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Case Report

Herpes Zoster and resultant Segmental Zoster Paresis: A Case Report and Literature Review

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Herpes zoster (HZV) is ascribed to the reactivation of the dormant varicella-zoster virus (VZV) in the dorsal root ganglia. The most commonly seen manifestation is a painful skin rash. However, several additional complications have been documented in the literature. A less-mentioned yet severe complication is segmental zoster paresis (SZP), which can cause limb paralysis. SZP's mechanism is still under debate, and a direct invasion of motor fibers due to anatomical adjacency has been proposed. Here we report a patient who had sudden onset of left upper limb paresis and gradually developed a skin rash afterward, which a dermatologist confirmed as HZV, and who received proper anti-viral management. However, the paresis persisted despite the skin rash subsiding. We concluded a diagnosis of SZP based on clinical manifestation, physical examination, musculoskeletal ultrasound examination, and electrodiagnostic studies. (Tw J Phys Med Rehabil 2022; 50(1): 77 - 83)

Key Words: Herpes zoster, segmental zoster paresis

INTRODUCTION

Herpes zoster (HZV, "shingles") occurs when the latent varicella-zoster virus (VZV) is reactivated in the dorsal root ganglion. It involves multiple sensory neurons with a resultant typical skin rash along a contiguously affected dermatome distribution.[1] HZV incidence is ~20%−30% throughout one's lifetime, the risk increases with age, and women are more susceptible.[1,2]

HZV usually is confined to the sensory system alone, with tingling pain and a burning sensation. Nevertheless, motor deficits can develop and cause limbs paresis--segmental zoster paresis (SZP).[3,4] Literature documenting SZP has described patients as developing motor weakness in the corresponding myotome after dermatomal skin eruptions.[5]

In the case we describe in this report, the patient presented with motor weakness before the skin rash occurred. Establishing an HZV diagnosis is effortless when a typical skin eruption exists; however, when weakness precedes the cutaneous manifestation, making a correct diagnosis can be difficult.

CASE REPORT

The patient, a 73-year-old woman, was robust before this episode with no systemic diseases like diabetes

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mellitus or hypertension. The patient had a sudden onset of left upper limb weakness when she woke up one morning. She recalled that she had soreness with pain over the left upper limb after a dancing contest in the past week, but she still could drive a car, cook without difficulty, and was independent in all activities of daily living. Therefore, she visited the outpatient Department of Neurosurgery the next day, where a cervical spine magnetic resonance imaging (MRI) was arranged for a week later.

Some skin rash gradually developed over the left upper limb and progressed in the following days, with a burning sensation, pain, and blisters' formation. The rash was so painful that she went to the Emergency Department for pain control. She received an intravenous steroid with analgesics and was referred to a dermatologist for further evaluation.

The patient visited the dermatologist ~1 week after the skin eruption occurred and was diagnosed with HZV. She received analgesics with anti-viral topical agents, and the pain was mitigated moderately. The neurosurgeon arranged cervical spine MRI revealed only a mild herniated intervertebral disc at the C5−C7 level with mild spinal dural sac compression and bilateral neuroforaminal stenosis (Figure 1).

The patient's pain was alleviated; however, the left upper limb weakness persisted without amelioration. Consequently, the patient visited our Rehabilitation Department for further evaluation and management ~1 month after the onset of the weakness.

The patient had poor active flexion and abduction of the left shoulder, with full passive range of motion on physical examination. In addition to poor muscle strength, she had a residual hyperpigmented skin rash with allodynia and paresthesia (Figure 2), deltoid muscle wasting, and mild atrophy of the supraspinatus muscle.

We graded the muscle strength of the left upper extremity with a manual muscle testing (MMT) system on a 0- to 5-point scale: shoulder abduction (1/5), shoulder flexion (1/5), elbow flexion (4−/5), elbow extension (4/5), wrist extension (4+/5), finger flexion (5/5), and finger abduction (4+/5). Muscle strength of the other limbs was normal (5/5). We detected allodynia with paresthesia over the ulnar side of the left upper arm and the entire forearm area. Deep tendon reflex revealed a bilaterally normoactive reflex of the triceps, biceps, and brachioradialis. Cranial nerve examination showed no abnormality.

Because of the prominent proximal weakness detected during the physical examination, we arranged a musculoskeletal ultrasound (US) to exclude rotator cuff injury, although the patient denied any recent traumatic history. US examination revealed only a suspicious mild partial tear and calcification of the supraspinatus tendon, which was disproportionate to the weakness presented, and a marked wasting of the left deltoid muscle (3.5 mm of the left side and 6.8 mm of the right side in thickness).

For further evaluation of the left upper limb, we performed nerve conduction studies (Table 1) and electromyography (EMG) (Table 2) on the 6th week after the weakness developed. We stimulated the bilateral medial, ulnar, radial, and lateral antebrachial cutaneous nerves in the sensory studies, comparing these with the unaffected side. In motor studies, we checked the bilateral medial, ulnar, and radial nerves, stimulating the bilateral Erb's point for the musculocutaneous nerve recording the biceps muscle, for the suprascapular nerve recording the supraspinatus muscle, and for the axillary nerve recording the deltoid muscle. The sensory nerve conduction studies yielded normal results, including peak latency, action potential amplitudes, and conduction velocities. The motor nerve conduction studies showed a markedly attenuated amplitude of the left musculocutaneous, suprascapular, and axillary nerves, with side-to-side differences of >50%.

According to findings based on nerve conduction studies, we checked the left biceps, supraspinatus, deltoid, and C5−C6 paraspinal muscles with EMG. We detected the active denervation sign, either fibrillation potential or positive sharp waves, over the left biceps and deltoid muscles. We detected polyphasic motor unit action potentials over the left supraspinatus and C5−C6 paraspinal muscles. We found decreased recruitment over all selected muscles, except for the deltoid, which was too weak to cooperate with the order.

Based on the clinical course, physical examination, imaging studies, and nerve conduction studies with EMG, we diagnosed HZV with related radiculopathy with resultant SZP, ongoing denervation, and reinnervation. In addition, we considered chronic C5−C6 radiculopathy as well.

We instructed the patient in home-based exercises

and scheduled regular follow-up visits in our outpatient department. The patient managed the neuropathic pain with Lyrica and Ultracet; she had palliation of pain that was tolerable. Nevertheless, the left-shoulder-movement weakness persisted without evident improvement. The patient had only mild progress in shoulder flexion, improving from 1/5 to 2-/5 by MMT grading.

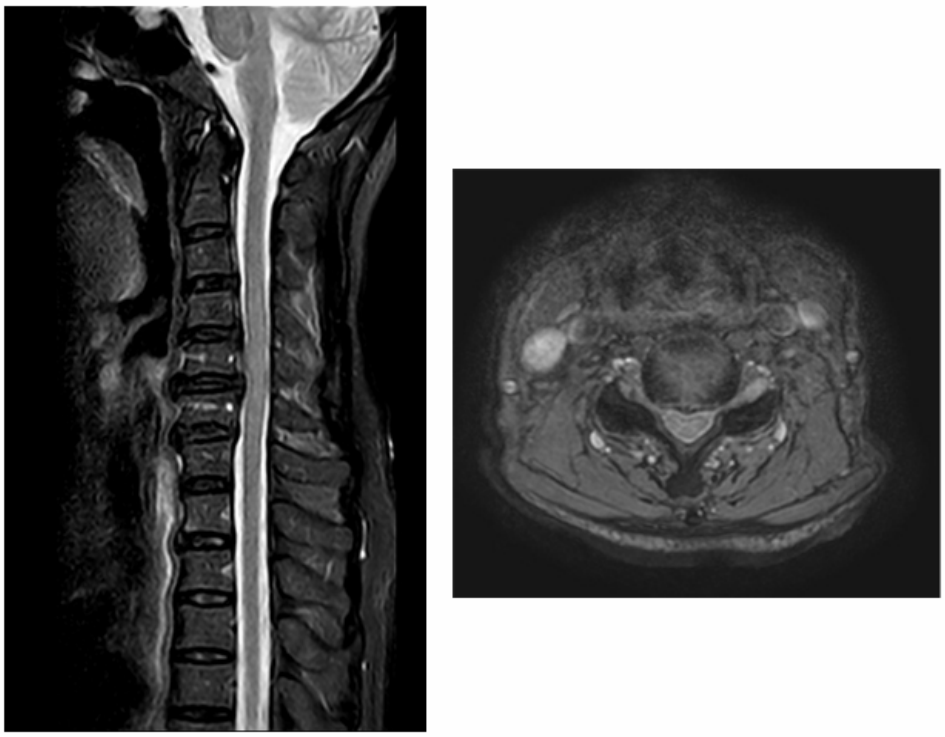

Figure 1. Cervical MRI of the patient in sagittal plane. Mild HIVD at C5 to C7 level with mild spinal canal narrowing.

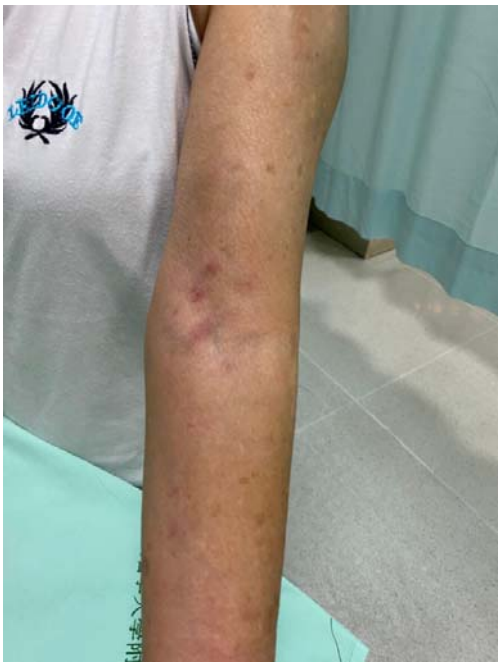

Figure 2. Residual hyperpigmented skin rash over left arm.

Table 1. Nerve conduction studies

Sensory Nerve	**Distal Peak Latency (ms)**		**Amplitude (μV)**		**Stimulation sites**	**Recording sites**	**Conduction Velocity (m/s)**	
	Right	Left	Right	Left			Right	Left
Median	3.2	3.0	36.3	42.5	Wrist	Digit 2	54	56
Ulnar	2.8	3.0	49.3	40.6	Wrist	Digit 5	67	61
Radial	2.2	2.3	33.1	24.3	Forearm	Snuffbox	56	59
lateral antebrachial cutaneous	2.6	2.9	26.4	24.2	Lateral elbow	Lateral forearm	67	58
medial antebrachial cutaneous	2.6	2.8	23.6	24.9	Medial elbow	Medial forearm	82	74
Motor Nerve	**Distal Onset Latency (ms)**		**Amplitude (mV)**		**Stimulation sites**	**Recording sites**	**Conduction Velocity (m/s)**	
	Right	Left	Right	Left			Right	Left
Median	3.4	3.3	9.3	8.8	Middle wrist and Antecubital fossa	Abductor pollicis brevis	50	51
Ulnar	2.7	2.9	6.1	5.2	Median Wrist and distal medial epicondyle	Abductor digiti minimi	60	65
Radial	1.9	1.8	4.2	3.2	Forearm and elbow	Extensor indicis proprius	54	47
Musculocutaneous	3.9	3.7	3.5	1.0	Erb' point	Biceps		
Suprascapular	2.0	1.9	5.5	2.3	Erb' point	Supraspinatus		
Axillary	3.3	3.0	6.6	0.1	Erb' point	Deltoid		

Table 2. Electromyography

Muscle	**Spontaneous activity**		**MUAP**			**Recruitment**
	Fib	PSW	Amplitude	Duration(ms)	Polyphasia	
Biceps	-	+	4 mV	5-10 ms	-	↓
Deltoid	+	+	400μV	5-10 ms	-	-*
Supraspinatus	-	-	2 mV	5-10 ms	+	↓
C5 paraspinal	-	-	2 mV	5-10 ms	+	↓
C6 paraspinal	-	-	4 mV	5-10 ms	+	↓

Fib = fibrillation potential; PSW = positive sharp waves; MUAP = motor unit action potential

*Recruitment of deltoid muscle: Too weak to follow order

DISCUSSION

HZV is a common disease caused by the reactivation of latent VZV in the dorsal root ganglia or cranial nerves. Hemorrhagic inflammation and the subsequent loss of myelin and axons can lead to neural dysfunction and cause further neuropathic pain.[3,6] The clinical course varies. Several neurological complications have serious sequelae, including aseptic meningitis, ophthalmologic involvement, postherpetic neuralgia, motor neuropathy, and stroke syndromes.[7] The thoracic dermatome is involved most frequently, followed by the lumbar and cervical regions, with the sacral area being the least.[8]

Several factors put some populations at a higher risk of acquiring HZV, such as older age (especially >50 years), immunocompromised conditions, ethnicity, genetics, mechanical trauma, psychological stress, diabetes, and female gender.[8,9] Among these, older age is the most compelling risk factor, as older adults are disproportionately affected. Older age and immunosenescence possibly could be the causes for our patient.

SZP is rare, with an incidence rate of ~0.5%-5.0%, and the interval between skin rash development and weakness generally is two weeks.[10] However, the incidence could be underestimated because diagnosis might be masked by pain-limited voluntary movement, thus deferring appropriate treatment timing. In our case, the patient already had developed weakness before the typical erythematous papules appeared. To the best of our knowledge, the literature has barely documented focal-limb paresis as the first manifestation of HZV.[11,12] The exact mechanism of SZP remains unclarified.

Direct extension to the motor fibers due to the inflammatory process has been proposed, given the adjacent anatomic relationship.[3] Some researchers have reported a slight predilection of upper-limb involvement, but this issue remains debatable.[13−15] Among SZP in the upper limbs, the C5−C7 segments are the most frequently involved, with the C8 segment being the least. For the lower limbs, the L1–L4 segments are more often affected.[5,16−18] Proximal muscle weakness is predominant in those afflicted with SZP for both the upper and lower extremities. Our patient presented with shoulder girdle weakness, corresponding with the findings of previous studies.

Nerve conduction studies and EMG can help evaluate the extent and severity of the weakness. Usually, fibrillation potential or positive sharp waves are seen on electrodiagnostic studies 2 weeks after the weakness first occurs.[18] In our case, we conducted an examination at the 6th week after onset. Both distal ongoing denervation (bicep and deltoid muscles) and proximal reinnervation (supraspinatus muscle) signs were revealed simultaneously, indicating that the weakness might be reversed (the patient has begun to recover). Despite the patient mentioned of sensory abnormality, the SNAP all showed within normal limits. We speculate that due to the SNAP recorded by elctrodiagnosis study is from large sensory fibers (Aα and Aβ fiber); however, what the patient complained is pain and tingling sensation, which is supplied by small sensory fibers (Aδ and C fiber) that would not be detected by electrodiagnostic studies.

Initially, the clinical manifestation of the patient led us to the speculation of brachial plexus injury; nevertheless, the electrodiagnostic studies revealed SNAP all within normal limits. It is known that sensory conduction studies and EMG are of help to differentiate radiculopathy from plexopathy. In preganglionic lesions, the sensory conduction studies are normal, while in postganglionic lesions the SNAPs show decreased or even absent response. In our case, by combining the clinical course and electrodiagnostic studies, HZV infection with radicular involvement should be considered. However, superimposed on chronic radiculopathy should be considered, due to MRI of cervical spine revealed spinal stenosis over C5-C7 level, yet the patient could not clearly recall whether there was sensory deficit before this episode. Polyphasia of C5 and C6 paraspinal muscles may result from reinnervation after the HZV infection episode or chronic radiculopathy related.

It is less common to see remarkable weakness in isolated radiculopathy due to every muscle is innervated by two or more myotomes; however, in extensive involvement, it may occur. Other considerations in normal SNAP with clinical sensory deficits including central nerve system lesion and conduction block distal to dorsal root ganglion. Nonetheless, there is no upper motor neuron sign of our case on physical examination, and MRI of cervical spine only revealed spinal stenosis over C5-C7 level without significant cord compression. Conduction block distal to dorsal root ganglion could not be concluded in the case, and may require further approach such as somatosensory evoked potential to help elucidate.

It was indistinguishable whether the lesions were at the nerve root or anterior horn cell level using the electrodiagnostic limitation studies unless a histopathological examination was done. MRI can help establish an early diagnosis of HZV plexopathy, in which the hyperintensity of the brachial plexus can be detected on the T2-weighted sequence.[18,19] SZP motor deficits usually are reversible, with functional recovery expected in 75% of cases in 1−2 years.[4] We encouraged our patient to maintain rehabilitation training and control neuropathic pain with medication.

CONCLUSION

SZP is a rare complication of cutaneous HZV. Establishing the correct diagnosis can be challenging in the early stages. Still, SZP should be listed as a differential diagnosis when a painful skin rash is accompanied by weakness, especially in older adults and immunocompromised individuals. Recognizing zoster-associated motor paresis can lead to prompt treatment, with a good recovery anticipated.

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帶狀皰疹誘發之肢體無力：病例報告及文獻回顧

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帶狀皰疹是歸因於背根神經節中休眠的水痘-帶狀皰疹病毒再激活而造成。最常見的表現是疼痛的皮疹。然而，文獻中也記錄了一些額外的併發症，其中一個較少被提及但嚴重的併發症是節段性帶狀皰疹麻痺，會導致肢體癱瘓。節段性帶狀皰疹麻痺的確切機制目前仍無定論，有人提出是由於解剖構造的相鄰而直接侵入運動纖維導致

我們提出一名患者，產生突發性的左上肢麻痺，而後再逐漸出現皮疹；經皮膚科醫生確診為帶狀皰疹感染，並接受了適當的抗病毒治療。然而，儘管皮疹已經消退，但麻痺的情形依舊存在。我們依據患者的臨床表現，透過理學檢查、肌肉骨骼超音波檢查、以及電學診斷，確立節段性帶狀皰疹麻痺的診斷。（台灣復健醫誌 2022；50(1)：77 - 83）

關鍵詞：帶狀疱疹(Herpes zoster)，節段性帶狀皰疹麻痺(segmental zoster paresis)

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