ZS, et al. A retrospective case series of segmental zoster paresis of limbs: Clinical, electrophysiological and imaging characteristics. BMC Neurol. 2018;18(1):121
3. Ismail A, Rao DG, Sharrack B. Pure motor herpes zoster induced brachial plexopathy. J Neurol. 2009;256(8):1343-45
4. Patel KR, Darweesh S, Lund D, Vanderkolk K. A rare complication of herpes zoster: Segmental zoster paresis. Cureus. 2022;14(7):e27261
5. Huang CT, Lee CY, Sung HY, et al. Association between diabetes mellitus and the risk of herpes zoster: A systematic review and meta-analysis. J Clin Endocrinol Metab. 2022;107(2):586-97
6. Sharma A, Jattan A. Diabetic amyotrophy: Atypical presentation compared with common diabetic neuropathies. Canadian Family Physician. 2023;69(8):542-44
7. Meng Y, Zhuang L, Jiang W, et al. Segmental zoster paresis: A literature review. Pain Physician. 2021;24(3):253
8. Aykac SC, Arı A, Ozoglan H, et al. Zoster-associated limb paralysis: Clinical and electrophysiological data of 15 cases with segmental zoster paresis with the literature review. Neurological Sciences and Neurophysiology. 2024;41(2):83-89
9. Gershon AA, Breuer J, Cohen JI, et al. Varicella zoster virus infection. Nat Rev Dis Primers. 2015;1(1):15016
10. Karasel S, Cebeci D. Herpes Zoster induced shoulder paresis: Case report. Cyprus Journal of Medical Sciences. 2022;7(1):152-54
11. Kesserwani H, Kesserwani HN. Post-herpetic brachial plexopathy: A rare case report with a side note on localizing brachial plexopathies and a literature review of post-herpetic segmental paresis. Cureus. 2020;12(10):e10747
12. Terzi R, Yılmaz Z. Herpes zoster sonrası brakiyel pleksus lezyonu: Olgu sunumu. Turkish Journal of Physical Medicine and Rehabilitation. 2013;59(1):69-72 [in Turkish]
13. Wood MJ, Kay R, Dworkin RH, et al. Oral acyclovir therapy accelerates pain resolution in patients with herpes zoster: A meta-analysis of placebo-controlled trials. Clin Infect Dis. 1996;22(2):341-47
14. Tyring S, Barbarash RA, Nahlik JE, et al. Famciclovir for the treatment of acute herpes zoster: effects on acute disease and postherpetic neuralgia: A randomized, double-blind, placebo-controlled trial. Annals of Internal Medicine. 1995;123(2):89-96
15. Peng L, Du B, Sun L, et al. Short term efficacy and safety of prednisone in herpes zoster and the effects on IL 6 and IL 10. Exp Ther Med. 2019;18(4):2893-900

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