C8 Myotome Herpes Zoster Paresis

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Abstract

Herpes zoster (HZ) infection is a reactivation of latent varicella zoster virus that causes pain and a rash in a dermatomal distribution. Previous reports suggest that 0.5-5% of HZ infections are associated with a myotomal paresis but the incidence may actually be much higher. We present a patient with HZ infection who had persistent right upper extremity weakness after resolution of the rash. Electrodiagnostic studies demonstrated decreased amplitudes in the median and ulnar nerves as well as denervation in the right C8 myotome. Repeat studies showed interval C8 reinnervation as well as normal nerve conduction studies.

Herpes zoster (HZ) infection, or shingles, occurs frequently in the general population. The infection usually presents with a viral prodrome and a characteristic and frequently painful maculopapular rash along a single dermatome. The erythematous macules then quickly evolve into vesicles and sometimes bullae. Occasionally, HZ infection can affect a myotome causing a transient paresis. When HZ paresis occurs in the upper extremity, it usually does so in the C5-7 distribution.[1-4] Paresis of the C8 myotome is rare and the presenting symptoms can be confused with cubital tunnel syndrome. We present a case of a 71-year-old woman with an HZ paresis in the C8 myotome. To our knowledge, there are only a few previously reported cases in the literature. In 1961, Grant and colleagues[5] described several cases of zoster paresis, including one that involved C6-8 myotomes. In 2000, Matondo and colleagues[6] reported a case of HZ paresis, of the ulnar nerve in the Tropical Doctor, and in 2005, Athwal and colleagues[7] reported a similar case in the *Journal of Hand Surgery* (British and European volume). Informed consent was obtained from the patient presented in this report to allow its publication.

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Am J Orthop. 2012;41(5):220-222.

Case Report

A 71-year-old woman presented to her primary care physician (PCP) after 4 days of pain in her right arm. According to the medical record, the pain was localized to the medial aspect of the arm and the ulnar digits of the hand. The PCP diagnosed the patient with HZ infection versus cervical radiculopathy and treated the patient with gabapentin and narcotic analgesics. The patient was provided with a prescription for valcyclovir, with instructions to start if a rash developed. Three days later, the patient noted groups of red vesicles on her hand and elbow in a C8 dermatomal distribution. She began the valacyclovir for HZ infection and the PCP titrated the gabapentin up for neuropathic pain control. Five weeks after the rash developed, the patient visited her PCP and was noted to have persistent right arm pain in the C8 dermatome that required narcotic analgesics, as well as a new report of development of right hand weakness.

The patient was referred to an orthopedic hand and upper extremity clinic. A physical examination revealed a positive Froment's sign and Wartenberg's sign with negative provocative maneuvers for carpal tunnel and cubital tunnel syndromes. The reported rash had resolved. There was decreased sensation to light touch in the third through fifth fingers and 3/5 strength in finger flexors and abductors. The patient was still reporting dermatomal arm pain and paresthesia. Therefore, gabapentin was increased to a therapeutic dose and electrodiagnostic studies were ordered.

Seven weeks after the onset of pain, a nerve conduction study demonstrated decreased right median and ulnar compound motor action potential amplitudes (Figure 1). Ulnar sensory nerve action potential amplitude was also decreased. Needle electromyogram (EMG) examination was notable for partial denervation of right C8-innervated muscles including the abductor pollicis brevis, first dorsal interosseous, and extensor indicis proprius. The patient was diagnosed with HZ paresis in the C8 myotome.

The patient went to physical therapy to maintain joint range of motion and strengthen complimentary muscles. Within a month, she was able to wean off narcotic pain medicine. Three months after weakness was noted, the patient's PCP documented interval partial improvement of right grip strength and began to taper the gabapentin. Two months later, repeat examination at the orthopedic clinic revealed negative Wartenberg's sign and negative Froment's sign.

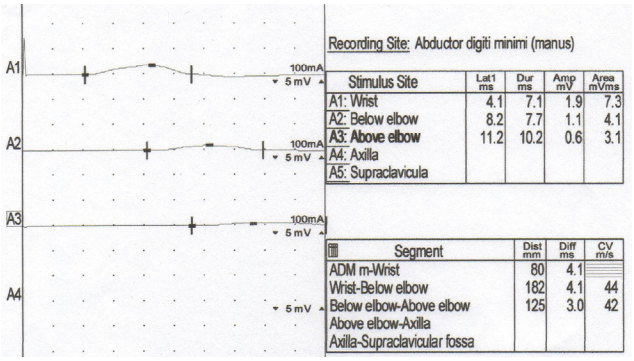

Figure 1. Ulnar motor nerve conduction study from 1/27/2009 demonstrating severely decreased amplitudes (normal value in our laboratory for ulnar compound motor action potential amplitude is > 5.0 mV) and mild uniform conduction slowing (this pattern of slowing is typical of axonal loss, as contrasted with the pattern of focal slowing and/or conduction block observed in cases of entrapment neuropathy).

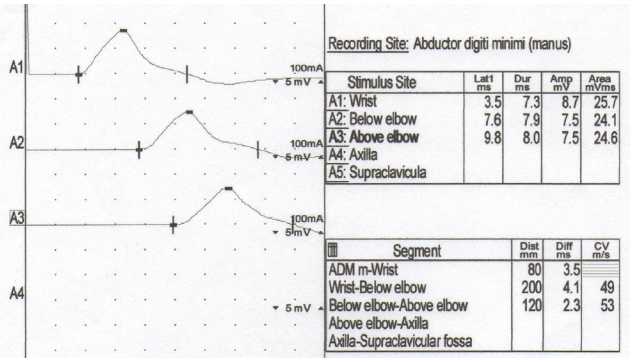

Figure 2. Ulnar motor nerve conduction study from 7/17/2009 demonstrating normal latency and significantly improved amplitudes.

However, the patient had persistent numbness in her third through fifth fingers and a repeat electrodiagnostic study was ordered to assess for ulnar neuropathy. Compressive neuropathy at the cubital tunnel was of specific interest due to the results of the previous study. Nerve conduction studies obtained 6 months after the first study were significantly improved (Figure 2). There was no significant evidence of median or ulnar focal demyelination, including a normal inching study at the medial elbow. Needle EMG examination of the right hand intrinsics was notable for evidence of partial chronic denervation of the right abductor pollicis brevis and first dorsal interosseous muscles with evidence of significant interval reinnervation since the prior study.

Thirteen months from the onset of the original HZ infection, the patient continued to experience hand numbness and weakness but her symptoms stabilized. She continued to perform daily home therapy and was taking amitriptyline and low-dose gabapentin.

Discussion

HZ infection, also commonly known as shingles, is caused by reactivation of the latent varicella zoster virus (VZV), also known as chicken pox. Prior to the development of the VZV vaccine in 1995, chicken pox was most prevalent in children less than 10 years old.[8] Primary VZV infection manifests with a viral prodrome followed by a characteristic diffuse pruritic vesiclar rash that can last in some form for 1 to 2 weeks. The virus then lies dormant in the dorsal root ganglion and can reactivate years later causing an HZ infection. HZ infection most commonly occurs in the fifth through seventh decades, but can occur at any point in an individual's lifespan, and has a lifetime risk possibly as high as 10-20%.[9] The most common causes of reactivation are immunosuppression and age-related decline in immunity.[10-11] During reactivation, symptoms manifest as a viral prodrome of pruritis, paresthesia, or allodynia along a unilateral dermatome. Fatigue, malaise, low-grade fever, and nausea can accompany the initial symptoms. Several days later, an erythematous, maculopapular, and vesicular rash follows in the same unilateral dermatomal distribution.

The thoracic and lumbar dermatomes are most commonly involved, but as this case report suggests, reactivation can occur in cervical dermatomes, as well as cranial nerve distributions.[1] The virus usually affects the sensory ganglion but can spread to one or more nerve roots, the spinal cord, the brachial plexus, or a peripheral nerve.[1,12] Myotomal involvement has been reported in past literature in 0.5-5% of HZ cases,[2,5,13] but may actually be much higher when evaluated by EMG studies. Mondelli and colleagues[12] reported myotomal paresis, detected on EMG, in 36% of 158 consecutive HZ infections that affected the head, upper, and lower limbs. Haanpaa and colleagues[14] reported an even higher rate of paresis but in a smaller sample size, 53% of 40 cases, and their study included EMG evaluation of cranial, cervical, thoracic, lumbar, and sacral regions. Subclinical motor paresis was detected by EMG in 17% of HZ cases in Mondelli's study[12] and 62% of cases in Haanpaa's study.[14] The reason for this discrepancy is that Mondelli's study[12] evaluated HZ in regions where muscle weakness could be more easily recognized by the patient and examiner, while Haanpaa's study[14] included thoracic, lumbar, and sacral nerves where muscle weakness is difficult to test and may go unnoticed. Pain can also complicate examination by limiting muscle testing and masking patient-recognized weakness. Myotomal paresis is therefore most likely under-recognized, underdiagnosed, and under-reported.

When myotomal paresis does occur, it usually does so simultaneously or within 2 weeks of the onset of rash and is most commonly noted in the form of facial paralysis or paresis of the proximal upper extremity musculature in the C5-7 distribution.[1-4] Involvement of the C8 myotome is considered rare.

Diagnosing C8 zoster paresis can be difficult, particularly in the setting of a patient who is a poor historian or who has had an undiagnosed or atypical presentation of HZ infection. As documented in our report, isolated C8 paresis presents with allodyna and/or paresthesia in

an ulnar nerve distribution and weakness of the hand intrinsic muscles. The infection could easily be confused with an acute onset of cubital tunnel syndome. Electromyographic and nerve conduction studies are necessary in this setting to distinguish between the diagnoses.

In patients with HZ paresis, sensory nerve action potentials (SNAP) may be reduced or absent. SNAP distal latencies and conduction velocities within the affected distribution are either unaffected or mildly prolonged. Compound motor action potential (CMAP) amplitudes will be reduced while motor latency and conduction velocity will likely only be mildly affected due to drop out of the fastest axons. In cubital tunnel syndrome, one would expect conduction block and/or focal conduction slowing at the elbow. Conduction block would present as decreased CMAP amplitude proximal to the lesion with a normal amplitude at distal stimulation sites. Another finding in cubital tunnel syndrome is decreased conduction velocity across the elbow with normal conduction velocity in the forearm. To increase the sensitivity of testing for conduction slowing, and to specifically localize the lesion, the electromyographer can perform an inching study across the elbow.[15] In the case of our patient, a serial study was ordered to confirm that there was no evidence of ulnar nerve compression, and although serial EMG and nerve conduction studies are not necessarily a standard of care, they should be considered if there is clinical uncertainty.

Prognosis for return of motor function after zoster paresis is considered good (75%), but return can be partial and time to recovery can vary over a period of months to years.[16] Early recognition HZ infection and early administration of anti-viral therapy has been found to reduce both peripheral sensory involvement and viral spread and may help to minimize motor involvement.[1,12,14] While there is little information in the literature about the role of physical or occupational therapy after zoster paresis, a referral is generally advocated to help maintain joint range of motion, strengthen and utilize complimentary muscles, and to strengthen involved muscles as the paresis resolves.[17]

Conclusion

HZ paresis is an uncommon complication of HZ infection and usually occurs in the C5-7 myotome. HZ paresis should be considered in the differential diagnosis of segmental upper extremity weakness to avoid unnecessary testing. HZ paresis is rare in the C8 myotome and should not be confused with the more commonly occurring cubital tunnel syndrome. Correct diagnosis is important to avoid unnecessary surgical procedures and their associated risks. Patients should be educated that the prognosis for partial or complete recovery is good, but may take months to years.

Authors' Disclosure Statement

The authors report no actual or potential conflict of interest in relation to this article.

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