Segmental Limb Paresis Due to Zoster Plexopathy: Case Report

Zoster Pleksopatiye Bağlı Segmenter Ekstremite Parezisi

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ABSTRACT Segmental zoster paresis (SZP) is focal, asymmetric and a rare cause of neurogenic weakness that may occur in a limb caused by herpes zoster (HZ). It typically occurs 2-3 weeks after the herpetic rash and usually affects the myotome that corresponds to the dermatomal distribution. HZ plexopathy may be overlooked in the acute period, or the motor paresis may be mistakenly diagnosed as another cervical or lumbar pathology after the vesicular lesions have disappeared. Three zoster plexopathy cases; two brachial and one lumbar were presented in this article. Electrophysiological study (EDX) findings, limb paresis, pain and erupted skin lesions with positive VZV antibodies indicated HZ plexopathy. EDX revealed patchy involvement of brachial and lumbar plexus. Antiviral treatment and physiotherapy improved muscle strength to mild paresis. An accurate and early diagnosis is vital for total recovery of muscle strength.

Key Words: Brachial plexus neuropathies; electromyography; herpes zoster; acyclovir; sacral plexopathy

ÖZET Segmenter zoster parezisi (SZP) fokal, asimetrik, herpes zoster (HZ) nedeniyle tek ekstremitede oluşabilen nadir bir nörojenik güçsüzlüktür. Tipik olarak herpetik döküntüden 2-3 hafta sonra meydana gelir ve genellikle dermatomal dağılıma karşılık gelen miyotomu etkiler. HZ pleksopati akut dönemde gözden kaçabilir veya lezyonlar kaybolduktan sonra motor pareziye yanlışlıkla diğer servikal ve lomber patoloji tanıları konulabilir. Bu makalede iki brakial ve bir lomber pleksopati olmak üzere üç HZ pleksopati olgusu sunuldu. Elektrofizyolojik çalışma (EDX) bulguları, ekstremite parezisi, ağrı ve kabuklanmış cilt lezyonları ile birlikte pozitif VZV antikorları HZ pleksopatiye işaret etti. EDX bulguları brakial ve lomber pleksusun yamalı tutulumu ile uyumluydu. Antiviral tedavi ve fizyoterapi tüm olgularda kas gücünü hafif pareziye geriletti. Akut fazda doğru ve erken tanı kas gücünün total geri dönüşü için gereklidir.

Anahtar Kelimeler: Brakiyal pleksus nöropatileri; elektromiyografi; herpes zoster; asiklovir; sakral pleksopati

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Herpes zoster (HZ) is the reactivation of dormant varicella zoster virus (VZV) in the dorsal root ganglia. The commonly involved nerve segments are thoracic, lumbar and trigeminal.[1,2] It may also spread to involve the nerve roots adjacent to the dorsal root ganglia causing a plexitis or neuritis.[3,4] Postherpetic neuralgia is the most common complication of HZ affecting more than 40% of patients. In contrast, the motor complications of herpes zoster are rare and their true incidence is unknown. Review of the literature suggests that motor involvement occur between 1-

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5% of patients with HZ. This percentage may increase with electrophysiological (EDX) studies.[5-7]

Segmental motor paresis (SMP) is focal, asymmetric neurogenic weakness that may occur in a limb affected by HZ.[2,5] It typically occurs 2 to 3 weeks after the herpetic rash and usually affects the myotome that corresponds to the dermatomal distribution. Weakness associated with HZ has been recognized since the 19th century and can affect bulbar, limb, or truncal muscles.[7] The incidence of SMP may increase with EDX studies revealed that subclinical motor involvement is more prevalent in patients with zoster infection. However, plexopathy presenting with severe motor paralysis due to zoster infection has been rarely described.[4,8] With this report we aimed to draw attention to this uncommon complication of herpes zoster with cases presenting severe brachial and lumbar plexitis and importance of early antiviral treatment.

CASE REPORTS

CASE 1

A 65-year-old male presented with motor paresis of the right upper extremity and pain and skin rashes on the C6-C7 dermatome, both of which had begun approximately 15 days earlier (Figure 1). One year previously, he had suffered pain and rashes in his right upper extremity, but had recovered spontaneously. On EDX examination, the amplitudes of the motor unit potentials (MUPs) of the right axillary, musculocutaneous, and infraspinatus nerves were lower on the right than on the left side. The median sensory amplitudes of the first and second fingers were low, but his other sensory responses were in the normal range. Electromyography examination of the right deltoid, biceps, infraspinatus, and supraspinatus muscles showed neurogenic MUP and MUP loss without spontaneous denervation. Cervical and brachial plexus magnetic resonance imaging (MRI) findings were normal, even in contrast-enhanced series. IgM and IgG against VZV were positive in serum. A neurological examination yielded Medical Research Council (MRC) scores for right upper limb proximal flexion of 3/5, abduction +3/5, and distal flexion and abduction of full muscle strength. Muscle strength on extension of the proximal limb was normal. Other neurological examination findings were also normal. Pregabalin tablets were prescribed for pain relief, and antiviral acyclovir tablets for treatment. Physiotherapy improved the patient's muscle strength. Two months after the first examination, the rashes had completely recovered. Proximal muscle strength had nearly fully improved (+4/5).

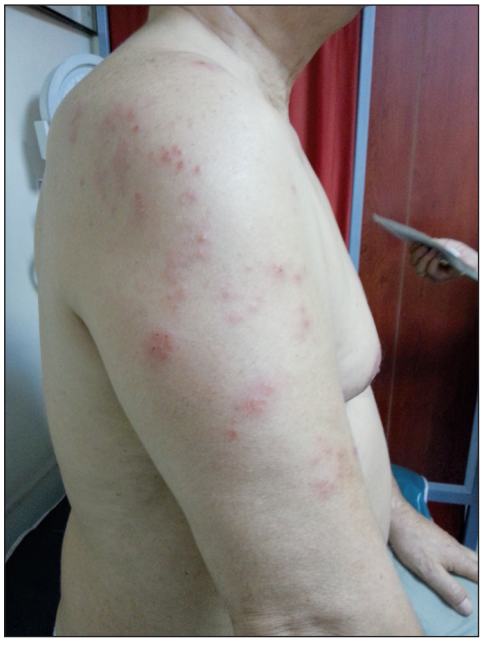

FIGURE 1: Vesicular eruptions on C5,C6 and C7 dermatomes.

CASE 2

A 56-year-old female was admitted to our clinic complaining of pain and weakness in her left leg that had persisted for approximately 15 days. One month earlier, she had experienced vesicular rashes on the L1–2–3 dermatomes, which meanwhile had partly recovered (Figure 2). Limb paresis had begun within the last 15 days. An EDX study showed lower MUP amplitudes for the left obturator and femoral nerves. Sensory responses for the lateral cutaneous, femoral, and saphenous nerves could not be obtained. Spontaneous denervation (fibrillation and positive sharp waves) and neurogenic polyphasic MUP and MUP loss were determined in the rectus femoris, vastus lateralis, and adductor muscles. MRI findings for the lumbar plexus were normal. Atrophy of the left quadratus lumborum

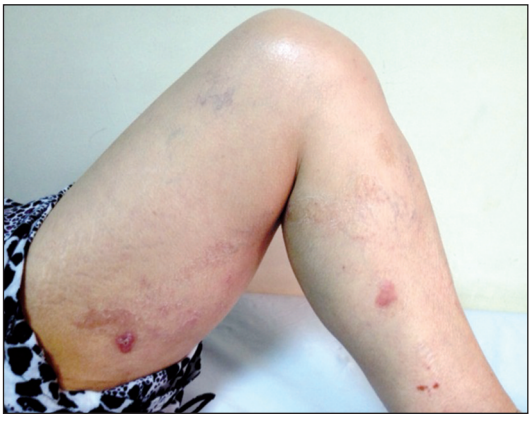

FIGURE 2: Skin eruptions in the healing process on L3 and L4 dermatomes.

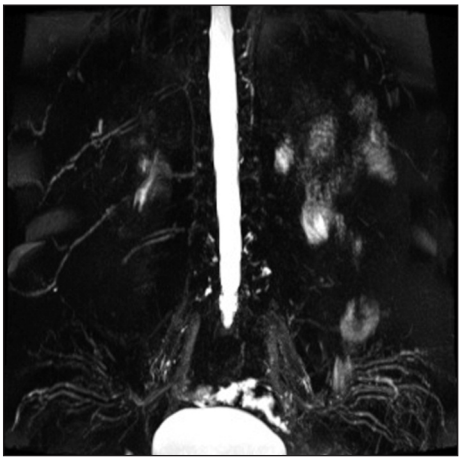

FIGURE 3: Normal lumbar plexus MRI. No contrast enhancement.

muscle was determined (Figure 3). Neurological examination of muscle strength yielded MRC muscle strength scores for left thigh flexion of -4/5 and adduction 4/5. Other neurological examination findings were normal. Pregabalin and antiviral acyclovir tablets were prescribed, together with physiotherapy. One month later, her left thigh muscle strength had improved to +4/5.

CASE 3

An 84-year-old male was admitted to our neurology clinic with pain, partly recovered vesicular rashes on the C4-5 dermatomes of approximately 1.5 month duration, and the new onset of motor paresis in his right arm. There were no abnormalities in his history except diabetes mellitus. Neurological examination yielded MRC muscle strength scores for flexion of +4/5 and abduction -4/5. The amplitudes of the musculocutaneous, axillary, and infraspinatus nerves were significantly lower on the right than on the left side. His right deltoid and biceps muscles showed fibrillation, with neurogenic MUPs and MUP loss. IgM and IgG against VZV were positive in serum. Brachial plexus MRI findings were normal (Figure 4). The patient was prescribed acyclovir as antiviral treatment, pregabalin for pain, and physiotherapy. After 2 months, his muscle strength had improved to +4/5 (mild paresis).

Written informed consent forms of each patient were taken.

DISCUSSION

The pathogenesis of zoster paresis is uncertain. Based on the association between the involved myotome and the dermatome of the rash, viral spread from the dorsal root ganglion to the anterior horn cells or anterior spinal nerve roots, resulting in inflammation and motor paresis, is suspected.[2,9] According to Hanakawa et al., the inflammation causes a neurological deficit by producing hypervascularity in the perineural structures or actual disruption of the blood-nerve barrier.[2,10] Fabian et al. suggested that inflammation of the brachial plexus is a distal extension of the dorsal ganglionitis, and in HZ, it may be a direct cause of reversible upper limb paresis. In a post-mortem study, they demonstrated that the motor neuropathy is an inflammatory demyelinating process, consistent with the recovery observed in a number of patients, and postulated that post-herpetic neuralgia is related to an ongoing inflammatory state.[3,5,11]

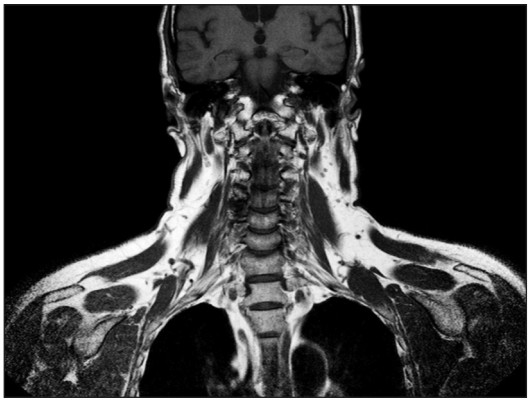

FIGURE 4: Normal brachial plexus MRI. No contrast enhancement.

Although in our patients, contrast-enhanced plexus MRI were normal may be due to subacute-chronic stage, evaluation with plexus MRI in addition to EDX studies could provide useful information about HZ plexopathy, especially in the acute phase .[8] Motor manifestations of HZ infection are rare, as 70% of patients develop the disease in areas where motor involvement is difficult to detect on physical examination, such as the upper cervical or thoracic nerve roots.[4] Motor paresis resolves completely or almost completely in 67–75% of the patients, as reported in the literature and as demonstrated in our patients.[5] The latency period between the onset of vesicular eruption and the development of limb weakness ranges from 1 day to 4 months, with an average of 2–3 weeks.[9] All three patients presented with rashes that appeared on the arm or leg, with the distribution of weakness not completely overlapping the affected dermatomes. This has been attributed, both clinically and electromyographically, to patchy involvement, and it may mimic several conditions causing pain and weakness around the shoulder, such as rotator cuff tear, acute calcifying tendinitis, impingement syndrome, cervical/lumbar radiculopathy, tumor of the brachial/lumbar plexus, and spinal cord and compressive nerve injuries. As indicated in the first case radial nerve was not influenced and inflammation did not compensate lateral cord exactly. The second and third cases were also presented with the patchy involvement. Parsonage–Turner syndrome, also known as neuralgic amyotrophy or acute brachial plexus neuritis, which is characterized by the sudden appearance of severe shoulder pain followed by progressive motor weakness, should be considered in the differential diagnosis.[8] However, the characteristic HZ skin rashes seen in our patients 3-6 weeks before the onset of weakness in the same arm or leg make this syndrome unlikely. Confirmation of the diagnosis of HZ is also obtained based on the denervation and neurogenic polyphasic MUP findings from EDX studies. The mainstay of treatment for motor paresis or brachial/lumbar plexus neuritis due to HZ is antivirals, pain relief, physiotherapy, and occupational therapy. The most commonly used effective antiviral agent is acyclovir, although the use of corticosteroids, to reduce pain and hasten healing, in combination with an antiviral drug has also been suggested.[1] In the management of VZV infection, early administration of antivirals is crucial because they reduce neural damage by inhibiting viral replication in the acute phase of the disease.[5,8]

In conclusion, herpetic brachial or lumbar plexopathy is a diagnostic challenge because it may mimic several conditions causing pain and weakness around the shoulder or leg. Affected patients differ with respect to both the clinical picture and the prognosis. To be aware of this rare complication, a detailed clinical history and physical examination as well as EDX studies are essential to distinguish the neurological structures involved and the optimal therapeutic approach.

REFERENCES

1. Jeevarethinam A, Ihuoma A, Ahmad N. Herpes zoster brachial plexopathy with predominant radial nerve palsy. Clin Med (Lond) 2009;9(5):500-1.
2. Yoleri O, Olmez N, Oztura I, Sengül I, Günaydin R, Memiş A. Segmental zoster paresis of the upper extremity: a case report. Arch Phys Med Rehabil 2005;86(7): 1492-94.
3. Ismail A, Rao DG, Sharrack B. Pure motor herpes zoster induced brachial plexopathy. J Neurol 2009;256(8):1343-5.
4. Alshekhlee A, Tay E, Buczek M, Shakir ZA, Katirji B. Herpes zoster with motor involvement: discordance between the distribution of skin rash and localization of peripheral nervous system dysfunction. J Clin Neuromusc Dis 2011;12(3):153-7.
5. Tikiz C, Mavioğlu H, Pirildar T, Tüzün Ç. [Soulder paresis due to herpes zoster neuritis: a case report]. Romatizma 2005;20(2):45-9.
6. Eyigor S, Durmaz B, Karapolat H. Monoparesis with complex regional pain syndrome-like symptoms due to brachial plexopathy caused by the varicella zoster virus: a case report. Arch Phys Med Rehabil 2006;87(12):1653-5.
7. Jones IK, Reda H, Watson JC. Clinical, electrophysiologic, and imaging features of zoster-associated limb paresis. Muscle Nerve 2014;50(2):177-85.
8. Choi JY, Kang CH, Kim BJ, Park KW, Yu SW. Brachial plexopathy following herpes zoster infection: two cases with MRI findings. J Neurol Sci 2009;285(1-2):224-6.
9. Melikoğlu M, Melikoğlu MA. An unusual cause of shoulder pain; herpes zoster induced brachial plexopathy, a case report and review of the literature. J Back Musculoskelet Rehabil 2013;26(3):243-5.
10. Hanakawa T, Hashimoto S, Kawamura J, Nakamura M, Suenaga T, Matsuo M. Magnetic resonance imaging in a patient with segmental zoster paresis. Neurology 1997;49(2): 631-2.
11. Fabian VA,Wood B, Crowley P, Kakulas BA. Herpes zoster brachial plexus neuritis. Clin Neuropathol 1997;16(2):61-4.