Brachial Plexopathy Associated with Herpes Zoster Infection: Report of Two Cases and Review of the Literature

Herpes Zoster Enfeksiyonu İle İlişkili Brakial Pleksus Lezyonu: İki Olgu Sunumu ve Literatür Derlemesi

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ABSTRACT

Herpes zoster is an infectious disease caused by the reactivation of the varicella zoster virus in dorsal sensory ganglia. Herpes zoster usually affects sensory nerves but can sometimes also damage motor neurons and nerves. Here we present two cases of brachial plexopathy after herpes zoster infection. Brachial plexus neuritis secondary to herpes zoster infection should be considered in the differential diagnosis in cases that develop acute paresis in the upper extremities.

Keywords: Brachial plexopathy, herpes zoster, paresis, rehabilitation

ÖZET

Herpes zoster, dorsal duyu ganglionlarında varisella zoster virüsünün reaktivasyonunun neden olduğu enfeksiyöz bir hastalıktır. Herpes zoster, genellikle duyu sinirlerini etkiler ama bazen motor nöron ve sinirlere de zarar verebilir. Burada herpes zoster enfeksiyonu sonrası brakial pleksopati gelişen iki olgu sunulmaktadır. Herpes zoster enfeksiyonuna sekonder brakial pleksus nöriti, üst ekstremitede akut parezi gelişen durumlarda ayırıcı tanıda mutlaka düşünülmelidir.

Anahtar sözcükler: Brakial pleksopati, herpes zoster, parezis, rehabilitasyon

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Introduction

Herpes zoster (HZ) is an infectious disease caused by the reactivation of the varicella zoster virus in the dorsal sensory ganglia. Vesicular skin eruptions, neuralgia, and sensory symptoms are manifestations of this infection (1). HZ usually affects the sensory nerves, but can also damage motor neurons and nerves (2,3). Motor paresis occurs in less than 5% of HZ patients. Brachial plexus involvement is a rare occurrence (4). We present two cases of brachial plexopathy after HZ infection.

Case 1

A 69-year-old man with a history of weakness and pain in his left arm was referred to our clinic. He had complained of pain in his left shoulder followed by a vesicular eruption on the same area four weeks ago. A diagnosis of HZ had been made by a dermatologist and medical treatment with acyclovir was started. A few days later, he complained of difficulty in moving the arm. His medical history included hypertension. Physical examination revealed hyperpigmented macular

lesions on the C5-C6 dermatomes in the left arm. Marked weakness was found in the left deltoid and biceps muscles (1/5), whereas moderate loss of muscle strength (3/5) was present in the triceps, wrist extensors and flexors, and finger extensors. Hypoesthesia was found in the left arm and additionally, reflexes of the biseps, triceps and brachioradialis were absent. Cranial, cervical and left brachial plexus magnetic resonance imaging (MRI) revealed no pathology. Electrodiagnostic evaluation was performed and supported a brachial plexus lesion that affected the middle and inferior and especially the superior trunks. Gabapentin and an analgesic medication was given to the patient for neuropathic pain. A physical therapy and rehabilitation programme which consisted of range of motion exercises of the left upper extremity joints to prevent contractures, progressive resistance exercises to improvement muscle strength and electrical stimulation for avoiding muscle atrophy was also given to him.

Case 2

A 72-year-old woman with a history of weakness and pain in her right shoulder was referred to our clinic. A vesicular eruption had developed on her right shoulder two days after the onset of pain. A few days later she complained of difficulty in moving the arm. Her medical history included diabetes mellitus and hypertension. On physical examination, there were vesicular lesions on the right C5 dermatome. The motor strength of the right deltoid was 1/5, biceps muscle 4/5, and other muscle groups 5/5. She had hypoesthesia and dysesthesia in the C5 dermatome and reflex of the biceps was absent. She did not accept imaging studies. The electrodiagnostic evaluation results supported a severe brachial plexus lesion of the upper trunk. Diagnosis of HZ infection was confirmed by a consultant dermatologist and antiviral treatment was recommended. The patient was prescribed gabapentin and tramadol cap to control her neuropathic pain. A physical therapy and rehabilitation programme including range of motion along with strengthening exercises was initiated for her.

Discussion

Herpes Zoster can affect both sensory and motor nerves, and lead to motor axonal injury in several sites such as the motor neuron, roots, plexus or peripheral nerve (5). Rare neurological complications of HZ infection are myelitis, meningoencephalitis, Brown-Sequard syndrome, plexus neuritis, polyradiculitis and segmentary zoster paresis (2,5-8). The incidence of motor fiber involvement secondary to HZ is estimated to be between 0.5 and 5% (4,9,10). Some studies have shown that subclinical motor involvement is not uncommon and reported denervation potentials in 40% to 50% of cutaneous zoster patients with electromyographic examinations (3,11). Inflammation of the motor nerves results in neurological deficits but the pathogenesis is not clear (8). The association between the involved myotome and dermatome of the rash indicates viral spread from the dorsal root ganglion to the anterior horn cells or anterior spinal nerve roots, resulting in inflammation. Hanakawa et al. reported that this inflammation causes a neurological deficit by producing hypervascularity in the perineural structures or actual disruption of the blood nerve barrier (12).

Fabian et al. first reported a brachial plexus inflammation associated with clinical HZ paresis. They found extensive lymphocytic infiltration, myelin breakdown, and preservation of axons without vasculitis and also determined that the cervical spinal cord had perivascular lymphocytic cuffing but no anterior horn necrosis. They suggested that the brachial plexus inflammation was a distal extension of a dorsal ganglionitis (8). Eyigör et al. reported a case of monoparesis secondary to brachial plexopathy following HZ infection in a 54-year-old male and demonstrated partial degeneration of the upper, middle and inferior trunks of the brachial plexus electropyhsiologically (10). Jeevarethinam et al. also presented an 83-year-old female patient with brachial plexopathy after HZ infection and they treated the patient successfully with acyclovir, gabapentin and physiotherapy (4). Terzi et al. reported a case of brachial plexus lesion secondary to HZ in a 57-year-old female patient. The patient was administered a rehabilitation program. Pain was nearly completely relieved but no change in muscle strength was detected (13). Here we present two cases with brachial plexopathy associated with HZ infection. There was a lesion in all three trunks in one case but the other case only demonstrated an upper trunk lesion.

It is not clear that why some patients develop motor weakness and others do not. Mondelli et al. conducted a study with 158 patients and suggested that a higher age at onset significantly correlates with the incidence of segmental HZ paresis and the severity of electrophysiological abnormalities (3). It has also been shown that diabet is associated with neurological complications in HZ infections but there is no specific data for HZ paresis (14). One of our cases had diabetes mellitus and both were older than 65 years.

Choi et al. described two cases of zoster brachial plexopathy. They found T2 hyperintensity and contrast enhancement in part of the brachial plexus and this result was compatible with both the clinical symptoms and the electrophysiological findings of their cases (15). Similarly,

Hanakawa et al. presented a case with HZ paresis and MRI showed contrast enhancement of the anterior roots of the affected segments, suggesting the presence of hypervascularity or disruption of the blood-nerve barrier caused by viral-induced inflammation in patients with HZ paresis (12). Unlike these reports, Yoleri et al. presented a case with brachial plexus lesion due to HZ infection and also found no pathology on the MRI studies (16). Kawajiri et al. presented three cases with HZ paresis of the limbs where the MRI again revealed no pathology (2). We also found no pathology with MRI studies in our first cases. Differences in MRI results can be due to techniques or imaging timing. MRI of the brachial plexus could provide valuable information for evaluating the location and extent of lesions together with electrophysiological studies. However, MRI has not revealed any pathology in some cases as mentioned above.

Herpes Zoster paresis management includes antiviral therapy, pain relief and a rehabilitation program (4). Mondelli reported that antiviral therapy of appropriate duration using the appropriate dose is associated with a reduced incidence of segmental HZ paresis and a reduced severity of electrophysiological alteration (17). Early initiation of a rehabilitation program is recommended (5). The goals of the rehabilitation include prevention of muscle atrophy and contractures, strengthening of weak muscles, and pain relief. A home exercise program should also be prescribed for each patient.

Herpes Zoster paresis usually has a good prognosis with more than half of the patients showing complete functional recovery. Complete or partial recovery has been reported in 75% of patients after a varying duration of 1 to 2 years (17,18). Poor improvement can be observed in some patients with HZ paresis and is considered to be due to the death of motor neuron cells.

In conclusion, physicians should be aware of HZ paresis, a rare complication of HZ infection. Brachial plexus neuritis secondary to HZ infection should be considered in the differential diagnosis in cases that develop acute paresis in the upper extremities. A detailed examination and keeping this condition in mind will provide an opportunity for early diagnosis and treatment.

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