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Case Report

Occurrence of Segmental Zoster Paresis in the Right Arm of an Adult with Multiple Sclerosis: A Case Report

Shih-Han Lin, Wen-Shiang Chen

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Segmental zoster paresis is a rare complication of cutaneous herpes zoster. Clinical diagnosis of segmental zoster paresis depends upon a history of painful vesicular eruptions in a dermatomal distribution, followed by muscle weakness in the related myotomes. We report the case of a 45-year-old woman with opticospinal type multiple sclerosis with recent cutaneous herpes zoster and right shoulder girdle weakness. Differential diagnosis included multiple sclerosis relapse, zoster myelitis, segmental zoster shoulder paresis, cervical radiculopathy, and shoulder joint or soft-tissue disorders. MRI with gadolinium enhancement of the spinal cord 1 week after the onset of symptoms showed neither new multiple sclerosis plaque nor abnormal contrast-enhanced lesion. Right shoulder soft-tissue sonography revealed normal joint and soft tissue. Needle electromyography 3 weeks after the onset of weakness indicated increased spontaneous activity at the right biceps, anterior deltoid, and C4-C7 paraspinal muscles, as well as increased polyphasic waves at the right biceps muscle. These findings were compatible with recent right cervical polyradiculopathies involving at least the C4 to C7 levels. Therefore, the most likely etiology for her right shoulder girdle weakness was segmental zoster paresis. The patient received early antiviral therapy and intensive rehabilitation. Two months later, her shoulder muscle strength improved although it hadn’t recovered to her premorbid status. The functional recovery in her right upper limb was good. Segmental zoster paresis is underdiagnosed in patients with segmental weakness. Needle electromyography is a good tool for assisting diagnosis and prognosis and for following up motor recovery. With early diagnosis, antiviral therapy and rehabilitation therapy can hasten neurological and functional recovery. (Tw J Phys Med Rehabil 2009; 37(1): 39 - 43 )

Key Words: multiple sclerosis, herpes zoster, segmental zoster paresis

INTRODUCTION

Multiple sclerosis (MS) is a common inflammatory demyelinating disease of the central nervous system that is largely found in temperate climates. The pathogenesis of MS remains unclear. The prevalence varies around the world, and Asia has a low incidence (fewer than 5 cases per 100,000 population). Optico-spinal and spinal-form MS, the so-called Asian type MS, is more common in Asian countries than in the West. According to the latest

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statistics in March, 2008 from the Bureau of National Health Insurance in Taiwan, there were 795 patients with MS with catastrophic illness cards. The estimated prevalence in Taiwan in 2007 was 3.3 cases per 100,000 population.

Pulsed corticosteroid treatment shortens an acute MS attack, and immunomodulatory agents, such as interferon and glatiramer acetate, lessen relapse and slow the progression of disability. Because of the inflammatory nature of the disease and the immunosuppressive treatment required, patients of MS are in an immunocompromised state.

Varicella-zoster virus (VZV) is the human herpesvirus that causes chickenpox. Herpes zoster is a painful, unilateral vesicular eruption with a dermatomal distribution due to reactivation of latent VZV in the dorsal root ganglia. In immunocompromised persons or immunocompetent elderly persons, VZV may cause neurologic complications such as postherpetic neuralgia, zoster myelitis, large-vessel encephalitis, small-vessel encephalitis, meningitis, zoster plexus neuritis, polyradiculitis and segmental zoster paresis. William Broadbent first reported motor involvement of the upper extremity due to herpes zoster in 1866. There have been reports of motor involvement of the lower extremities, trigeminal and facial nerves, and the abdominal, intercostal and diaphragmatic muscles. However, it is challenging to identify the cause of motor weakness in patients with underlying neurological diseases and recent VZV reactivation. We report a case of MS with recent cutaneous herpes zoster and right shoulder girdle weakness.

**CASE REPORT**

A 45-year-old woman with a 10-year-history of MS (opticospinal form and remitting-relapsing type) was admitted to our rehabilitation unit. She had received pulse methylprednisolone therapy 1 month before this admission due to relapse of her MS, and had taken a low-dose oral steroid (40 mg prednisolone per day) upon admission. Since her recent MS relapse, she became paraplegic and was confined to a wheelchair. Upon admission, neurological examination revealed normal bilateral upper limb and poor lower limb muscle strength. The bilateral biceps, triceps and brachioradialis reflexes were symmetric and normal. In addition, she had normal sensation above the level of T2. After admission, she was given upper limb strengthening exercises and transfer skill training. There was no history of trauma or overuse of her shoulder, neck or head regions. Seven days after admission, painful erythematous vesicular eruptions developed on her right neck, shoulder and upper arm. Meanwhile, on the same day, she found she couldn’t raise her right upper arm. A diagnosis of herpes zoster in the right C5 and C6 dermatomes was made by a dermatologist. In addition to low-dose oral steroid therapy for MS, oral valacyclovir, 1 gm 3 times daily for 7 days, was prescribed. Three days after antiviral therapy begun, her right shoulder and upper arm pain improved. Neurological examination disclosed deterioration of right shoulder girdle power as follows: flexion (2/5), extension (2/5), abduction (2/5), and adduction (2/5). Mildly decreased right elbow flexion (4/5) was also observed. On individual muscle testing, there was severe weakness in the right deltoid (2/5), the right infraspinatus (2/5), and the right biceps (4/5) (Table 1). The muscle strength of right elbow extension, finger flexion, and finger abduction were preserved. The right biceps reflex was reduced, but the triceps and brachioradialis reflexes were unaffected. The patient had intact sensation (pin-prick, temperature, and joint position sense) in her right upper arm.

On the musculoskeletal examination, there was no limitation in right shoulder passive range of motion (ROM). Limited active ROM was noted in her right shoulder, as follows: 0 to 30 degrees in flexion, 0 to 60 degrees in extension, 0 to 50 degrees in abduction, 0 to 30 degrees in adduction, 0 to 40 degrees in external rotation, and 0 to 95 degrees in internal rotation (Table 1). There was neither atrophy nor fasciculation in the right shoulder musculature. The provocative tests for detecting rotator cuff injury were normal. Tenderness was noted on palpation around the posterior shoulder capsule. The patient’s cervical spine motion was not limited, and the Spurling’s test was normal.

The possible differential diagnosis of this patient’s right shoulder girdle weakness included MS relapse, zoster myelitis, segmental zoster shoulder paresis, cervical radiculopathy, and shoulder joint or soft tissue disorders. Therefore, series of laboratory studies were arranged. Right shoulder soft-tissue sonography revealed normal shoulder joint anatomy without rotator cuff pathology. MRI with gadolinium enhancement of the spinal cord one
week after the onset showed no focal contrast-enhanced lesion. There were multiple areas of increased signal intensity on T2-weighted images of the spinal cord at cervical (C2-C6) and upper to mid-thoracic levels. These findings were the same as her previous MRI at her last MS attack, and there’s no newly discovered lesion. Needle electromyography 3 weeks after the onset of weakness showed increased spontaneous activity (positive wave and fibrillation) at the right biceps, anterior deltoid and C4-C7 paraspinal muscles, as well as increased polyphasic waves at the right biceps muscle. These findings were compatible with recent right cervical polyradiculopathies involving at least levels C4 to C7. Normal motor unit action potential was recorded in the deltoid muscle (the weakest muscle documented by physical examination), which might indicate a fair-to-good prognosis for recovery. Furthermore, increased giant waves without spontaneous activities at the right brachioradialis muscle and increased high amplitude motor unit action potentials without spontaneous activities at the right triceps muscle were also noted. This abnormality was consistent with a superimposed chronic axonal degeneration proximal to sensory ganglion, which involved mainly C6 to C7. This might be attributed to the results of her previous MS lesions in the cervical spinal cord. The patient had a history of an MS attack of her left shoulder, with weakness and numbness 18 months before, which recovered within one month after pulse steroid therapy. On the other hand, the motor and sensory nerve conduction studies at right median, radial and ulnar nerves were within normal limits.

The patient received intensive physical and occupational therapy with shoulder muscle strengthening, range-of-motion exercises and functional task training. Two months later, her right shoulder girdle muscle power had recovered to the following degree: flexion (4/5), extension (4/5), abduction (4/5) and adduction (4/5). On individual muscle testing, muscle power had improved in the right deltoid (4/5), right infraspinatus (4/5), and right biceps (5/5) (Table 1). Although her shoulder muscle strength hasn’t recovered to its premorbid status, the functional recovery in her right upper arm was good.

### DISCUSSION

In immunocompetent elderly persons or immunocompromised patients, VZV may produce disease of the central nervous system and the peripheral nervous system as well. In our patient, the differential diagnosis of new-onset motor weakness was more challenging than usual, due to her history of frequent MS relapses and her immunocompromised status. The most likely differential diagnosis of central nervous involvement in this patient was MS relapse and zoster myelitis. Generally, the detection of VZV DNA or antibody to VZV in cerebrospinal fluid indicated acute zoster infection of the central nervous system. But VZV DNA can be present in the cerebrospinal fluid (CSF) of patients with a wide range of neurological symptoms, including MS.\(^6\) In one study investigating the possible involvement of VZV in MS relapse, VZV DNA was detected in 95% of MS patients during relapse and in 17% during remission.\(^7\) Therefore, the presence of VZV DNA in the CSF was not a reliable tool for differentiating MS relapse or zoster myelitis infection. In this patient, the neurological examination revealed reduced right biceps reflex. MRI with gadolinium enhancement of the spinal cord showed neither new plaque nor a new lesion. Central-nervous-system-related

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**Table 1. Right shoulder girdle muscle power and active range of motion**

<table>
<thead>
<tr>
<th>Manual muscle power testing</th>
<th>Active range of motion (degree) during onset</th>
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<tbody>
<tr>
<td>Before onset</td>
<td>Onset</td>
</tr>
<tr>
<td>Right shoulder flexion</td>
<td>5/5</td>
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<tr>
<td>Right shoulder extension</td>
<td>5/5</td>
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<td>Right shoulder abduction</td>
<td>5/5</td>
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<tr>
<td>Right shoulder adduction</td>
<td>5/5</td>
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<tr>
<td>Right elbow flexion</td>
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right shoulder girdle weakness seemed less likely. As for peripheral causes of her shoulder pain and weakness, the most common differential diagnoses included musculoskeletal conditions and neurological conditions involving the peripheral nervous system. The possible musculoskeletal causes were: rotator cuff injury, impingement syndrome, adhesive capsulitis, glenohumeral arthritis, myopathy and cervical spine disease, among others. Results of the physical examination, right shoulder sonography, and laboratory studies excluded musculoskeletal abnormalities and cervical radiculopathy attributable to cervical spine disease.

Based on the clinical data, the most likely etiology for her right shoulder girdle weakness was segmental zoster paresis. The clinical diagnosis of segmental zoster paresis depends upon a history of painful vesicular eruptions in a dermatomal distribution, followed by muscle weakness in the related myotomes. Imaging and electrodiagnostic studies may help differentiate other disorders that cause similar symptoms and may also help localize the site of the lesion. Needle electromyography is a good clinical tool for diagnosing motor involvement of herpes zoster infection, predicting its prognosis, and following up motor recovery. In such cases, needle EMG usually shows abnormal spontaneous activities and fibrillation in the clinically weak muscles.[8,9] These abnormalities appear 2 weeks after the onset of weakness and can persist for months. The EMG findings in our patient were compatible with the diagnosis of acute polyradiculopathy involving at least the C4 to C7 levels. Hence, zoster polyradiculopathy was the most likely cause, based on the temporal relationship between the herpetic eruptions and right shoulder weakness.

The prognosis of segmental zoster paresis in immunocompetent individuals is generally good. However, there have been few studies of the prognosis for immunocompromised patients. Early antiviral therapy could reduce severity of segmental zoster paresis, and rehabilitation could promote functional recovery.

Segmental zoster paresis is a rare complication of cutaneous herpes zoster, and thus is underdiagnosed in most clinical settings. It should be included in the differential diagnosis of segmental muscle weakness, especially in immunocompromised patients or in the elderly. Early diagnosis may lead to early antiviral therapy and rehabilitation and hasten neurological and functional recovery.

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多發性硬化症病患之右上臂皰疹性輕癱：病例報告

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皰疹性輕癱是帶狀皰疹感染較少見的後遺症，它的臨床特徵是與帶狀皰疹發疹皮節相對應肌節的運動麻痺。我們報告一個 45 歲的視神經脊髓型多發性硬化症患者，在右側頸第五、六皮節帶狀皰疹發疹後，發生右肩無力。鑑別診斷包括多發性硬化症復發、帶狀皰疹脊髓炎、皰疹性輕癱與肩部或頸椎附近肌肉骨骼系統的病變。發病一週後的磁振造影檢查並未在脊髓發現新的多發性硬化症斑塊或不正常的顯影，而右肩軟組織超音波也沒有發現骨骼肌肉系統病變。發病三週後的針極肌電圖檢查顯示右三角肌、肱二頭肌與頸椎第四到第七節椎邊肌有自發性活動電位，這與患者因肌節受皰疹感染而導致頸神經根病變的症狀相吻合，因此皰疹性輕癱導致右肩無力為這個患者最可能的診斷。除了早期抗病毒藥物的給予外，我們為患者安排積極的肌肉強化運動與關節活動。發病二個月後追蹤病人的復原情形，她的右肩帶肌力雖未恢復到發病前的狀態，但可以從事大部分的功能性活動。皰疹性運動麻痺是一個在臨床上鑑別診斷肢體無力常被忽略的病因，而針極肌電圖是輔助診斷、預測預後與追蹤復原情形的良好工具。提高臨床診斷率並及早投予抗病毒藥物治療能加快復原的速度，早期復健能促進病人的功能性恢復。（台灣復健醫誌 2009；37(1): 39 - 43）

關鍵詞：多發性硬化症(multiple sclerosis)，帶狀皰疹/herpes zoster)，皰疹性輕癱(segmental zoster paresis)