

## The Role of Electromyography when Zoster Disguises A more Sinister Process: A Case Report

Mary E. Lynch\* and Anna Sophia Del Fabro

Department of Physical Medicine and Rehabilitation, Mayo Clinic, USA

\*Corresponding author: Mary E. Lynch, Resident Physician, Department of Physical Medicine and Rehabilitation, Mayo Clinic, Mayo Clinic 200 First Street SW Rochester, Minnesota 55905, USA, Tel: 507-266-8913; Fax: 507-284-3431; E-mail: Breen.Mary@mayo.edu

Received date: July 24, 2018; Accepted date: August 14, 2018; Published date: August 16, 2018

Copyright: ©2018 Lynch ME, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.

### Abstract

This case demonstrates how electromyography (EMG) helped uncover a more sinister process when the clinical course of zoster-associated limb paresis was not as expected during acute inpatient rehabilitation. An underlying nerve disorder complicated this case, a possible example of the dual nerve disorder phenomenon that is recognized by many clinicians. Ongoing investigation and discussion with neurology colleagues was necessary to re-evaluate so that the patient ultimately received effective treatment for an inflammatory immune mediated demyelinating polyradiculopathy.

**Keywords:** Polyradiculopathy; Zoster; Electromyography

### Background

Investigation by needle electromyography (EMG) is an extension of the clinical history and physical exam for a patient with a suspected neuromuscular disorder. It can be used to evaluate anterior horn cells, peripheral nerves, neuromuscular junctions, and muscles. EMG provides information about the type of lesion, lesion location, severity of condition, and often the duration or stage of a condition [1]. One such condition is zoster-associated limb paresis (ZALP).

Herpes zoster, also known as shingles, has many neurologic complications. Zoster-associated limb paresis has been frequently described in the literature but is uncommonly anticipated when a zoster rash occurs. The severity of impairment can vary greatly from trace weakness to complete paralysis. There can be discrepancies between the affected myotomal and dermatomal distributions [2]. This can lead to difficulty when differentiating from other musculoskeletal, nerve root, or peripheral nerve disorders [3]. In one case series of 49 patients with ZALP at Mayo Clinic by Jones et al., the mean minimal duration of weakness was 193 days and the incidence of post-herpetic neuralgia at one month was 92% [4]. It has not been definitively elucidated whether this process results in motor damage from the anterior horn cell, ventral root, plexus, or peripheral nerve. However, the EMG findings described by Jones et al. found that the damage was localized to the plexus or peripheral nerve in 63% of their 49 cases [4]. Mondelli et al. described electromyographic abnormal spontaneous activity at rest in muscles of at least 1 myotome corresponding to the affected dermatome. Interestingly, signs also extended to muscles corresponding to contiguous unaffected dermatomes and to muscles on the other side in some patients without clinical signs or symptoms of polyneuropathy [5]. This suggests that EMG can detect more diffuse nerve involvement than can be detected on visual inspection of the skin or manual motor testing.

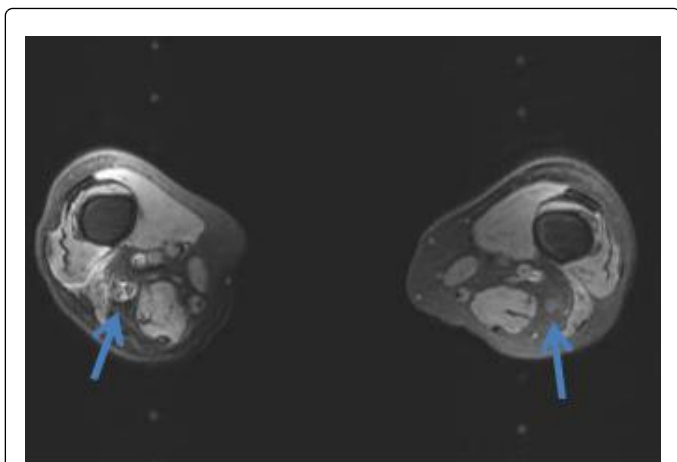
The double crush nerve entrapment theory, described in 1973 by Upton and McComas, states that neural function can be impaired because single axons have been compressed in one area, leaving the

remainder of the axon especially susceptible for damage at another site [6]. This has been loosely expanded to hypothesize that having underlying peripheral nervous system damage makes one more vulnerable to further nerve damage from a different source. Although clinicians recognize this phenomenon in their practice, the underlying pathophysiology of dual nerve disorders has not been established [7]. In our case report, we describe a gentleman with a pre-morbid peripheral neuropathy who was thought to be severely affected by ZALP until serial EMGs revealed a more sinister process. If he did indeed have ZALP, this raises the question of the diagnosis of dual nerve disorder or even triple nerve disorder.

### Case Description

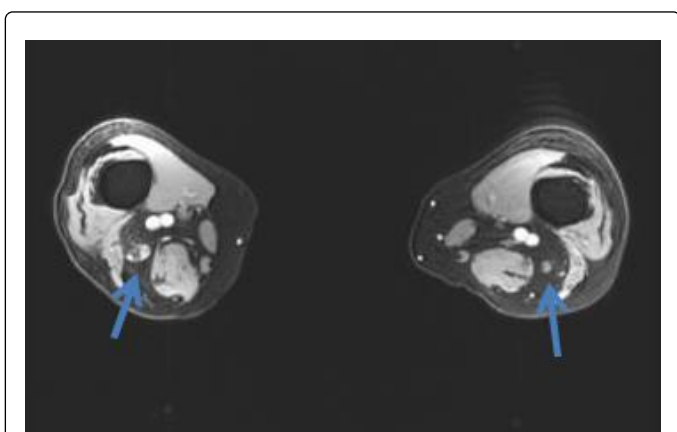
This patient is a 70-year-old man with history of non-Hodgkin's mucosa-associated lymphoid tissue (MALT) B-cell lymphoma in remission. He had previously undergone an EMG study due to mild bilateral foot sensory changes which showed a chronic, length-dependent, mixed axonal and demyelinating, sensorimotor, large fiber peripheral neuropathy. Differential diagnosis at that time was glucose-associated peripheral neuropathy versus antibody-associated neuropathy versus chemotherapy-induced neuropathy. He did not have any testing to further delineate.

Six weeks after his initial EMG, he had a rapid decline in his bilateral lower extremity strength, right more affected than left, in the setting of a bilateral S1 and right sided L1 dermatome zoster rash. Repeat EMG at presentation (now his second EMG) was not substantially changed from prior and was still without signs of lumbosacral radiculopathy or plexopathy. Antiviral therapy is thought to reduce the possibility of segmental zoster paresis and the severity of the peripheral nerve damage; therefore, he was empirically treated with acyclovir. Subsequently, he was transferred to an acute inpatient rehabilitation facility. At the time of admission to rehabilitation, his diagnosis was pre-morbid chronic polyneuropathy with a superimposed zoster-associated radicular plexitis. The severity of his right lower extremity weakness from the zoster was thought to be an illustration of dual nerve disorder.



**Figure 1:** MRI T2 image of bilateral thighs demonstrates asymmetry of nerve enhancement. Sciatic nerve has divided into peroneal and tibial trunks at this level.

During his rehabilitation stay, he developed right lower extremity radicular pain, neurogenic bowel and bladder, and bilateral arm weakness. Bowel and bladder dysfunction has been associated with zoster infection in the lumbosacral region so this symptom was less alarming [8,9]. However, his left leg weakness progressed and his right leg worsened to only minimal activation in most major muscle groups. This was not the anticipated course of his initial diagnosis and further workup was indicated. CT-PET scan did not show any evidence of lymphoma recurrence. A nerve biopsy was non-diagnostic. MRI of the lumbar spine showed mild irregularity and low-level enhancement of multiple cauda equina nerve roots with slightly more prominent enhancement of multiple right-sided nerve roots, most noticeably L2, L5, and S1. MRI of the lumbar plexus showed enlargement and enhancement of the right-sided lumbosacral plexus, sciatic, peroneal, and tibial nerves.



**Figure 2:** MRI T1 post gadolinium image of Figure 1.

Figure 1 shows the right tibial and peroneal nerve enhancement as compared to left on T2 image and Figure 2 shows the same level post gadolinium. He had his third EMG six weeks after his presentation of the rash and weakness. This showed polyradiculoneuropathy with ongoing denervation or incomplete reinnervation in right thoracic,

right L3-S1 and left L5/S1 myotomes, suggesting bilateral lumbosacral radiculoplexi neuropathy with right thoracic radiculopathies. These diffuse findings pointed towards a more malicious underlying process rather than zoster radiculoplexitis alone.

He was re-admitted to the acute hospital with further progression of his weakness and bilateral facial nerve involvement. In the absence of any compelling evidence for lymphoma on nerve biopsy, CSF studies, and imaging, he was treated with immunotherapy for an inflammatory immune mediated demyelinating polyradiculopathy. He received a five-day course of IV immunoglobulin and methylprednisolone 1 g IV with some improvement in bilateral upper extremity strength, left lower extremity strength, and bowel and bladder function. Unfortunately, he had no improvement in the right lower extremity strength. He continued on oral prednisone 60 mg and methotrexate 20 mg per day.

There is still a possibility of a low-grade lymphoma such as diffuse infiltrative lymphocytosis syndrome (DILS), or a paraneoplastic syndrome driving this process. At this time, he is following closely with his hematologist and able to live at home with the physical assistance of his wife.

## Discussion

Zoster can spread beyond the affected dermatome and result in myotomal weakness [4]. Serial EMGs can reveal the extent of involvement as well as the possibility that a different disease process is present and evolving rather than the one originally diagnosed. Patients with underlying peripheral neuropathy may be more susceptible to further nerve damage from an unforeseen secondary disease process, such as zoster or immune mediated inflammatory neuropathy. Multiple authors have characterized zoster-associated paresis, however concomitant peripheral neuropathy has not been correlated with paresis severity. In fact, the larger reviews in the literature that have discussed EMG characteristics excluded patients with pre-morbid peripheral nervous system disorders such as peripheral neuropathy. This could be the aim of future research.

This case highlights the importance of performing serial EMGs when initial evaluation is medically complicated with unexpected deficits. In our patient, the delayed study revealed more diffuse involvement and favored a more sinister process that changed the course of treatment. We will never know if our patient's acute polyneuropathy simply began in the right leg, making the zoster rash a red herring, or if he indeed had ZALP prior to developing his immune mediated process. We do know that ongoing evaluation and curiosity about his symptoms fitting his current diagnosis ultimately led to an effective treatment.

## Conclusion

When a patient is admitted to acute inpatient rehabilitation with a specific neurologic diagnosis, there is generally an expected clinical course. When the patient's actual course is not as expected, physiatrists should feel empowered to discuss ongoing workup or evolving differential diagnoses with neurology colleagues. EMG can be a helpful tool in this process of re-evaluation.

## References

1. Rubin D, Daube J (2016) Clinical Neurophysiology. 4th ed. Oxford University Press.

2. Alshekhlee A, Tay E, Buczek M, Shakir ZA, Katirji (2011) Herpes zoster with motor involvement: discordance between the distribution of skin rash and localization of peripheral nervous system dysfunction. *J Clin Neuromuscul Dis* 12: 153-157.
3. Sprenger De Rover WB, Alazzaw S, Hallam PJ, Hutchinson R, Di Mascio L (2011) Herpes zoster virus: an unusual but potentially treatable cause of sciatica and foot drop. *Orthopedics* 34: e965-968.
4. Jones LK Jr, Reda H, Watson JC (2014) Clinical, electrophysiologic, and imaging features of zoster-associated limb paresis. *Muscle Nerve* 50: 177-185.
5. Mondelli M, Romano C, Rossi S, Cioni R (2002) Herpes zoster of the head and limbs: electroneuromyographic and clinical findings in 158 consecutive cases. *Arch Phys Med Rehabil* 83: 1215-1221.
6. Upton AR, McComas AJ (1973) The double crush in nerve entrapment syndromes. *Lancet* 18: 359-362.
7. Schmid AB, Coppieters MW (2011) The double crush syndrome revisited-a Delphi study to reveal current expert views on mechanisms underlying dual nerve disorders. *Man Ther* 16: 557-562.
8. Matsuo T, Oba K, Miyata Y, Igawa T, Sakai H (2014) Four cases of urinary dysfunction associated with sacral herpes zoster. *Hinyokika Kiyo* 60: 87-90.
9. Bhalerao VM, Gedam BS, Mukharjee AK (2015) Herpes zoster infection presenting with urinary retention and constipation. *J Assoc Physicians India* 63: 63-65.