



# A Brachial Plexus Lesion Secondary to Herpes Zoster: A Case Report

## Herpes Zoster Sonrası Brakiyel Pleksus Lezyonu: Olgu Sunumu

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### Summary

Brachial plexus lesions secondary to Herpes zoster is a rare neurological complication. This condition should be kept in mind in the differential diagnosis of acute weakness in the upper extremities. In this case report, we present a 57-year-old female patient, who presented to our clinic with complaints of weakness and pain in the left arm 3 days after the development of a herpetic lesion on the left shoulder and underwent an investigation that revealed a brachial plexus lesion. *Turk J Phys Med Rehab 2013;59:69-72.*

**Key Words:** Brachial plexus lesion, herpes zoster

### Özet

Brakial pleksus lezyonu Herpes zostere bağlı nadiren gelişen nörolojik komplikasyonlardan biridir. Üst ekstremitelerde herpes zostere bağlı ani gelişen kuvvetsizliklerin ayırıcı tanısında düşünülmelidir. Bu olgu sunumunda 57 yaşında kadın hasta sol omuz üzerinde herpetik lezyon gelişiminden 3 gün sonra oluşan sol kolda kuvvetsizlik ve ağrı şikayeti ile kliniğimize başvurmuş olup, yapılan tetkikler sonucunda brakial pleksus lezyonu saptanması üzerine sunulmuş ve literatür eşliğinde tartışılmıştır. *Türk Fiz Tıp Rehab Derg 2013;59:69-72.*

**Anahtar Kelimeler:** Brakial pleksus lezyonu, herpes zoster

### Introduction

Herpes zoster (HZ) is a common infectious disease that leads to dermatomal vesicular skin lesions and spreads after reactivation of the varicella zoster (VZ) virus in the dorsal root ganglions. The cellular immune system is significantly involved in the incidence of lesions, which increases with age (1).

Herpes zoster can easily be diagnosed when it manifests with only a dermatomal rash. Although these symptoms are rare, the diagnosis and treatment may be delayed when they lead to motor debility as well as dermatomal rashes (2).

In this case report, we present a patient with weakness in the left arm occurring after VZ infection.

### Case Report

A 57-year-old female patient presented to our clinic with complaints of pain and weakness in the left arm that had started approximately 20 days previously. The patient was complaining about difficulty in using her left arm for daily activities, and a burning-like pain was occasionally observed during the day.

A detailed inquiry of the patient revealed that vesicular skin lesions had occurred on the left arm and shoulder together with the onset of the pain, and at this point, the lesions had not completely regressed. She had developed weakness in the left arm 3 days after the appearance of the skin lesions. There was no history of trauma to the head-neck area or the left arm. Her medical history included use of antihypertensive agents and a cholecystectomy that was previously performed nearly 1 year earlier.

Physical examination revealed normal and minimally painful cervical range of motion (ROM). No radicular pain was detected in the Spurling's test. Pigmented macular skin lesions were observed in regions consistent with C5 dermatome in the left arm (Figure 1). While the passive ROM of the left shoulder was normal but painful in all directions, it was highly limited and painful in all directions upon active movement. There was no marked atrophy or fasciculation in the left shoulder muscles. The left elbow, wrist, and finger ROM were normal and free of pain. Neurological examination revealed shoulder girdle flexion, extension, and adduction muscle strength of 1/5, elbow flexion

of 3/5 and extension of 4/5, wrist extension and flexion muscle strength of 4+/5, and finger adduction and abduction of 4/5. Biceps and brachioradialis reflexes were considered hypoactive. In addition, hypoesthesia was found in the C5 dermatome. Next, a dermatology consultation was sought for the patient's skin lesions. The skin lesions were considered to represent an HZ infection. No antiviral treatment was recommended.

Laboratory investigations revealed normal hemogram, erythrocyte sedimentation rate, C-reactive protein, and biochemical tests. Cranial magnetic resonance imaging (MRI) revealed no peculiarities other than mild cerebral atrophy. Cervical MRI detected changes consistent with cervical arthrosis and multiple protrusions that did not lead to compression in the cervical roots. An MRI of the left shoulder showed supraspinatus tendinitis and degenerative changes. As the weakness in the left arm could not be explained by MRI or the other investigations, neurologic consultation was sought, and an electroneuromyography (ENMG) test was performed.

Bilateral nerve conduction study and ENMG were performed for the musculocutaneous, axillary and the radial nerves. Brachial plexus was stimulated at 2.5 cm above the clavicle at the level of the 6th cervical vertebra at the triangular localization between the clavicle and posterolateral line of the sternocleidomastoid muscle. Motor response of the musculocutaneous nerve was recorded from the biceps muscle, the axillary nerve from the deltoid muscle, and the radial nerve from the triceps muscle by using superficial electrodes. Amplitude of motor responses was decreased for the left musculocutaneous and the radial nerves. No response was obtained from the lateral antebrachial cutaneous nerve. Sensory nerve conduction velocity was reduced, amplitude was decreased and the latency was prolonged in the 3<sup>rd</sup> finger-wrist segment in the median nerve. Normal motor and sensory responses were obtained from the peripheral nerves, which innervate the lower trunk. ENMG showed spontaneous denervation activity in muscles of the musculocutaneous and axillary nerves and chronic neurogenic changes in the muscles innervated by the radial nerve.



Figure 1. Skin lesions beginning to heal after Herpes zoster infection.

Normal motor unit potentials (MUPs), recruitment (i.e. firing rate of MUP), and interference patterns were observed in the paraspinal muscles. Cervical paraspinal muscle evaluation is performed while patient is in prone position. A pillow may be placed under patient's chest to keep head in flexion and forehead downwards. Patient is asked to hold his/her up to extension. If relaxation could not be obtained, patient is asked to suppress his/her head to the bed. With this maneuver, interference and recruitment can be evaluated visually and subjectively. We evaluated visually. These findings showed that the brachial plexus on the left and the axons of the upper and middle trunks were involved.

The patient was administered gabapentin, tramadol, and an analgesic for the neuropathic pain. She was also given a physical therapy and rehabilitation program, and she was administered ROM and strengthening exercises. After six weeks, she confirmed during the examination that her pain was nearly completely relieved. Her examination revealed increased shoulder ROM and partially improved muscle strength. The skin lesions were still present but showed regression. Minimal atrophy was detected in the deltoid muscle, and electrical stimulation was administered to prevent the progression of the deltoid atrophy. No change in muscle strength was detected relative to the previous values, and no progression of the atrophy was detected in the deltoid muscle at the follow-up visit performed 3 months later.

## Discussion

Neurologic complications secondary to HZ are rare except in cases of postherpetic neuralgia and meningitis (3). Rare neurologic complications secondary to HZ include zoster myelitis, meningoencephalitis, Brown-Sequard syndrome, plexus neuritis, polyradiculitis, and segmentary zoster paresis (4-7). HZ may not only be involved in the sensory system but may also affect the motor nervous system, which may lead to weakness in some patients (7).

The incidence of involvement of motor fibers secondary to HZ is reported to be between 0.5 and 3% (8). The pathogenesis of muscular paresis is not clear. The dermatomal and myotomal association in these patients suggests direct diffusion of the virus from the dorsal root ganglions to the anterior horn cells (9). It has been shown that inflammation of the motor nerves results in neurologic deficits (6). Patients with HZ infections who develop motor complications are primarily middle-aged and elderly people. Pediatric and young adult populations rarely develop motor complications. However, when they do, the cranial and truncal muscles are often affected (10). Smoking and diabetes are associated with neurologic complications (11). Our patient was 57 years old and had no history of smoking or systemic disease other than hypertension.

In the literature, there are a few cases where HZ resulted in involvement of the brachial plexus. Fabian et al. (6) defined brachial plexus neuritis as secondary to HZ in their postmortem investigation of a 78-year-old female patient with monoplegia in the left upper extremity died due to myocardial infarction. They observed increased lymphocytic infiltration and myelin breakdown on histological investigation. They explained the motor neuropathy as a consequence of the inflammatory demyelination process.

Jeevarethinam et al. (12) presented a case of brachial plexopathy with a severe manifestation of radial nerve paralysis, and they obtained a good response to acyclovir, gabapentin, and physical therapy. Ohtake et al. (13) described a 73-year-old female who developed monoparesis in the right upper extremity following brachial plexus neuritis secondary to HZ. Ismail et al. (14) demonstrated that involvement of the brachial plexus resulted in poor improvement after an HZ infection in a 78-year-old male patient. Eyigör et al. (15) reported a case of monoparesis secondary to brachial plexopathy following HZ in a 54-year-old male patient who exhibited symptoms similar to complex regional pain syndrome. In that case, the partial degeneration of the upper, middle and the inferior trunks of the brachial plexus were demonstrated electrophysiologically. Hoque et al. (16) described a case of bilateral diaphragm paresis secondary to brachial neuritis following thoracic HZ. Our patient exhibited involvement of the upper and middle trunks of the brachial plexus, and moderate improvement was achieved.

In cases where paresis follows an HZ infection, the diagnosis is established based on the history of weakness following painful skin lesions, exclusion of other causes of weakness, and an ENMG test (17). In our case, the diagnosis of a brachial plexitis secondary to HZ was established based on the development of weakness following the occurrence of herpetic skin lesions on the left shoulder, findings from the cervical, cranial, and left shoulder MRI, a failure to explain the weakness in the left arm, and demonstration of the involvement of the upper and middle trunks of the brachial plexus in the left shoulder.

The interval between the occurrence of the skin lesions and muscle weakness following an HZ infection is variable. Usually, while the weakness occurs days after the rash, a few cases have been shown to develop weakness months after the rash (18,19). There is a single case in the literature that was difficult to diagnose, where the motor paresis developed 3 days before the skin lesions (20). Our patients developed muscle weakness 3 days after the skin lesions. Zoster paresis usually has a good prognosis. Functional improvement occurs in 50% of cases (21). The improvement period is variable; improvement periods ranging between 1 and 2 years have been reported in the literature (22). In certain local cases, the period of improvement has been reported to range between 3 and 9 months (20). Poor improvement in some patients with zoster paresis could be explained by the death of motor neuron cells. Glial scar formation has been demonstrated in a patient with postherpetic polyradiculitis on MRI, which may explain why some patients fail to improve completely (23). Minimal improvement was observed 12 weeks later in a case with brachial plexus involvement, and the same level of improvement was observed at 6 and 12 months (14). In the sixth week, we observed a moderate improvement in muscle strength in our patient. In the third month, no major change in muscle strength was detected. We scheduled a follow-up with the patient to monitor the improvement.

In the literature, there are some reports suggesting that an antiviral treatment at an appropriate dose and for an appropriate period following an HZ infection could be effective in reducing the incidence of segmental zoster paresis and the

severity of electrophysiological changes. Steroids are suggested to be beneficial in suppressing severe infections in skin lesions, and the initiation of IV acyclovir and IV steroids is recommended as soon as zoster paresis is suspected (24).

We administered gabapentin, tramadol, and analgesic treatment to our patient for the medical therapy of neuropathic pain. The skin lesions had developed 20 days previously and had started to regress at the time of examination, and the weakness has begun to improve relative to the onset of the disease, thus, we did not administer a treatment with acyclovir and steroids. If the relevant medical treatment had been given to our patient in the early period of the disease process, the improvement could have been better. The patient was put on a physical therapy and rehabilitation program to prevent muscle atrophy and contracture as well as to strengthen the muscles. We believe that physical therapy and rehabilitation were significantly involved in the moderate motor improvement and the lack of a joint contracture.

As a conclusion, brachial plexus neuritis secondary to HZ should be considered in the differential diagnosis of acute paresis developing in the upper extremity. A good understanding of the neurologic complications secondary to HZ would provide the opportunity for early diagnosis and treatment.

#### Conflict of Interest

Authors reported no conflicts of interest.

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